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Further characterization of the loss of antenna chlorophyll-binding protein CP43 from Photosystem II during donor-side photoinhibition

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Abstract

The photo-induced reduction in the level of the antenna chlorophyll-binding proteins CP43 and CP47, as well as the reaction-center-forming protein D1, in Photosystem II was studied with PS II membranes and PS II complexes that had been treated with alkaline Tris. The loss of CP43, as detected by sodium dodecyl sulfate/urea polyacrylamide gel electrophoresis, was attributed mostly to specific cross-linking reactions among CP43, CP47 and the D1 protein. The cross-linking of the D1 protein and the α-subunit of cytochrome *b*-559 was also observed. The cross-linking reaction was stimulated by elevated pH and temperature, but it was independent of oxygen. The addition of cations induced a simultaneous decrease in the amount of the product of cross-linking between CP43 and the D1 protein and increase in the amounts of a 38 kDa cross-linking form of the D1 protein and a 16 kDa degradation product of the D1 protein. These results indicate that the cross-linking of the proteins in the PS II complex and the degradation of the D1 protein during photoinhibition are closely related and electrostatically regulated. The cross-linking between CP43 and the D1 protein probably affects the efficiency of degradation of the D1 protein.

Keywords: Photoinhibition; CP43; D1 protein; Reaction center; Photosystem II; Chloroplast

1. Introduction

Photosystem II (PS II) is one of the multi-subunit complexes in thylakoid membranes and catalyzes the reduction of plastoquinone and the oxidation of water to produce molecular oxygen [1]. Despite its functional importance, PS II is a target for photoinhibition [2] and impairment of PS II leads to degradation of the D1 protein that forms the reaction center of PS II. It is known that the D1 protein has a relatively higher turnover rate than other proteins in chloroplasts and the turnover is accelerated by irradiation of PS II [3]. The light-induced degradation of the D1 protein has been examined in vitro in studies with the reaction center and core complexes of PS II [4,5]. Acceptor-side and donor-side mechanisms have been proposed for the photoinhibition of PS II and the degradation of the D1 protein [6]. The primary sites of cleavage of the protein differ depending on the mechanism of photoinactivation [7]. In the case of acceptor-side inhibition, 23 kDa (N-terminal), 16 kDa (C-terminal) and 10 kDa (C-terminal) fragments were detected [8]. By contrast, 24 kDa (C-terminal) and 9 kDa (N-terminal) fragments were identified when the PS II reaction center complex was studied [5], and a 16 kDa (C-terminal) fragment was found in Tris-washed thylakoid membranes and in various preparations of PS II other than the reaction center complex [9]. The photoinhibition on the donor side of PS II occurs with a high quantum yield, while strong light is required for the photoinactivation of the acceptor side [10]. D1 proteins that have been damaged by photoinhibition are cleaved and removed from the PS II complexes. A proteolytic activity within PS II has been shown to be responsible for the cleavage [8]. Salter et al. showed that radiolabeled

Abbreviations: PS II, Photosystem II; D1 and D2 proteins, reaction center-forming proteins of PS II; CP43 and CP47, antenna chlorophyll *a*-binding proteins with molecular masses of 43 kDa and 47 kDa, respectively; Y_Z and Y_D, redox-active tyrosine residues of the D1 protein and the D2 protein, respectively; SDS-PAGE, sodium dodecyl sulfate polyacrylamide gel electrophoresis; PVDF, poly(vinylidene difluoride); Mes, 2-(*N*-morpholino)ethanesulfonic acid; Ches, 2-(cyclohexylamino)ethanesulfonic acid; Tricine, *N*-[tris(hydroxymethyl)methyl]-glycine; Hepes, *N*-2-hydroxyethylpiperazine-*N*'-2-ethanesulfonic acid; Taps, *N*-tris (hydromethyl) methyl-3-aminopropanesulfonic acid; Caps, 3-cyclohexylamino-1-propanesulfonic acid; DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea; Q_A, the secondary electron acceptor of PS II.

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diisopropyl fluorophosphate, which modifies the active site of serine-type proteinase, inhibits the degradation of the D1 protein and is also able to bind to CP43 in PS II [4]. This result indicates that CP43 itself is the proteinase responsible for the degradation of the D1 protein.

