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The high-affinity binding site for manganese on the oxidizing side of Photosystem II

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Electron donation to Photosystem II (PS II) by diphenylcarbazide (DPC) is interrupted by the presence of endogenous Mn in PS II particles. Removal of this Mn by Tris treatment greatly stimulates the electron transport with DPC as donor. Binding of low concentration of exogenous Mn(II) to Tris-treated PS II particles inhibits DPC photooxidation competitively with DPC. This phenomenon was used to locate a highly specific Mn(II) binding site on the oxidizing side of Photosystem II with dissociation constant about 0.15 μM. The binding of Mn(II) is electrostatic in nature. Its affinity depends not only on the ionic strength, but also on the anion species of the salt in the medium. The effectiveness in decreasing the affinity follows the order $F^- > SO_4^{2-} > CH_3COO^- > Cl^- > Br^- > NO_3^-$. This observation is interpreted as follows: smaller ions, like F^-, CH_3COO^-, and larger ions, like SO_4^{2-}, have inhibitory effects on Mn(II) binding, whereas ions with optimal size, like Cl^-, Br^- and NO_3^-, can stabilize the binding, resembling the anion requirement for reactivation of Cl^--depleted chloroplasts. We suggest that the binding site for Mn(II) we observed is the site for the endogenous Mn in the O_2-evolving complex of PS II. This site remains after Tris treatment, which removes all the endogenous Mn as well as the three extrinsic proteins, indicating that it is on the intrinsic component(s) of PS II reaction centers. Furthermore, the Cl^- requirement for O_2 evolution may be attributed, at least partly to its stabilizing effect on Mn binding.

Introduction

Although the role of Mn in photosynthetic O_2 evolution has been studied extensively, uncertainties about its mechanism still remain [1–3]. One of the complications is that there are at least three distinguishable pools of bound Mn in chloroplasts. One pool, termed weakly bound Mn, can be removed by EDTA washing or by incubation with a low concentration of divalent cations [4,5]. This pool of Mn is thought to be nonfunctional, because its removal does not affect O_2-evolution activity [6]. There are several treatments on chloroplasts, such as Tris [7,8], NH_2OH [4,9] and heat treatments [6,10], which release another pool of Mn concomitant with loss of O_2-evolving capacity. This pool, classified as strongly bound, is considered to be involved in water oxidation. The third pool, termed very strongly bound, is not removed by the treatments mentioned above. Its function is still not clear. However, it has been shown that O_2 evolution can be reactivated in Tris-treated 'inside-out thylakoid vesicles' [11], suggesting that the pool of very strongly bound Mn is associated with water oxidation.

Abbreviations: Chl, chlorophyll; DPC, diphenylcarbazide; DCIP, 2,6-dichlorophenylindophenol; Mes, 4-morpholineethanesulfonic acid; PS II, Photosystem II; P-680, the reaction center of Photosystem II; Z, the primary electron donor to P-680.

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As for the Mn binding site(s), a 33 kDa extrinsic protein has been found to be closely related to the activity of water oxidation [12,13]. Furthermore, it has been shown that the protein binds Mn when released from thylakoids or PS II preparations by osmotic shock or by a method using butanol in the presence of mild chemical oxidants [14,15]. However, it has also been shown that the 33 kDa protein along with two other extrinsic proteins of sizes 24 and 18 kDa can be washed away (by 1 M $CaCl_2$) without affecting the Mn content of PS II particles [16]. Partial reactivation of O_2 evolution by high concentrations of Cl^- has been demonstrated in these PS II particles [17]. Thus, it appears that the binding site(s) for the functional Mn in PS II is not yet clear.

Cl^- has been shown to be an essential cofactor for O_2 evolution [18]. Its function, however, can be partially substituted by other anions, with effectiveness following the order $Cl^- > Br^- \gg NO_3^- > I^-$. Smaller ions, such as F^- and OH^-, act as competitive inhibitors, whereas larger ions, like SO_4^{2-}, ClO_4^- and PO_4^{3-}, have no effect [19]. It has been suggested that Cl^- participates in the reaction as a ligand bound to the functional Mn, and stabilizes the S-states of the O_2-evolving complex [20,21]. On the other hand, it has also been proposed that Cl^- neutralizes positive charges to produce an active conformation of the O_2-evolving complex [22,23].

