Journal of Bioenergetics and Biomembranes (1976) 8, 143–156

Phenylenediamine Restoration of Photosynthetic Electron Flux in DBMIB-Inhibited Chloroplasts*

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Revise received 13 November 1975

Abstract

Phenylenediamines have been studied and compared as to their effectiveness in stimulating photosynthetic electron flux in DBMIB-inhibited chloroplasts. It has been found that N-substituted as well as C-substituted *p*-phenylenediamines accelerate the rate of ferricyanide reduction, a photosystem II photoreaction, under conditions where the radical cations of N-substituted *p*-phenylenediamines are stable. The P/e_2 ratios for these partial reactions are between 0.4 and 0.5; this is taken as evidence that N-substituted *p*-phenylenediamines are reduced by the choloroplasts close to the outer surface. Both N- and C-substituted *p*-phenylenediamines are capable of bypassing the site of DBMIB inhibition and restoring electron flow from water to methylviologen. N-substituted *p*-phenylenediamines appear to be more effective even at high concentrations of DBMIB. The P/e_2 ratios for these reactions are on the order of 0.75–1.0; this is taken as evidence that the bypass reaction for N-substituted *p*-phenylenediamines occurs on the inside of the thylakoid membrane.

* Abbreviations: DBMIB, 2,5-dibromo-3-methyl-6-isopropyl-*p*-benzoquinone (dibromothymoquinone); PD, *p*-phenylenediamine; TMPD, *N,N,N',N'*-tetramethyl-*p*-phenylenediamine; DMPD, *N,N*-dimethyl-*p*-phenylenediamine; FeCN, potassium ferricyanide; MV, methylviologen; BSA, bovine serum albumin (Cohn-Fract. V-Serva).

Introduction

Noncyclic electron transport, driven by both chloroplast photosystems, is coupled to phosphorylation presumably through a pH gradient maintained by (1) the deposition of the protons from water oxidation on the inside of the thylakoid membrane and (2) from the vectorially driven plastoquinone "Mitchellian loop" bringing protons from the outside into the inside (for a recent review see Trebst, [1]). Selective inhibitors and electron acceptors and donors have enabled the study of the partial reactions driven by either of the two photosystems without the need of physically separating photosystem I and II particles. Photosystem II electron transport has generally been isolated using either the electron transport inhibitor dibromothymoquinone (DBMIB) which, at low concentrations, prevents the reoxidation of the plastoquinone pool by photosystem I [2–4], or with KCN-treated chloroplasts, a treatment which has been demonstrated to inhibit electron flux through plastocyanin [5–7].

Ferricyanide reduction and concomitant phosphorylation, in the presence of DBMIB, can be stimulated by the addition of lipophilic electron transport mediators (so called class III acceptors) [8, 9]. The P/e_2 ratios measured for these photosystem II reactions are on the order of 0.5, i.e., half the overall P/e_2 ratio for noncyclic phosphorylation. Energy conservation in this partial reaction is probably associated with the release of protons from water oxidation on the inside of the thylakoid membrane.

Electron transport to $NADP^+$, in the presence of DBMIB, can also be restored by using a donor couple (e.g., ascorbate and either diaminodurene or dichlorophenolindophenol) [10, 11]. The P/e_2 ratios for these reactions are also around 0.5 [12, 13]. The chemistry of the electron transport mediator plays a crucial role in determining whether or not a particular donor to photosystem I will be coupled. Hauska et al. [14] have interpreted these latter results as indicating that diaminodurene brings protons across the membrane and deposits them stoichiometrically inside upon oxidation, forming an artificial energy-conserving site for photosystem I. Compounds such as *N,N,N,'N'*–tetramethyl-*p*-phenylenediamine (TMPD) do not involve hydrogen transfer upon oxidation–reduction and therefore electron transport from ascorbate plus TMPD to $NADP^+$ is not coupled to phosphorylation.

A third mode of electron transport has recently been shown by Trebst and Reimer [15, 16] to take place in DBMIB-poisoned chloroplasts. DBMIB inhibition of $NADPH^+$ reduction as well as of oxygen evolution was shown to be reversed by the addition of catalytic amounts of just N-substituted phenylenediamines (without the addition of ascorbate). The observation that the P/e_2 ratio in the restored $NADP^+$ reduction system reflected the P/e_2 ratio of uninhibited chloroplasts (close to 1.0)

led Trebst and Reimer [16] to suggest that TMPD and other N-substituted phenylenediamines work primarily on the inside of the chloroplast membrane making a "bypass" around the DBMIB inhibition site.