We found previously by SDS/urea-polyacrylamide gel electrophoresis that the D1 protein, CP43 and CP47 are lost from the PS II membranes during treatment with Tris in weak light (20 μE m^{-2} s^{-1}) [11]. The loss of the proteins appeared to be caused by donor-side inhibition of PS II since it could be prevented in the presence of artificial donors of electron to PS II. We have now characterized in greater detail the depletion of CP43 during illumination from PS II membranes and from oxygen-evolving complexes of PS II treated with Tris at alkaline pH, and we present data showing that significant cross-linking among CP43, CP47 and the D1 protein is induced under these conditions. Upon addition of cations at a concentration that induces stacking of thylakoid membranes, the levels of cross-linked products were decreased and a 16 kDa fragment of the D1 protein appeared simultaneously. We suggest that cross-linking between the D1 protein and CP43 is closely related to the process of degradation of the D1 protein and that such cross-linking affects the efficiency of generation of degradation products under photoinhibitory conditions.

2. Materials and methods

Membranes of PS II were prepared from spinach thylakoids [12]. PS II complexes that retained the extrinsic 33 kDa protein and manganese were obtained by solubilization of PS II membranes with *n*-octyl β-D-glucopyranoside in the presence of 0.6 M NaCl [13]. For treatment with Tris, PS II membranes and PS II complexes were separately suspended in 0.8 M Tris-HCl (pH 9.0), at 0.5 mg chlorophyll ml^{-1}, incubated on ice for 20 min in darkness, pelleted by centrifugation and washed with either a solution of 0.4 M sucrose, 40 mM Mes-NaOH and 10 mM NaCl (pH 6.5) (SMN), or a solution of 0.4 M sucrose, 40 mM Ches-NaOH and 10 mM NaCl (pH 9.0) (SCN). Each solution also included 2 mM EDTA.

The PS II membranes and the PS II complexes that had been treated with Tris were resuspended in SMN or SCN and the solutions were adjusted to 0.5 mg chlorophyll ml^{-1} and 0.1 mg chlorophyll ml^{-1}, respectively. These preparations were illuminated with weak light (20 μE m^{-2} s^{-1}) as described elsewhere [11]. Anaerobic conditions were achieved by the addition of 10 mM glucose, 0.2 mg ml^{-1} glucose oxidase and 0.2 mg ml^{-1} catalase to the cuvette that contained the suspension of PS II membranes. Silicone oil was layered on the surface of the solution. Lowering the tension of oxygen to the zero level was monitored by an oxygen electrode (Rank Brothers, UK). After incubation at 25°C for 5 min in darkness, the cuvette was illuminated as described above. An aliquot withdrawn from each irradiated suspension was added to an equal volume of a solution of 125 mM Tris-HCl, 6% SDS, 8% β-mercaptoethanol, 6 mM EDTA, 8 M urea (pH 6.8). Proteins in the preparations of PS II were fractionated by sodium dodecyl sulfate/urea-polyacrylamide gel electrophoresis (SDS/urea-PAGE; 10–20% polyacrylamide gradient gel with 6 M urea) [11].

Antibodies were raised against the D1 protein, CP47, CP43 and the D2 protein, which were obtained by preparative SDS-PAGE of reaction center complexes or core complexes of PS II from spinach. The antibody against the α-subunit of cytochrome *b*-559 was a gift from Dr. Bertil Andersson, University of Stockholm. Further purification of the antibodies was performed as described elsewhere [14]. Immunological detection of the proteins by western blotting was performed with an enhanced chemiluminescence system (Amersham, Japan) in accordance with the manufacturer's protocol.