Most commonly used electron donors cannot compete with H_2O, the natural donor, but they interact with Z, the primary electron donor to P-680, in Mn-depleted PS II [24]. In this study, we found that the endogenous Mn is the major barrier to electron donation by DPC. We also found that the photooxidiation of DPC by Tris-treated PS II particles which have been depleted of Mn was inhibited by adding low concentrations of exogenous Mn(II). This phenomenon was used to locate a highly specific Mn(II) binding site on the oxidizing side of PS II. The binding of Mn(II) is electrostatic in nature and is influenced by the species of anions present in the medium in a way similar to the anion requirement for reactivation of O_2 evolution in Cl^--depleted PS II. We suggest that the Mn(II) binding site we observed is likely to be the site occupied by the endogenous Mn in the O_2-evolving complex. It is located on the intrinsic component(s) of PS II and the Mn binding on this site is stabilized by the presence of anions like Cl^-.

Materials and Methods

PS II particles from local market spinach were prepared with Triton X-100 as in Ref. 25 and stored in liquid nitrogen. The typical electron-transport activity was about 200 μmol DCIP/mg Chl per h. The PS II particles were washed with 10 mM NaCl/20 mM Mes-NaOH (pH 6.1) before use. For salt treatments, the particles (400 μg Chl/ml) were incubated at room temperature for 15 min in a medium containing 20 mM Mes-NaOH (pH 6.1) and either 1 M NaCl or 1 M $CaCl_2$. For Tris treatment, the particles (400 μg Chl/ml) were incubated in 0.8 M Tris (pH 8.5) for 15 min at room temperature. The three types of treated PS II particle were then washed twice with and resuspended in 15 mM Mes-NaOH (pH 6.1). In some experiments, $CaCl_2$ washed PS II particles were further treated with EDTA. The particles (400 μg Chl/ml) were extracted twice with 15 mM EDTA followed by washing twice with and resuspension in 15 mM Mes-NaOH (pH 6.1). Chlorophyll concentration was estimated according to Ref. 26. All steps were carried out at 0–4°C.

The electron-transport activity of PS II was measured by DCIP reduction at room temperature using a homemade spectrophotometer as described in Ref. 27. The reaction cuvette routinely contained 3 ml medium of 15 mM Mes-NaOH (pH 6.1), 33 μM DCIP and a designated amount of DPC if desired. $MnCl_2$ or $MnSO_4$ was added as inhibitor when indicated. In some experiments, salts of various species were also added to investigate their effect on the inhibition by Mn(II). The pH effect experiments were performed in the same reaction medium, except that Mes-NaOH (pH 6.1) buffer was replaced by Tricine-NaOH (pH 7.7). The PS II particles were added at a chlorophyll concentration of 5 μg/ml. The reduction was monitored by absorbance change at 590 nm and an extinction coefficient of 14.5 $mM^{-1} \cdot cm^{-1}$ was used.

The assay for Mn content was done at room temperature with PS II particles (1 mg Chl/ml) pretreated with 0.5 M HCl. A Bruker ER-200D

EPR spectrometer at 9.74 GHz was used. Instrumental conditions: microwave power, 20 mW; modulation amplitude, 20 G; time constant, 0.5 s; gain, $4 \cdot 10^5$. To estimate the total number of Mn bound at various Mn(II) concentrations, Tris-treated PS II particles (5 μg/ml) were incubated at room temperature for 15 min in a medium containing a designated concentration of $MnCl_2$. The particles were then collected by centrifugation followed by acid treatment. The contribution from unbound Mn, under this condition, was estimated to be less than 1%.

Results

In Table I, it is shown that the isolated PS II particles contained about 6.8 Mn/400 Chl. NaCl wash, which removes two proteins of molecular masses 24 and 18 kDa from PS II particles [28], resulted in no loss of Mn. PS II particles washed by $CaCl_2$ lose an additional 33 kDa protein [16], but still retained almost all the Mn. EDTA extraction after $CaCl_2$ washing removed about 50% of Mn, whereas Tris treatment removed almost all the Mn from the PS II particles.