We have reexamined the reactions catalyzed by N- and C-substituted phenylenediamines in order to learn more about the location, with respect to membranes sidedness, where these substances react with the electron transport chain. In this paper we show that both N- and C-substituted phenylenediamines, under conditions where the radical cation is relatively stable, stimulate the reduction of ferricyanide and the concomitant phosphorylation. In addition, we compare the effectiveness of TMPD and phenylenediamine (PD) in the bypass of the DBMIB inhibition site for another reaction requiring both photosystems, i.e., methylviologen-catalyzed oxygen consumption, and show that in this reaction TMPD seems to bypass the DBMIB inhibition site on the inside of the membrane.

Materials and Methods

Chloroplasts were prepared as previously described [17] in buffer containing 0.4 M sucrose, 0.02 M Tris-HCl (pH 7.6), 10 mM KCl, 5.0 mM $MgCl_2$, and 2.0 mg/ml BSA and resuspended in the same buffer without BSA. The chlorophyll concentration was determined as described by Arnon [18].

contained in 3.0 ml, chloroplasts equivalent to 20 μg Chl/ml, 50 mM spectrophotometer modified for side illumination. The cuvette was continually stirred and illuminated with red light isolated from a Schott glass filter (No. RG 645) and passed through 7 cm water before impinging upon the sample. Unless otherwise stated, reaction mixtures contained in 3.0 ml, chloroplasts equivalent to 20 μg Chl/ml, 50 mM Tricine–NaOH buffer (pH 8.0), 10 mM KCl, 5.0 mM $MgCl_2$, 5.0 mM ADP, 3.3 mM [^{32}P] phosphate (approx. 7.5×10^5 cpm/ml), 1.0 mM potassium ferricyanide, and 1.0 mM potassium ferrocyanide.

Methylviologen-catalyzed oxygen consumption was measured in a Clark-type Teflon-covered electrode described by Delieu and Walker [19] at 20° C. Reaction mixtures contained, in 3.0 ml, chloroplasts equivalent to 20 μg Chl/ml, 50 mM Tricine–NaOH buffer (pH 8.0), 10 mM KCl, 5.0 mM $MgCl_2$, 0.5 mM methylviologen, 0.1 mM NaN_3, 5.0 mM ADP, and 3.3 mM [^{32}P] phosphate (approx. 7.5×10^5 cpm/ml) and were illuminated with white light.

ATP formation was measured by the incorporation of [^{32}P] phosphate into organic phosphate as described by Conover et al. [20] with the modification that after extraction of unesterified phosphate into the organic solvents, aliquots of the inorganic phase containing