3. Results

When the PS II complexes that had been treated with Tris were exposed to weak light (20 μE m^{-2} s^{-1}) for 30 min at 25°C, reductions in levels of CP43, CP47, the D1 protein and a 10 kDa polypeptide were demonstrated by SDS/urea-PAGE, as reported previously in the case of PS II membranes that had been treated in the same way [11] (Fig. 1). The amounts of these proteins did not change significantly in the control PS II complexes that had been treated by alkaline Tris and then kept at pH 6.5. The PS II complexes are devoid of light-harvesting chlorophyll-protein complexes and, therefore, may have a simpler protein composition than the PS II membranes. In the PS II complexes, a protein band existed just below the D1 protein in the SDS/urea-PAGE gel. The protein, which did not cross-react either with the antibody against the D1 protein or with that against the D2 protein, may correspond to CP29. In our earlier study, we were not able to detect the degradation fragments of CP43 and the D1 protein by western blotting, probably because of the low titer of the specific antibodies used and the low sensitivity of the immunodetection system. In this study, we used newly prepared antibodies and a sensitive immunodetection system to identify the products of the photoinhibition of PS II membranes and of PS II complexes that had been washed with Tris. SDS-PAGE and western blotting of the illuminated preparations of PS II revealed that the antibody against the D1 protein recognized, in addition to the normal D1 band at about 34 kDa, two polypeptides with molecular masses of 41 and 38 kDa, respectively, as well as several polypeptides with molecular masses greater than that of the D1/D2 heterodimer (Fig. 2). No new bands of protein were produced as a result of the illumination that cross-reacted with the antibody against the D2 protein. The

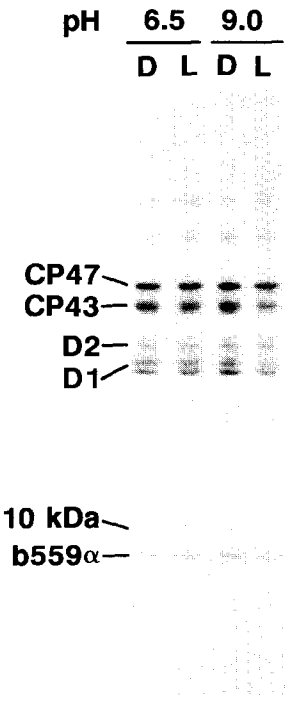

Fig. 1. Photoinduced loss of proteins from PS II complexes that had been treated with alkaline Tris. The PS II complexes, washed with 0.8 M Tris (pH 9.0) and suspended in SMN (pH 6.5) or SCN (pH 9.0; see text for details) at 100 μg chlorophyll ml^{-1}, were illuminated at 20 $\mu E\ m^{-2}s^{-1}$ at 25°C for 30 min. Polypeptides were separated by SDS/urea-PAGE (10–20% gradient gel with 6 M urea) and stained with Coomassie brilliant blue R-250. L, PS II complexes that were illuminated; D, PS II complexes that were kept in darkness (dark control).

polypeptides with higher molecular masses that cross-reacted with the antibody against the D1 protein were also recognized by the antibodies against CP43 and CP47. At least part of the photoinduced loss of CP43 and CP47 from PS II observed under alkaline conditions may be attributable to cross-linking between the D1 protein, CP43 and CP47. In particular, the products of cross-linking between the D1 protein and CP43 seemed to be especially prominent. These results indicate that CP43 is located close to the D1 protein and that the proteins form a heterodimer during photoinhibition.

The 41 kDa polypeptide detected with the antibody against the D1 protein was also recognized by the antibody against the α-subunit of cytochrome *b*-559 (Fig. 2). This 41 kDa polypeptide has been observed regardless of the conditions of photoinactivation [5,8,15], and it has been identified as a product of cross-linking between the uncleaved D1 protein and the α-subunit of cytochrome *b*-559 [16]. The antibody against the D1 protein also recognized a polypeptide of 38 kDa. Since none of the antibodies against the D2 protein, CP43, CP47 and the α-subunit of cytochrome *b*-559 cross-reacted with the 38 kDa protein (Fig. 2), it is possible that an unidentified polypeptide with a molecular mass of 5 kDa in PS II cross-reacts with the D1 protein to produce the 38 kDa polypeptide. The yield of the 41 kDa cross-linking products between the D1 protein and the α-subunit of cytochrome *b*-559, and the yield of the 38 kDa polypeptide were dependent on the preparations of PS II. The 41 kDa band was observed more