Since most commonly used reductants cannot compete with H_2O as electron donor to PS II, the small difference in the ability of PS II particles to photoreduce DCIP in the presence or absence of exogenous electron donor DPC indicates that DPC has no access to the reaction center of PS II. A similar phenomenon was observed in NaCl- and $CaCl_2$-washed PS II particles, which retain the normal amount of Mn. Somewhat higher activity of DCIP reduction supported by DPC (as compared with $H_2O \rightarrow$ DCIP) was obtained in $CaCl_2$/EDTA-treated PS II particles which have lost about 50% of Mn, and a dramatic stimulation in such an activity was observed in Tris-treated PS II particles which contain no Mn. It is believed that DPC interacts directly with Z in the absence of endogenous Mn [24]. The close relationship between the Mn content and the ability of DPC to act as an electron donor implies that the endogenous Mn is the obstruction between DPC and the reaction center of PS II. A similar observation was made in Ref. 29 with benzidine as donor.

If a low concentration of exogenous $MnCl_2$ was added to Tris-treated PS II particles, the electron-transport activity from DPC to DCIP decreased accordingly, whereas no inhibition was observed in untreated particles (Fig. 1). Similar results were obtained by using either $MnCl_2$ or $MnSO_4$, indicating the inhibition was due to Mn(II). This inhibition is specific for Mn(II). To achieve 50% inhibition, we found that, for Co(II) or Zn(II), a concentration higher than 20 μM was required, whereas for Mg(II) or Ca(II), hardly any inhibition was observed in the same concentration range. The inhibition was also found to be reversible. Reactivation of DPC to DCIP activity was observed upon decreasing the concentration of Mn(II), implying that the binding of Mn(II) is a reversible process (data not shown).

TABLE I

Mn CONTENTS AND ELECTRON-TRANSPORT ACTIVITY OF PS II PARTICLES SUBJECTED TO VARIOUS TREATMENTS

Treatments on PS II particles, Mn contents and assays for electron-transport activity (in μmol DCIP/mg Chl per h) were carried out as described in Materials and Methods. Assay medium contained 5 μg Chl/ml PS II particles. 15 mM Mes-NaOH (pH 6.1), 33 μM DCIP and 200 μM DPC if desired.

Treatments	Control	NaCl	$CaCl_2$	$CaCl_2$/EDTA	Tris
Mn/400 Chl	6.8	6.3	5.8	3.1	0.65
$H_2O \rightarrow$ DCIP	198	50	6	0	0
DPC → DCIP	205	66	29	68	273

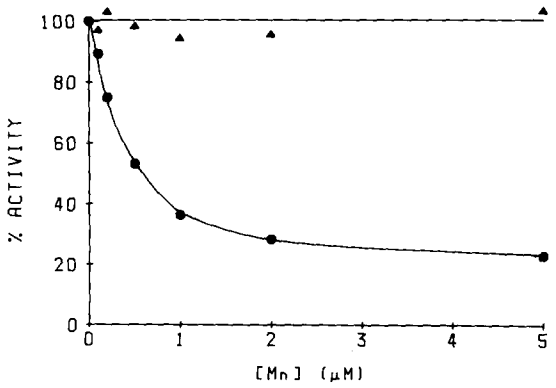

Fig. 1. The effect of Mn(II) on the electron-transport activity from DPC to DCIP of PS II particles. Assays were carried out using Tris-treated (●) or untreated (▲) PS II particles as described in Materials and Methods. Assay medium contained 5 μg Chl/ml PS II particles, 15 mM Mes-NaOH (pH 6.1), 33 μM DCIP, 200 μM DPC and various amounts of $MnCl_2$.