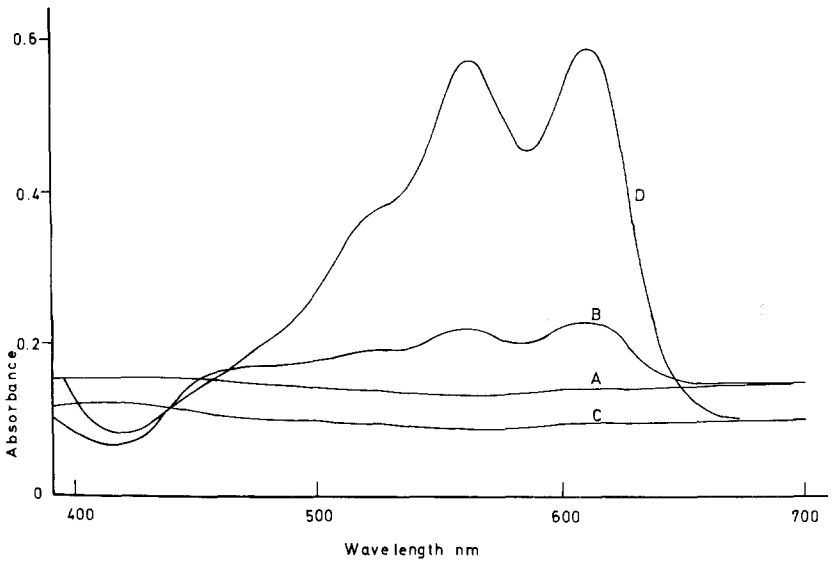


Figure 1. Spectrum of the TMPD radical cation. Difference spectra were measured with a Cary–15R spectrophotometer in 3.0-ml reaction mixtures containing 20 mM Tris-HCl buffer (pH 7.6), 10 mM KCl, and 5 mM $MgCl_2$. The following curves describe additions to the sample cuvette compared to (minus) the reference cuvette. Curve A, 1.0 mM ferricyanide minus 1.0 mM ferricyanide; curve B, 1.0 mM ferricyanide, 50 μM TMPD minus 1.0 mM ferricyanide; curve C, 1.0 mM ferricyanide, 1.0 mM ferrocyanide minus 1.0 mM ferricyanide, 1.0 mM ferrocyanide; curve D, 1.0 mM ferricyanide, 1.0 mM ferrocyanide, 50 μM TMPD minus 1.0 mM ferricyanide, 1.0 mM ferrocyanide. All spectra were measured approx. 5.0 min after preparation of the sample.

organic phosphate were pipetted into scintillation vials containing 5.0 ml of New England Nuclear Aquasol Universal L.S.C. cocktail and were counted in a Packard Tri-Carb Liquid Scintillation Spectrometer model 3385.

DBMIB and gramicidin were dissolved in methanol and added to the reaction mixtures as the methanolic solutions. The concentration of organic solvents never exceeded 1% of the reaction mixture volume.

The spectrum of TMPD was measured using a Cary 15-R recording spectrophotometer. Details are given in the legend to Fig. 1.

Results

Stability of TMPD Radical Cation

The rate of ferricyanide reduction by photosystem II in DBMIB-poisoned chloroplasts has previously been reported to be stimulated

three- to fourfold by C-substituted *p*-phenylenediamines and quinones [8, 9]. In contrast, N-substituted *p*-phenylenediamines [e.g., TMPD and *N,N*-dimethyl-*p*-phenylenediamine (DMPD)] were found to be relatively inactive under the same conditions [15]. We have observed, however, that the characteristic blue color of the TMPD radical cation quickly vanishes in the ferricyanide reaction mixture. Fig. 1 shows the spectrum of TMPD taken 5 min after the addition of 50 μM TMPD to a buffered solution containing either 1.0 mM ferricyanide (curve B) or a mixture of 1.0 mM ferricyanide plus 1.0 mM ferrocyanide (curve D). Clearly the concentration of Wursters Blue in the redox-poised solution is six- to sevenfold greater than in the ferricyanide solution. Attempts to titrate the disappearance of the radical cation intermediate as a function of increasing redox potential were unsuccessful due to the instability of the intermediate at redox potentials greater than 350–360 mV. Once TMPD is fully oxidized by ferricyanide, it cannot be re-reduced by ascorbate or dithionite and, therefore, it is believed that further oxidation of the TMPD radical cation leads to the eventual destruction of TMPD. If this were the case, it could explain the inability of TMPD to stimulate the rate of ferricyanide reduction in reaction mixtures containing only ferricyanide.

Photoreductions by Photosystem II: Phenylenediamine-Mediated Reduction of Ferricyanide

Figure 2 shows the concentration dependencies for two phenylenediamines, TMPD and PD, on ferricyanide reduction in the presence of DBMIB using a redox-poised system (equimolar ferri-ferrocyanide solution). Both phenylenediamines accelerate the rate of reduction of ferricyanide under these conditions. The TMPD radical cation must be formed under conditions where it is stable in order for catalytic amounts of TMPD to be able to mediate electron flux from the electron transport chain to ferricyanide. There are, however, some qualitative differences between PD and TMPD. Between 100 and 400 μM TMPD, the increase in the rate of ferricyanide reduction is linear and is clearly not saturated by 400 μM (higher concentrations of TMPD could not be tested under our experimental conditions due to the intense blue color of the radical cation). In contrast, PD-stimulated ferricyanide reduction saturates around 80–100 μM. Another marked difference is the contrasting effect of uncouplers on the phenylenediamine-mediated rate of electron transport (data shown for gramicidin; however, no differences were observed between gramicidin and ammonium chloride). In agreement with the results previously reported by Trebst and Reimer [16], gramicidin is seen to inhibit the PD stimulation of ferricyanide reduction at low PD concentrations. However, by increasing concentrations of PD, gramicidin has no effect on the rate of electron flux (at pH 7.6). In the