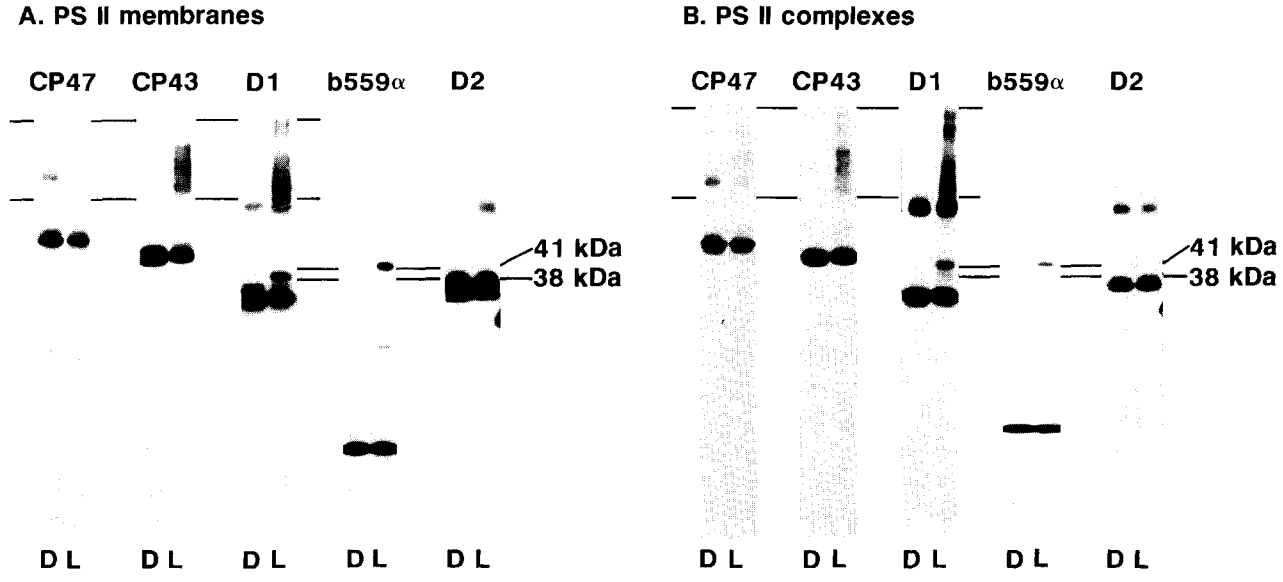

Fig. 2. Detection of products of cross-linking in PS II complexes and PS II membranes that had been treated with Tris and illuminated. PS II membranes (panel A) and PS II complexes (panel B), washed with Tris (0.8 M, pH 9.0) and suspended in SCN (pH 9.0), were illuminated as described in the legend to Fig. 1. After illumination, polypeptides were separated by SDS/urea-PAGE, transferred to PVDF membranes and analyzed immunologically by use of antibodies against D1, D2, CP47, CP43 and the α-subunit of cytochrome *b*-559. D, preparations of PS II that were kept in the dark; L, preparations of PS II that were illuminated.

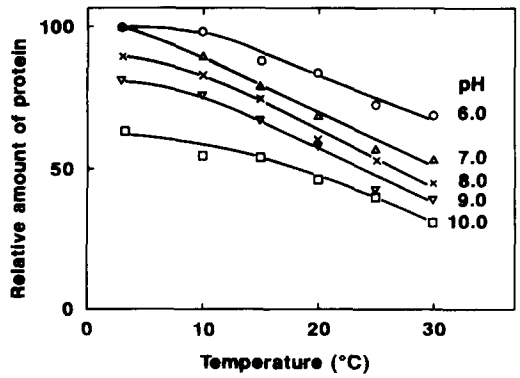

Fig. 3. Effects of pH and temperature on the photo-induced loss of CP43 from PS II membranes that had been treated with Tris. The PS II membranes, after washing with Tris (0.8 M, pH 9.0), were resuspended in buffers at various pH values (0.4 M sucrose, 10 mM NaCl plus one of the following buffers; Hepes (pH 7.0), Taps (pH 8.0), Ches (pH 9.0), or Caps (pH 10.0)) at 0.5 mg chlorophyll ml^{-1} and exposed to weak light (light intensity, 20 $\mu E\ m^{-2}s^{-1}$) at the temperature shown in the figure. Polypeptides were separated by SDS/urea-PAGE, stained with Coomassie brilliant blue R-250 and quantified with a densitometer.

