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Review

Molecular mechanisms of photodamage in the Photosystem II complex ☆

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ABSTRACT

Light induced damage of the photosynthetic apparatus is an important and highly complex phenomenon, which affects primarily the Photosystem II complex. Here the author summarizes the current state of understanding of the molecular mechanisms, which are involved in the light induced inactivation of Photosystem II electron transport together with the relevant mechanisms of photoprotection. Short wavelength ultraviolet radiation impairs primarily the Mn_4Ca catalytic site of the water oxidizing complex with additional effects on the quinone electron acceptors and tyrosine donors of PSII. The main mechanism of photodamage by visible light appears to be mediated by acceptor side modifications, which develop under conditions of excess excitation in which the capacity of light-independent photosynthetic processes limits the utilization of electrons produced in the initial photoreactions. This situation of excess excitation facilitates the reduction of intersystem electron carriers and Photosystem II acceptors, and thereby induces the formation of reactive oxygen species, especially singlet oxygen whose production is sensitized by triplet chlorophyll formation in the reaction center of Photosystem II. The highly reactive singlet oxygen and other reactive oxygen species, such as H_2O_2 and O_2^-, which can also be formed in Photosystem II initiate damage of electron transport components and protein structure. In parallel with the excess excitation dependent mechanism of photodamage inactivation of the Mn_4Ca cluster by visible light may also occur, which impairs electron transfer through the Photosystem II complex and initiates further functional and structural damage of the reaction center via formation of highly oxidizing radicals, such as $P680^{+\bullet}$ and $Tyr\text{-}Z^{+\bullet}$. However, the available data do not support the hypothesis that the Mn-dependent mechanism would be the exclusive or dominating pathway of photodamage in the visible spectral range. This article is part of a Special Issue entitled: Photosystem II.

1. Introduction

The main driving force of photosynthesis is light, which is a highly energetic and potentially dangerous energy source that can damage the photosynthetic apparatus (see Ref. [1]). The light induced decline of photosynthetic activity is referred to as photoinhibition, and this important phenomenon has been a topic of intense research in the last decades. In the photosynthetic apparatus the major site of photoinhibition is the PSII complex whose electron transport is inhibited and protein structure is damaged as a consequence of light exposure (see Ref. [2]). However, PSI can also be damaged by light, especially under conditions when electron transport from PSII becomes limited (see Ref. [3,4]).

The PSII complex, which is embedded in the thylakoid membrane catalyzes the light-driven oxidation of water, evolving one molecule of O_2 and four protons per two H_2O molecules oxidized. PSII consists more than 20 protein subunits, about 36 chlorophyll molecules, protein bound plastoquinone, as well as Mn, Ca, Cl and Fe, which act as redox cofactors of light driven electron transfer [5,6]. The reaction center of PSII is made up by the heterodimer of the D1 and D2 proteins, which bind or contain the redox cofactors involved in light induced electron transport [7]. The 3-dimensional structure of PSII has been determined by X-ray crystallography at 3.5–2.9 Å resolution [5,6,8–10], with a significant recent refinement to 1.9 Å [11], which provides a strong basis for understanding the molecular background of its function and light sensitivity. During light driven PSII electron transfer the primary event is charge separation between the excited reaction center chlorophyll assembly ($P680^{\bullet}$) and the pheophytin (Phe) electron acceptor molecule. This light induced event produces the primary charge separated radical state ($P680^{+\bullet}Phe^{-\bullet}$), which is then stabilized by rapid electron transport processes both at the donor and acceptor sides of PSII. On the acceptor side the electron from reduced Phe is transferred to the first, Q_A, and the second, Q_B, electron acceptors, which are protein bound plastoquinone (PQ) molecules, and then to the mobile pool of PQs in the lipid phase of the thylakoid membrane. On the donor side, $P680^{\bullet+}$ is reduced by Tyr-Z, a redox

Abbreviations: Chl, chlorophyll; DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea; LHC, light-harvesting complex; NPQ, non-photochemical quenching; $^1O_2$, $O_2(a^1\Delta_g)$ configuration of excited singlet state of oxygen; PSI, Photosystem I; PSII, Photosystem II; Phe, pheophytin; P680, reaction center Chl; PQ, plastoquinone; ROS, reactive oxygen species; Q_A and Q_B, the first and second quinone electron acceptors of PSII, respectively; Tyr-Z, tyrosine-Z intermediate electron donor between the water oxidizing complex and P680; UV, ultraviolet

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active tyrosine of the D1 protein, which then extracts an electron from the water-oxidizing complex (for a recent review see Ref. [12] in the current special issue). Water oxidation is catalyzed by a Mn_4Ca cluster, which undergoes cyclic changes of light-induced oxidation. The complex cycles through five oxidation states denoted as S_0,…,S_4 and oxygen is released during the $S_3 \rightarrow S_4 \rightarrow S_0$ transition, in which S_4 is a short-lived intermediate (for reviews see Refs. [13–17]).