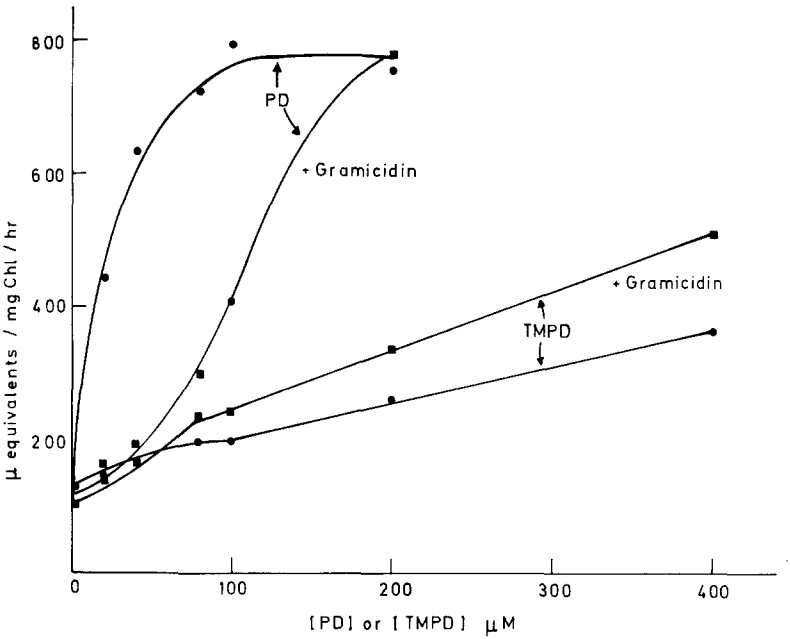


Figure 2. Phenylenediamine stimulation of ferricyanide reduction in DBMIB-inhibited chloroplasts. Reaction mixtures contained, in 3.0 ml, chloroplasts equivalent to 23.3 μg Chl/ml, 20 mM Tris-HCl buffer (pH 7.6), 10 mM KCl, 5 mM $MgCl_2$, 1.0 mM ferricyanide, 1.0 mM ferrocyanide, 0.33 μM DBMIB, and 20 μM gramicidin, where indicated. Ferricyanide reduction was measured as described in *Materials and Methods.*

case of TMPD, uncouplers have no or only a marginal effect on the rate of ferricyanide reduction at low TMPD concentrations, which are ineffective in increasing the rate of ferricyanide reduction. Above 80 μM TMPD, where the stimulation of the basal rate of ferricyanide reduction by TMPD is more prominent, uncouplers actually markedly stimulate the rate of ferricyanide reduction.

The stimulatory effect of gramicidin on the basal rate of ferricyanide reduction with TMPD as the electron transport mediator could be due to an uncoupling of "site I" phosphorylation [as suggested by Gould and Ort, ref 21]. Also, from Fig. 2 it cannot be excluded that, at concentrations greater than 80 μM, the reduction site for ferricyanide is not in photosystem I. If, however, the cause for the TMPD stimulation of ferricyanide reduction were due to a bypass of TMPD around the DBMIB inhibition site, enabling ferricyanide to be reduced by photosystem I, then one would anticipate that the P/e_2 ratio for

TABLE I. Phenylenediamine-stimulated coupled electron transport in the presence of DBMIB

Additions to reaction mixtures[a]	μM ATP (mg Chl/hr)	μEq (mg Chl/hr)	P/e_2
None	140	308	0.91
DBMIB	9	94	0.19
DBMIB + PD	45	300	0.30
DBMIB + TMPD	24	204	0.24
DBMIB + DMPD	26	226	0.23

[a] Reaction mixtures were as described in *Materials and Methods* and contained, in addition, 1.0 μM DBMIB and 125 μM each of TMPD, PD, and DMPD, where indicated.

TMPD-stimulated ferricyanide reduction would be closer to the control P/e_2 ratio (i.e., the P/e_2 ratio in the absence of DBMIB). Table I compares the influence of two N-substituted *p*-phenylenediamines, TMPD and DMPD, with PD on the P/e_2 ratio for the reaction water to ferricyanide in the presence of DBMIB. Although both TMPD and DMPD are capable of significantly stimulating ferricyanide reduction (117 and 140%, respectively) in the poised system, the P/e_2 ratios remain low and are characteristic of the P/e_2 ratios for photosystem II electron acceptors. Figure 5 shows concentrations curves for the phenylenediamine stimulation of ferricyanide reduction and the accompanying photophosphorylation. As seen in the upper curves, the P/e_2 ratios for water to ferricyanide are independent of the concentration of the mediator used and are about the same for both TMPD (Fig. 5A; $P/e_2 = 0.34$) and PD (Fig. 5B; $P/e_2 = 0.30$). It is rather unlikely, then, that high concentrations (100–400 μM) of TMPD, which significantly stimulate ferricyanide reduction in the presence of DBMIB (2.5–4.5-fold, respectively, see Fig. 5A), bypass the DBMIB inhibition site allowing ferricyanide to be reduced at photosystem I.