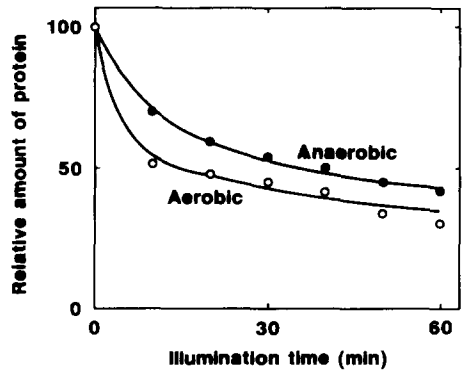

Fig. 4. Time-courses of the photo-induced loss of CP43 under aerobic and anaerobic conditions. PS II membranes, after washing with Tris (0.8 M, pH 9.0) were suspended in SCN (pH 9.0) and illuminated as described in the legend to Fig. 1 at 25°C under aerobic and anaerobic conditions. Samples were withdrawn at the times indicated and immediately solubilized with a solubilization medium for SDS-PAGE. After separation of proteins by SDS/urea-PAGE, the amount of CP43 was quantified densitometrically.

clearly in the PS II complexes, whereas it was easier to see the 38 kDa band in the PS II membranes than in the PS II complexes. At the present stage, we do not know exactly which specific condition in the PS II preparations is responsible for the formation of each polypeptide band. Our preliminary results suggest that the kinetics of appearance of the 38 kDa polypeptide is similar to that of the stacking of the thylakoids (data not shown). It is possible that the presence of LHC II in the PS II membranes regulates the yield of the polypeptide.

The photo-induced loss of CP43 from PS II membranes that had been pretreated with Tris, which is probably related to the cross-linking of CP43 and the D1 protein as described above, depended on both pH and temperature. The amount of CP43 decreased with increases in the pH or the temperature of the suspension of PS II membranes (Fig. 3). It should be noted, however, that loss of CP43 was still observed near 0°C at alkaline pH. This result contrasts with the degradation of the D1 protein by the acceptor-side inactivation of PS II during illumination with strong light. In this latter case, the degradation does not occur at low temperature [15]. The optimum pH for the degradation of the D1 protein by the acceptor-side mechanism is 7.5 [4], and the profile with respect to pH is different from that of the loss of CP43 observed here.

Oxygen is involved in the degradation of the D1 protein in the acceptor-side photoinactivation of PS II [17]. By contrast, the degradation of the D1 protein by donor-side inactivation is independent of oxygen [18]. In the present study, loss of CP43 during photoinhibition was assayed in the presence and in the absence of oxygen. The loss of CP43 occurred even in the absence of oxygen, although the rate of loss was lower than that under aerobic conditions (Fig. 4).

The photo-induced cross-linking of CP43 and the D1 protein observed at alkaline pH in Tris-washed PS II membranes was affected by cations. When $MgCl_2$ was added to the PS II membranes, levels of cross-linked products between CP43 and the D1 protein decreased significantly (Fig. 5). At the same time, a degradation product of the D1 protein appeared, with a molecular mass of 16 kDa; it may correspond to the polypeptide reported

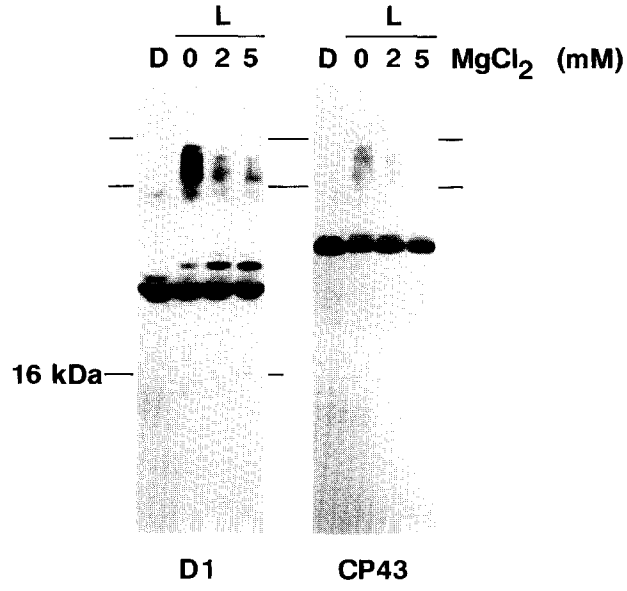

Fig. 5. Effects of cations on the degradation of the D1 protein and on the cross-linking between the D1 protein and CP43 in PS II membranes. PS II membranes, after washing with Tris (0.8 M, pH 9.0), were resuspended in SCN (pH 9.0) that contained $MgCl_2$ (at the concentrations shown in the figure) and exposed to weak light (intensity, 20 $\mu E\ m^{-2}s^{-1}$) at 25°C for 30 min. Polypeptides were separated by SDS/urea-PAGE, electroblotted onto PVDF membranes and analyzed immunologically with antibodies against the D1 protein and CP43. 'D' represents PS II membranes that were kept in darkness.