In addition, we investigated the light-intensity dependence of the electron-transport activity (DPC → DCIP) of Mn(II)-inhibited (0.5 μM $MnCl_2$) and uninhibited Tris-treated PS II particles, respectively. As shown in Fig. 2, the activity of Mn(II)-inhibited PS II particles saturated at much lower light intensity than that of uninhibited particles. The result indicates that the inhibition by Mn(II) can be ascribed to a decreased rate at all PS II centers, rather than to complete inhibition in a fraction of the PS II population. Unfortunately, the number of Mn(II) binding at this specific site could not be determined by EPR spectrometer. It was found that addition of 0.5 μM $MnCl_2$, a concentration which will induce about 50% inhibition (see Fig. 1), to Tris-treated PS II particles resulted in a total binding of 7 Mn/400 Chl. The amount of Mn bound increased to 13 Mn/400 Chl when 1 μM $MnCl_2$ was added. The unrealistically large number of Mn(II) binding indicates the presence of other, non-specific Mn(II) binding on PS II particles [30].

In order to examine the mechanism of Mn(II) binding, we used steady-state kinetics to study the type of inhibition by Mn(II). The results are shown in Fig. 3. Fig. 3A is a Lineweaver–Burk plot which shows a competitive interaction between DPC and Mn(II). The Michaelis constant K_m for DPC in the absence of Mn(II) is about 150 μM, as estimated by the intersection of the no-Mn line

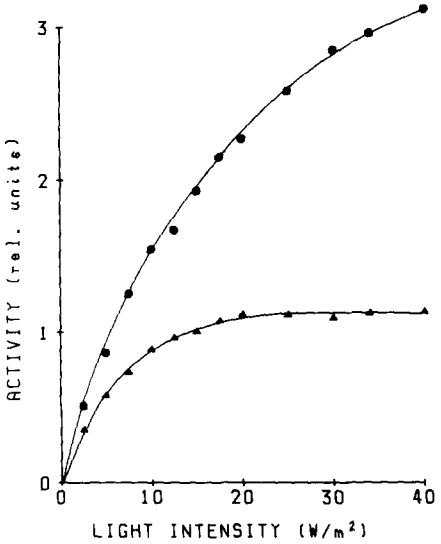

Fig. 2. Light-intensity dependence of the electron-transport activity (DPC → DCIP) of Mn(II) inhibited and uninhibited Tris-treated PS II particles. Activity assays were carried out in the presence (▲) or absence (●) of 0.5 μM $MnCl_2$, as described in Fig. 1.