Photoreductions by Photosystem I and II: Phenylenediamine Induced Bypass Around the DBMIB Inhibition Site

Figures 3, 4, and 5 compare the effectiveness of TMPD and PD in restoring electron flow in DBMIB-poised chloroplasts for a reaction that requires both photosystems, electron transport from water to methylviologen (measured as oxygen consumption). Both phenylenediamines are capable of "reversing" the DBMIB inhibition of electron transport although there are some differences as to the efficiency of the restoration. Figure 4 shows that, in the presence of low concentrations

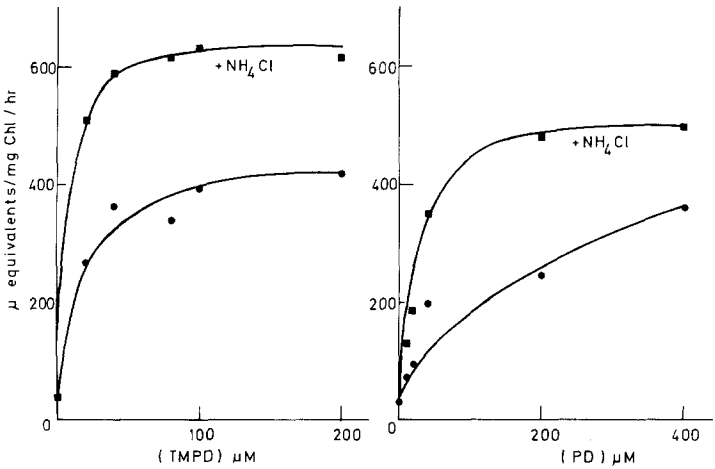


Figure 3. Restoration of methylviologen-catalyzed oxygen consumption by phenylenediamines in DBMIB-inhibited chloroplasts. Reaction mixtures contained, in 3.0 ml, chloroplasts equivalent to 21.2 μg Chl/ml, 20 mM Tris-HCl (pH 7.6), 10 mM KCl, 5 mM $MgCl_2$, 0.5 mM methylviologen, 0.1 mM NaN_3, 0.33 μM DBMIB, and, where indicated, 5.0 mM ammonium chloride (NH_4Cl). Oxygen consumption was measured as described in *Materials and Methods.*

of DBMIB (up to 10^{-7} M DBMIB in a reaction mixture containing 20 μg Chl/ml), both TMPD and PD are capable of restoring methylviologen reduction. Above 10^{-7} M DBMIB, both reactions remain inhibited, TMPD being less sensitive than PD. However, as shown in Fig. 5, the DBMIB inhibition of the PD-induced bypass can be overcome by increasing concentrations of PD. In contrast to photoreductions by photosystem II, Fig. 3 shows that the basal rate of the bypass reaction with both phenylenediamines is stimulated by uncouplers (no differences were seen between gramicidin and ammonium chloride).

Figures 4 and 5 compare the P/e_2 ratios for the bypass reactions as a function of DBMIB and phenylenediamine concentration, respectively. The P/e_2 ratios are seen to be relatively concentration independent and are about 0.75 for TMPD and range from 0.80 to 1.0 for PD.

Discussion

Figure 6 presents two hypothetical models for the interaction of the electron transport mediators, PD and TMPD, with the chloroplast electron transport chain in terms of the sidedness of the chloroplast membrane. In Fig. 6A, the reduction of the mediators is depicted as