by Barbato et al. as the degradation product of the D1 protein in donor-side inhibition [16]. As the level of $MgCl_2$ rose, we also noticed that the 38 kDa band became clearly visible in the SDS/urea-PAGE gel. We carried out kinetic analysis of the cross-linking products (the D1 protein-CP43 and the 38 kDa form of the D1 protein) vs. the 16 kDa fragment of the D1 protein at different concentrations of $MgCl_2$ to see the relationship among them. The results showed that the amounts of these products change in parallel fashion (data not shown). The cross-linking of the D1 protein and CP43 may compete with the generation of the 16 kDa fragment of the D1 protein, but the possibility that the 16 kDa fragment was derived from the 38 kDa form can not be excluded. Salts of other monovalent and divalent cations had the same effect as $MgCl_2$, and divalent cations were generally more effective than monovalent cations. For instance, 200 mM NaCl had almost the same effect as 5 mM $MgCl_2$ (data not shown).

4. Discussion

We reported previously the analysis by SDS/urea-PAGE of the loss of CP43, CP47 and the D1 protein from PS II membranes during treatment with Tris in weak light (20 μE $m^{-2}s^{-1}$) [11]. Although the loss of CP43 was the most prominent among these processes, no products of degradation of CP43 were detected. In the present study, we again failed to detect such degradation products, even with a preparation of PS II with a simpler protein composition, antibodies with improved specificity and titer, and a sensitive detection system. Instead, a smeared band was detected over the range of molecular masses from 70 to 150 kDa in the gel after SDS/urea-PAGE (Fig. 2). Since the band cross-reacted with antibodies both against the D1 protein and CP43, we concluded that at least a part of the band represents a product of the cross-linking reaction between the two proteins. When PS II membranes that had been subjected to photoinhibition were treated with lysyl endopeptidase, the smeared band disappeared and new bands with higher mobility appeared in the gel (data not shown). The monomeric D1 protein, which contains no lysine, was unaffected under these conditions. We assume that a cross-linking reaction also occurs between the D1 protein and CP47, or between CP43 and CP47, judging from the cross-reactivity with the specific antibodies, but the D2 protein and the other protein components of PS II seem to be not involved. The relative reactivity of the antibody against the D2 protein may be critical to show that there is no cross-linking products between the D2 protein and other proteins. As both the antibodies against the D1 and D2 proteins cross-reacted with the D1-D2 heterodimer with similar efficiency (Fig. 2 B), we suggest that the titer of the antibody against the D2 protein is high enough to detect the cross-linking products between the D2 protein and other protein components, if they are really present. Prasil et al. discussed the aggregation of the D1 protein and the other proteins of PS II under photoinhibitory conditions [19], and their observation may correspond to the cross-linking observed here.

The details of the putative cross-reactions remain to be elucidated. Cross-linking of the proteins in PS II with chemical cross-linkers that generates heterodimers results in a well defined band after SDS-PAGE. The corresponding molecular mass is exactly equal to the sum of the molecular masses of the respective proteins involved [20]. Since the band observed in our study was diffuse, the products of the cross-linking probably consist of several components that are formed by reactions between the photo-damaged and decomposed fragments of the proteins and/or the photo-damaged but undegraded proteins. The stoichiometry of the proteins involved in the cross-linking is not known.