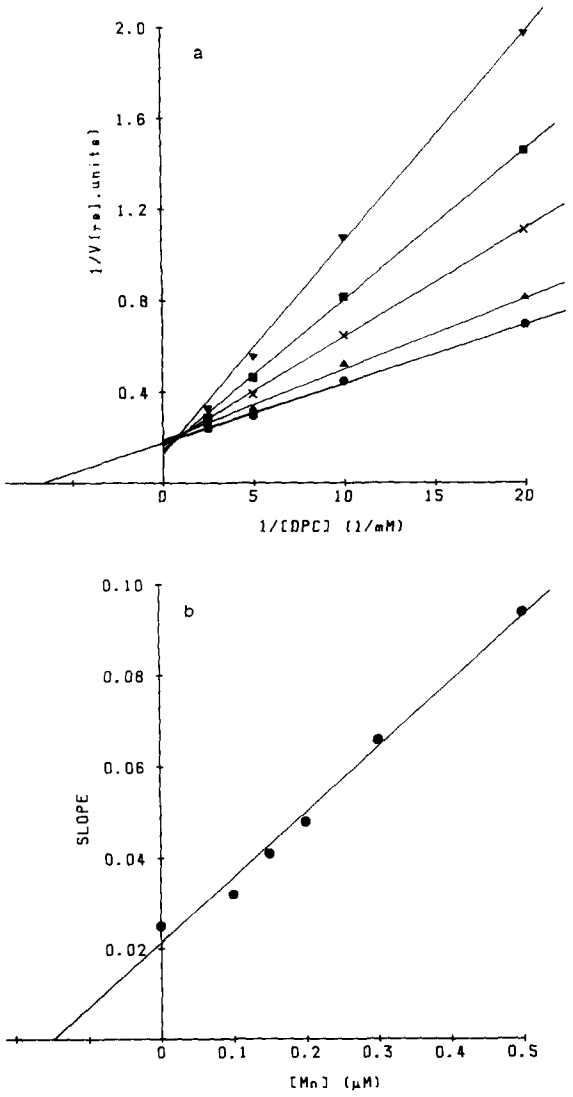

Fig. 3. (a) Lineweaver–Burk plot of Mn(II)-induced inhibition of the electron-transport activity from DPC to DCIP of Tris-treated PS II particles. ●, No $MnCl_2$; ▲, 0.1 μM $MnCl_2$; ×, 0.2 μM $MnCl_2$; ■, 0.3 μM $MnCl_2$; ▼, 0.5 μM $MnCl_2$. (b) Replot of the slope obtained from (a) vs. Mn(II) concentration. Assays were carried out as described in Materials and Methods. Assay medium contained 5 μg Chl/mg Tris-treated PS II particles, 15 mM Mes-NaOH (pH 6.1), 33 μM DCIP and various amounts of DPC and $MnCl_2$.

with the negative X-axis. The slope replot shown in Fig. 3B gives the dissociation constnat K_I of 0.15 μM for Mn(II). A straight line in Fig. 3B indicates that the competition between Mn(II) and DPC is linear, i.e., they compete for the same binding site [31].

The nature of Mn(II) binding was studied further by investigating the salt effect on the binding of Mn(II). We found that the inhibition declined with increasing concentration of NaCl. The weakening of the Mn(II) binding by NaCl was indicated by the increase of the K_I values for Mn(II) along with NaCl concentration as presented in Table II. We therefore conclude that the interaction between Mn(II) and its specific binding site is electrostatic in nature. However, the competitive relationship between Mn(II) and DPC (data not shown) as well as the K_m for DPC were not affected by NaCl (Table II) suggesting that DPC, although competing with Mn(II) for the same binding site, does not bind electrostatically. This is consistent with the non-polar nature of DPC.

Since it has been shown that Cl^- is a special cofactor for the O_2-evolving complex and its function can be partially substituted by several anions like Br^-, NO_3^- and inhibited by F^- [26], we also investigated the effects of various salts on the binding of Mn(II). As shown in Fig. 4, with the ionic strength kept at 50 mM, LiCl and KCl show the same inhibitory effect as NaCl, indicating that the effect is due to the Cl^- anion. Other sodium salts tested had various effects on preventing the binding of Mn(II). The order of effectiveness is $F^- > SO_4^{2-} > CH_3COO^- > Cl^- > Br > NO_3^-$. The K_I values for Mn(II) in the presence of the two most effective anions, F^- and SO_4^{2-}, as well as

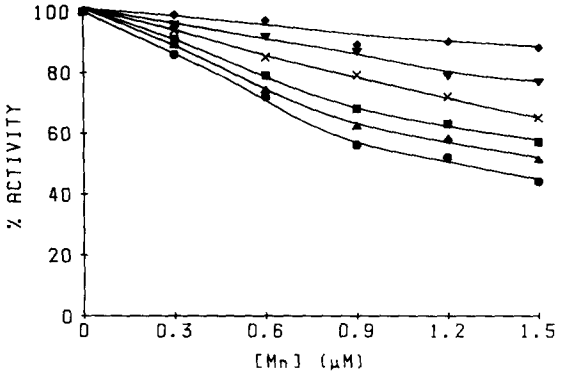

Fig. 4. The effect of various salts on Mn(II)-induced inhibition of the electron-transport activity from DPC to DCIP of Tris-treated PS II particles. The ionic strength of added salts were kept at 50 mM. ●, 50 mM $NaNO_3$; ▲, 50 mM NaBr; ■, 50 mM NaCl or LiCl or KCl; ×, 50 mM CH_3COONa; ▼, 17 mM Na_2SO_4; ◆, 50 mM NaF. Assays were performed as described in Fig. 1. Mn(II) was added as $MnSO_4$.

their corresponding K_m for DPC, are given in Table II. It was found that the competitive relationship between DPC and Mn(II) still remains (data not shown), but the K_I values increase significantly. The results indicate that, in addition to the ionic strength, the species of anions is also an important factor in determining the affinity for Mn(II) binding.