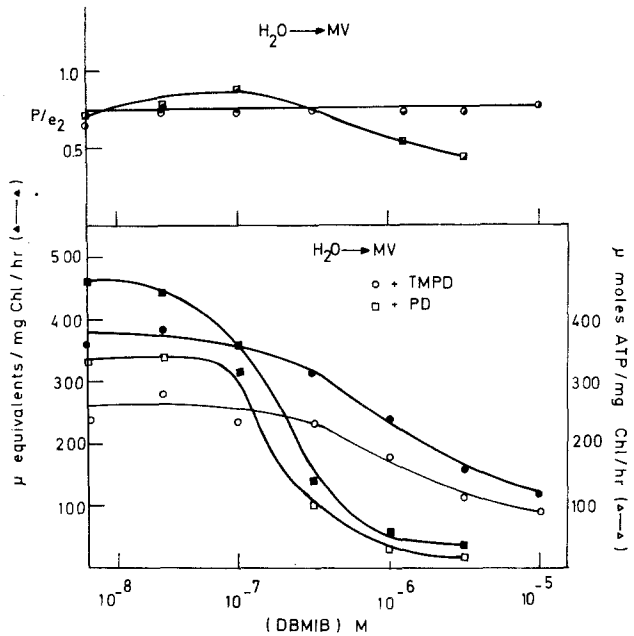


Figure 4. Sensitivity of the phenylenediamine-induced bypass for the reaction water to methylviologen as a function of the DBMIB concentration. Reaction mixtures were prepared and oxygen consumption measured as described in *Materials and Methods*. Samples were illuminated for 3.0 min and the [^{32}P]ATP formed during this time period was determined as described in *Materials and Methods*. The concentrations of TMPD and PD were 80 and 100 μM, respectively.

occurring on the outside of the membrane, while in Fig. 6B the reduction takes place inside. The two models differ only in their predication of the P/e_2 ratios for photoreductions mediated by TMPD as would be expected from the chemistry of N- and C-substituted phenylenediamines [22].

Photoreductions by photosystem II, i.e., ferricyanide reduction, mediated by TMPD and PD differ in their response to uncouplers (Fig. 2). This observation can be explained in terms of the location of the reduction sites. Because PD does not form a stable radical cation, oxidation and reduction reactions of PD involve both protons and electrons. Both species of PD, the diamine and the diimine, are electrically neutral and should be able to penetrate and cross the membrane. If PD were reduced on the inside of the membrane, the proton brought across the membrane by plastohydroquinone would be brought back outside by PD (where PD would again be oxidized by

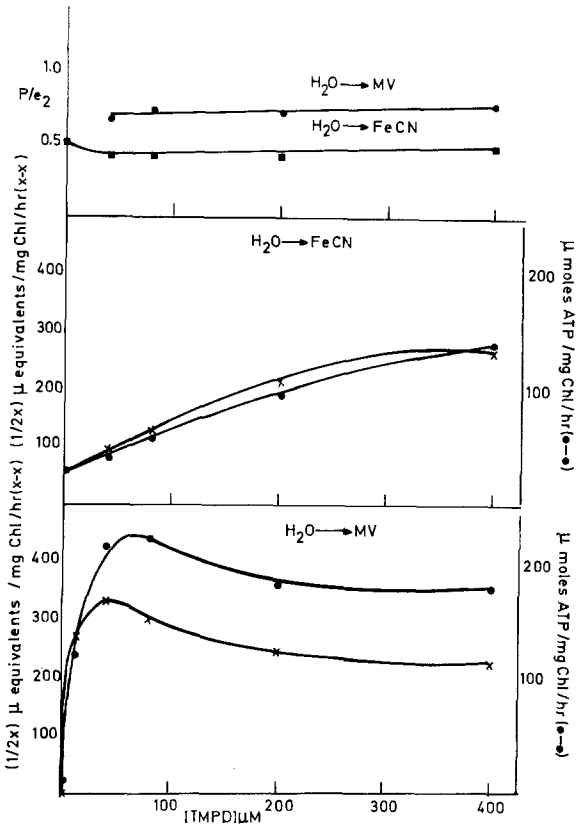
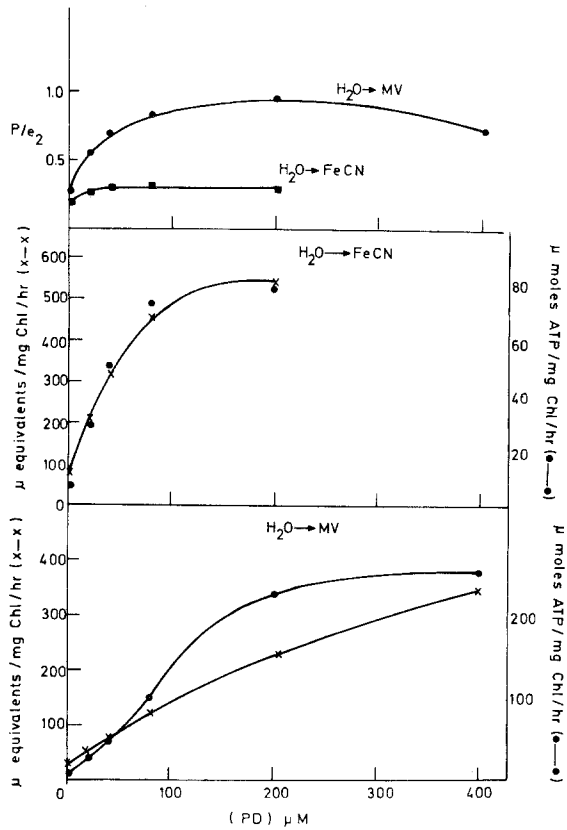