Both the light-induced cross-linking of the proteins in PS II and the degradation of the D1 protein caused by donor-side photoinactivation of PS II were affected by the ambient temperature. The reactions were retarded when the temperature was low (Fig. 3), suggesting the participation of enzyme-dependent processes. It should be noted, however, that, in contrast to the degradation of the D1 protein, the cross-linking proceeds even at temperatures close to 0°C. A possible explanation is that the temperature-independent cross-linking of the proteins occurs even in the absence of the degradation of the proteins. Cross-linking was observed under both aerobic and anaerobic conditions. Therefore, toxic oxygen molecules, which may be produced by the irradiation of PS II in the presence of oxygen under certain conditions and damage the proteins in PS II, are not directly related to the cross-linking process. The optimum pH differs between the degradation of the D1 protein and the cross-linking reaction. The degradation of the D1 protein was most prominent at pH 7.5–8.0 [4,18], while cross-linking was enhanced with increasing pH (Fig. 3). These results suggest that the degradation of the D1 protein is driven by an enzyme-dependent process, while the cross-linking of the proteins in PS II includes a process that is independent of enzymatic reactions.

Irreversible damage to PS II, which leads to the degradation of the D1 protein, is caused by either the acceptor-side or donor-side photoinhibition of PS II. The photoinduced loss of CP43 (cross-linking between CP43 and the D1 protein) is apparently caused by donor-side photoinhibition of PS II because the loss was prevented in the presence of artificial donors of electron to PS II and by the electron transfer inhibitor, DCMU [11]. The mechanism of donor-side photoinhibition has been studied in detail with PS II membranes that were treated with hydroxylamine [21–23]. Blubaugh et al. indicated that the components on the donor side of PS II are destroyed by weak light and the order of susceptibility of the components to such damage was proposed to be chlorophyll/carotenoid > Y_Z > Y_D ≫ P680, pheophytin, Q_A [22]. Under strong light, emission of

the A_T-band of thermoluminescence from PS II, which reflects the charge recombination of Q_A^- and a putative oxidized histidine residue [23], as well as the photoreactivation of PS II, was rapidly impaired, as compared with the loss of the EPR signals that correspond to Y_Z^+ and Y_D^+ [24]. Thus, illumination of PS II that has been deprived of Mn results in destruction or modification of amino acid residues in the D1 protein and/or in the other protein components of PS II. Preparations of PS II that have been treated with Tris and exposed to weak light are probably inactivated by the same mechanism as the mechanism that is operative upon treatment with hydroxylamine because, in both cases, the membranes are devoid of the catalytic Mn that is required for the evolution of oxygen and are susceptible to donor-side photoinhibition. We assume that the cation radicals, namely, $P680^+$, Y_Z^+ and chlorophyll$^+$, that are produced on the donor side of PS II are responsible for the cross-linking reactions of the proteins observed here. The cross-linking reaction seems to occur in a limited domain of PS II, probably near the site at which cation radicals are produced, at alkaline pH.

The addition of cations to the suspension of PS II membranes induced a decrease in the level of the product of cross-linking between CP43 and the D1 protein and an increase in the level of the degradation product (16 kDa) of the D1 protein and the 38 kDa cross-linking product of the D1 protein as well (Fig. 5). No specific cation was linked to these phenomena, but divalent cations were more effective than monovalent cations. Thus, the effect of cations is apparently not chemical but electrostatic. In general, the addition of salts induces the non-specific aggregation of proteins by shielding their surface charges. By contrast, the cross-linking of the proteins observed here was prevented by the addition of salts. Considering also the enhancement of the cross-linking of proteins at alkaline pH (Fig. 3), we suggest that the cross-linking and the aggregation of the proteins are separate processes. When the cross-linking reaction between the D1 protein and CP43 was prevented by the addition of salts, formation of the 38 kDa form of the D1 protein and degradation of the D1 protein were stimulated. It is well known that the addition of cations to a suspension of thylakoid membranes leads to stacking of the membranes [25,26]. Both the rearrangement of thylakoids and rather localized changes in the PS II complexes may be involved in the regulation by cations of the photo-induced cross-linking and degradation of proteins. Similar effects of cations were also observed in experiments with thylakoid membranes (data not shown), suggesting that these effects are not confined to PS II in vitro. The cross-linking of the proteins in PS II, in particular that between CP43 and the D1 protein and also between the D1 protein and an unknown PS II protein component to form the 38 kDa product, is probably a crucial factor for determining the efficiency of the repair cycle of photodamaged PS II complexes, and it appears to be controlled by electrostatic interactions between related proteins in PS II.

Acknowledgements

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