The pH effect on the Mn(II) binding was also studied. We found that, at pH 7.7, Mn(II) became a noncompetitive inhibitor to DPC electron donation (data not shown). Furthermore, the K_I value for Mn(II) (0.55 μ) was about 2.5-fold larger than the corresponding value obtained at pH 6.1, suggesting that high pH, besides altering the manner of inhibition, also weakens the binding of Mn(II).

TABLE II

THE EFFECT OF VARIOUS SALTS ON K_I VALUES FOR Mn(II) AND K_m VALUES FOR DPC DERIVED FROM STEADY-STATE KINETIC EXPERIMENTS

K_m, the Michaelis constants for DPC, and K_I, the dissociation constants for Mn(II), were obtained by using the plotting procedure as described in Fig. 3.

Salt concentration	0 mM NaCl	10 mM NaCl	50 mM NaCl	100 mM NaCl	50 mM NaF	17 mM Na_2SO_4
K_I for Mn (μM)	0.15	0.21	0.53	0.76	4.50	1.36
K_m for DPC (μM)	149	152	163	169	110	118

Discussion

The high-affinity binding site for Mn(II) we observed on the oxidizing side of PS II is very likely to be the binding site for the endogenous Mn in the O_2-evolving complex for the following four reasons.

(1) The binding site is very specific for Mn(II) and the affinity for Mn(II) is high (0.15 μM) at low salt concentration.

(2) Mn(II) at the binding site acts as a competitive inhibitor to DPC electron donation in Tris-treated PS II particles which have been depleted of the endogenous Mn. The competitive relationship can explain the interrupting effect of the endogenous Mn on DPC electron donation to PS II (see Table I), if we assume that the binding of Mn(II) is at the site for the endogenous Mn.

(3) The binding of Mn(II), although electrostatic, is highly dependent on the species of anions in the medium. At the same ionic strength, F^-, SO_4^{2-} and CH_3COO^- are very effective in preventing the binding of Mn(II). In contrast, NO_3^-, Br^- and Cl^- are able to stabilize the Mn(II) binding as compared to F^-, SO_4^{2-} and CH_3COO^-. Qualitatively, these are similar to the findings in the studies of anion requirement for reactivation of the O_2 evolution in Cl^--depleted PS II, where Br^- and NO_3^- can partially substitute for Cl^-; SO_4^{2-} and CH_3COO^- have no effect; and F^- is a strong inhibitor. Fig. 5 shows the percentage of inhibition by 1.5 μM Mn(II) of the electron-transport acativity of Tris-treated PS II particles with the presence of various anions vs. the ion volume. It resembles the plot of reactivation of O_2 evolution by various anions in Cl^--depleted thylakoids vs. the anion volume (Fig. 9, Ref. 19), except that the optimal volume is shifted from 0.25 nm^3 to 0.32 nm^3. Since the percentage of inhibition by Mn(II) in Fig. 5 is a function of the binding affinity for Mn(II), the similar effects of various anions on O_2 evolution and Mn(II) binding suggest that the Mn(II) binding site is likely to be the same for the endogenous Mn in the O_2-evolving complex. As for the shift of the optimal volume, it is probably due to the fact that chloroplasts were used in Ref. 19, whereas here PS II particles were studied.

(4) When the pH was shifted from 6.1 to 7.7,

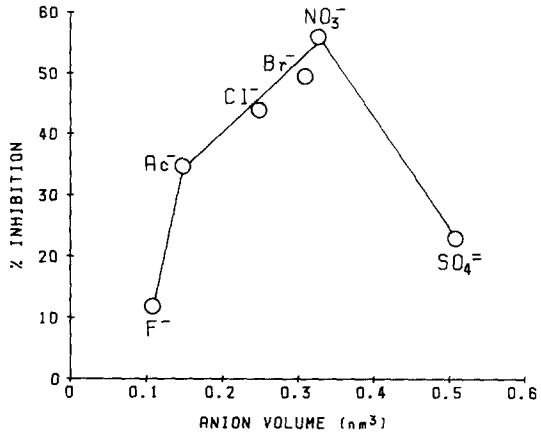

Fig. 5. The percentages of inhibition by 1.5 μM Mn(II) of the electron-transport activity (DPC → DCIP) of Tris-treated PS II particles with the presence of various anions vs. the ion volume. Data of percentage inhibition were taken from Fig. 4. The anion volumes were calculated for a spherical ion from the Pauling radius.