Figure 5 (above and facing page). Phenylenediamine concentration curves for the restoration of coupled ferricyanide reduction and methylviologen-catalyzed oxygen consumption in DBMIB-inhibited chloroplasts. Reaction mixtures were as described in *Materials and Methods.* The concentration of DBMIB was 0.33 μM.

ferricyanide) and the plastoquinone contribution to the pH gradient needed for phosphorylation would be compensated for. This mode of action for lipophilic acceptors has been termed "stoichiometric uncoupling" [16]. In the presence of an uncoupler, the concentration of protons inside the thylakoid would be decreased and the rate of PD reduction correspondingly inhibited.

Reduction of PD could also occur on the outside of the membrane, as indicated by the first model in Fig. 6, and this difference would not be detected by the P/e_2 ratio. However, it then becomes more difficult to understand the inhibition of uncouplers and, therefore, we favor the



interpretation that PD is reduced inside.

Gould and Izawa [23] have suggested, from the DCMU sensitivity of the PD-stimulated ferricyanide reduction, that PD might be reduced before plastoquinone. If this were the case, the electron carrier that reduced PD must also be able to transport protons across the membrane since reduced PD would again bring protons back outside where it would be oxidized by ferricyanide. Other possible proton-transporting candidates aside from plastoquinone are, at this time, not known and we favor, therefore, the interpretation that PD is reduced directly by plastoquinone.

Higher concentrations of PD, which appear to be uncoupler insensitive, might oxidize components closer to the outer surface (that part of the plastoquinone pool which picks up protons from the outside). Under these circumstances, stoichiometric uncoupling would

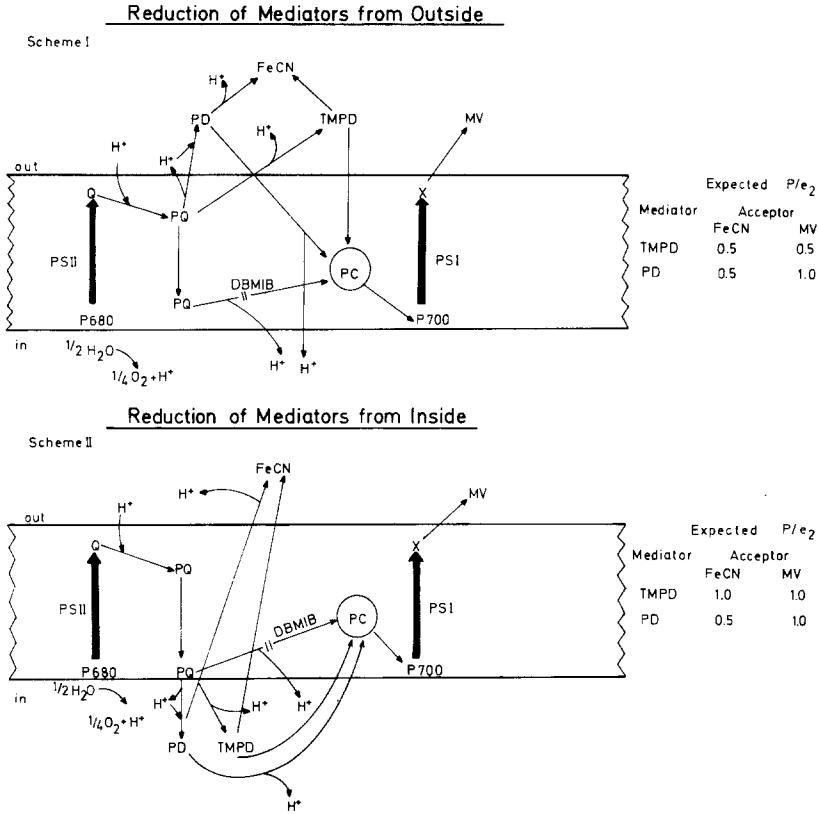


Figure 6. Hypothetical models for the interaction of PD and TMPD with the chloroplast electron transport chain and predicated P/e_2 ratios. See Discussion for details. PSI, PSII, photosystems I and II, respectively; PQ, plastoquinone; PC, plastocyanin; P680, P700, reaction center pigments for photosystems II and I, respectively.