we found that the affinity for Mn(II) was lowered. This is in accordance with the finding that washing PS II particles at high pH results in removal of Mn from the O_2-evolving complex, indicating that the binding of endogenous Mn is destabilized at high pH. In addition, if Cl^- plays a role in stabilizing the binding of Mn(II), then the lowering in Mn(II) affinity at high pH may be attributed to the removal of Cl^-, which is consistent with the finding that OH^- reversibly inhibits O_2 evolution competitively with Cl^- [19,23]. However, the competitive relationship between Mn(II) and DPC disappears upon raising the pH, indicating that high pH not only alters the affinity for Mn(II) but also changes the manner of interacation between Mn(II), DPC and PS II. The mechanism behind it is not yet clear.

If we identify the Mn(II)-binding site with the one for the endogenous Mn in the O_2-evolving complex, then the results of this report have two implications. The first is that the binding site for endogenous Mn is not located on any of the extrinsic proteins removable by Tris treatment, but on the intrinsic component(s) of PS II. This is inconsistent with the suggestion that the extrinsic 33 kDa protein is the Mn-binding protein [14,15]. The second is that the requirement of Cl^- in O_2 evolution probably involves a stabilizing effect of Cl^- on Mn binding. A similar conclusion was

drawn from the observations that when PS II particles are washed with urea/NaCl or $CaCl_2$, the three extrinsic proteins but only a small portion of Mn are released [12,17]. Omission of NaCl during urea wash or incubating $CaCl_2$-washed PS II in low salt medium (10 mM NaCl) leads to the release of 2 Mn/PS II suggesting that Cl^- plays a role in stabilizing the binding of endogenous Mn.

The endogenous Mn bind very tightly to PS II centers with affinity greater than that to EDTA (K_A for Mn-EDTA is 10^{14}); they are classified as strongly and very strongly bound in the three pools of Mn associated with thylakoid membranes (see Introduction). Removal of the three extrinsic proteins (by $CaCl_2$ washing) lowers the affinity for (or increases the accessibility to) 50% of the Mn to a level that allows its extraction by EDTA (see Table I). This probably corresponds to the strongly bound Mn. The remaining 50% of the Mn in our PS II particles can be extracted by Tris washing. For the moment, we do not know whether the Mn(II) binding we observed belongs to the pool of strongly or very strongly bound.

Recently, Tamura and Cheniae [32] also demonstrated the ligation of Mn(II) into the O_2-evolving complex of PS II particles which have been depleted of their endogenous Mn and the three extrinsic proteins. The process, shown by reactivation of O_2 evolution, required light and Ca(II). Our observation on the binding of Mn(II) showed at least two differences from theirs: the first, no Ca(II) was needed and the second, the Mn(II) concentration required to achieve half-maximal effect was much lower (0.5 μM vs. 0.25 mM). The differences may be attributed to the different ways in inactivating the PS II particles (Tris treatment vs. $CaCl_2$ washing plus $DCIPH_2$) and/or in measuring the Mn(II) binding (inhibition on DPC to DCIP vs. reactivation of O_2 evolution).

Klimov et al. [33] also showed that their Tris-washed PS II preparations can be reactivated by adding $MnCl_2$, but at a very low concentration (0.1–0.2 μM in the absence of Mg^{2+} and 0.05–0.1 μM in the presence of Mg^{2+} or other divalent cation of metals). We could not observe any reactivation on our Tris-treated PS II particles. Nevertheless, the K_I for Mn(II) we found at low salt concentration agrees with their value in the absence of Mg^{2+}.

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