not apply because plastoquinone would not have the opportunity to bring protons across the membrane. The P/e_2 ratio would not be expected to be a function of the PD concentration since either PD brings a proton back outside, as it comes across the membrane, or the proton is never brought across the membrane by plastohydroquinone.

TMPD, upon oxidation, forms a stable radical cation (unlike the uncharged species of the oxidized form of PD). If it were reduced on the inside of the membrane [assuming that the charged species could penetrate the thylakoid membrane which it probably has to do when being used as a photosystem I donor (inside → outside)] by plasto-

hydroquinone, as indicated by the second model in Fig. 6, one would expect that the proton brought across the membrane by plastohydroquinone could be used for phosphorylation and one would anticipate a P/e_2 ratio of approximately 1.0. This is clearly not the case. The P/e_2 ratio is about 0.3–0.4 and suggests that the charged species of TMPD, when it is formed outside the membrane (TMPD being oxidized on the outside by ferricyanide), cannot cross the membrane against the positive membrane potential inside. $TMPD^+$ would then be reduced from photosystem II "outside" (according to the first model in Fig. 6).

On the other hand, *p*-phenylenediamines have the capacity to bypass the DBMIB inhibition site. This has been demonstrated by Trebst and Reimer [15, 16] who measured the reversal of DBMIB-inhibited $NADP^+$ reduction by N-substituted *p*-phenylenediamines. Heathcote and Hall [24] also demonstrated a diaminodurene bypass for the reaction H_2O to methylviologen. Both Trebst and Reimer [15] and Heathcote and Hall [24] have reported that PD is not capable of bypassing the DBMIB inhibition site. We have, however, found that PD induces a bypass, the rate of which depends on the concentration of both PD and DBMIB.

TMPD is more active than PD in reversing the DBMIB inhibition of methylviologen reduction (Fig. 4), although the P/e_2 ratio is generally slightly lower for TMPD than PD (Fig. 5). It is possible, although unlikely, that this difference is due to PD catalyzing a cyclic phosphorylation (competing with methylviologen for electrons at photosystem I). However, another explanation may again be found in the chemistry of the two compounds. Regardless where PD or TMPD are reduced or oxidized (either inside or outside), the protons associated with the oxidation of water would be liberated internally (contributing 0.4–0.5 to the P/e_2 ratio). If PD is the mediator and is reduced at the outside before the DBMIB block (the first model in Fig. 6), the protons associated with the plastoquinone "Mitchellian loop" would be lost. However, PD would move across the membrane and, in the reduced form, would bring a proton inside. This proton would be liberated upon oxidation by photosystem I (contributing another 0.4–0.5 to the P/e_2 ratio, total P/e_2 ratio: 0.8–0.9). When PD bridges the DBMIB block internally (the second model in Fig. 6), the plastoquinone loop would remain intact and the P/e_2 ratio would again be 0.8–0.9. Thus, with PD and other mediators that involve both electrons and protons in a 1 : 1 stoichiometry, it is not possible to determine on which side of the membrane they function in bypassing the DBMIB inhibition site.

The situation is different for TMPD. The reduced uncharged form penetrates the membrane, but upon oxidation by photosystem I cannot liberate protons internally. If TMPD oxidizes plastohydroquinone completely internally (completing the Mitchell loop), the P/e_2 ratio would be the same as that for PD, i.e., 0.8–0.9. If, however, TMPD moves back outside to be re-reduced by plastohydroquinone, the P/e_2

ratio would be 0.3–0.4. A combination of these two modes of action would tend to drop the P/e_2 ratio from 1.0 to about the observed 0.75, suggesting that the second alternative does not operate to a large extent. Therefore, the bypass of the DBMIB inhibition site has to operate in the inside space.

Acknowledgments

I wish to thank Prof. A. Trebst for stimulating discussions throughout the course of this work and am grateful for the skillful technical assistance of Mrs G. Psczolla and for financial support from the Deutsche Forschungsgemeinschaft.

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