Although significant efforts have been devoted to clarify the mechanisms of photoinhibition no consensus has been reached yet, and the molecular background of light sensitivity of PSII has turned out to be an unexpectedly complex story as covered by extensive reviews [1,2,18–27]. This is due to the complexity of events, which underline the photoinhibitory phenomenon and makes it rather difficult, if not impossible to identify a single mechanism that could be generalized to the wide range of light intensity and spectral conditions under which photodamage of PSII occurs. It is clear from the available literature data that the main mechanisms, which directly induce or lead to photodamage, are the production of ROS ($^1O_2$, $O_2^{-\bullet}$, $OH^{-\bullet}$, H_2O_2), and inactivation of the catalytic Mn_4Ca cluster, which leads to the formation of oxidized radicals ($Tyr\text{-}Z^{+\bullet}$, $P680^{+\bullet}$). However, there are significantly different views about the contribution of the above listed mechanisms of photodamage to the actual loss of PSII activity under different experimental conditions. The critical and debated questions are: Can the inactivation of the Mn_4Ca cluster occur only in the UV, or also in the visible spectral range? Does the production ROS induce direct damage of PSII structure and function, or they inhibit only the process of PSII repair? Do changes in the reduction level of intersystem electron carriers affect only the efficiency of PSII repair, or modulate also the rate of photodamage? And finally, does photodamage of PSII proceeds only via a single mechanism under all conditions, or different damage mechanisms exists, which occur simultaneously, but with different efficiency under specific environmental conditions?

Here the author summarizes recent progress in the understanding of the molecular mechanisms of light-induced damage of the PSII complex with the aim to establish consensus among the various models and clarify their validity under different experimental conditions.

2. Photodamage of PSII by UV light

The UV spectral range of solar radiation is an important environmental factor, which has a significant potential for damaging the photosynthetic apparatus. The main targets of UV radiation in photosynthetic organisms are the nucleic acid molecules, the Calvin–Bensson cycle enzymes, as well as the PSII complex (see Ref. [28]). Based on their absorption in the UV range the quinone electron acceptors, the catalytic Mn_4Ca cluster of water oxidation, and the tyrosine electron donors have been suggested as potential target sites of UV-induced damage in PSII (see Refs. [28,29]). Comparison of the absorption spectra of the putative target molecules with action spectra of photosynthetic functions, as well as detailed biophysical characterization of UV damaged PSII complexes regarding the redox function of the putative targets has lead to the consensus that the most important target of UV-B (280–315 nm) light in PSII is the Mn cluster [30–35]. However, additional damaging effects occur at the primary (Q_A) and secondary (Q_B) quinone electron acceptors and the Tyr-D and Tyr-Z electron donors as well [33,36,37]. The damaging mechanism of UV-A radiation (315–400 nm) is very similar to that of UV-B [37,38]. Although the damaging effects of UV-C radiation (220–280 nm) on PSII function have not been characterized at the same detail as those of UV-B and UV-A the Mn_4Ca cluster is most likely the primary target also in the UV-C spectral region. The exact mechanism of UV-induced impairment of the Mn_4Ca cluster is not clear, but it leads to an all or nothing type inhibition of the S-state cycle of water oxidation [31,33,39]. This effect is most likely related to the absorption of UV quanta in the higher valency states of Mn, which leads to the breakup of a bridging ligand between two Mn ions as occurs in a model compound [40]. The involvement of higher valency Mn is supported by the data which show that the water-oxidizing complex is most prone to UV damage in the S_2 and S_3 oxidation states [41] in which the Mn ions are in the Mn(III) and Mn(IV) states, as well as by the similarity of the absorption spectra of synthetic Mn (III) and Mn (IV) containing complexes to the action spectra of UV-induced inactivation of PSII [26,28].

Induction of ROS formation is characteristic also for the UV range and dominated by $OH^{-\bullet}$ in the UV-B [42]. Under exposure to UV-A light singlet oxygen production can also be observed in photosynthetic systems, but the mechanism of its production is different from the sensitization by chlorophyll triplets as occurs in the visible range, and seems to be independent of photosynthetic functions [43].

Among the three UV ranges the shortest wavelength UV-C (200–280 nm) is the most damaging one. However, UV-C radiation has no significant physiological relevance since solar radiation below 280 nm does not reach the surface of Earth due to absorption in the atmosphere. The damaging efficiency of UV-B radiation is ca. 50% higher than that of UV-A [38]. However, the intensity of UV-A in the sunlight at the surface of Earth is significantly higher than that of UV-B. Therefore, the total damaging potential of the UV-A component of sunlight can reach 50 to 75% of the total UV induced damage as shown in Antarctic phytoplankton [44,45], and cultured *Dunaliella salina* [46]. Similar results were obtained with higher plants [47]. However, acclimation of plants via synthesis of UV absorbing compounds can significantly decrease the actual damage caused by UV-A light [47] and in some species UV-A can even be utilized as an additional source of light energy to drive photosynthesis [48,49].

3. Photodamage by visible light

In contrast to the widely accepted consensus view about the primary role of the Mn_4Ca cluster in sensitizing UV-induced photodamage of PSII the situation is significantly more complex in case of visible light (400–700 nm). Modified or impaired function of the Q_A and Q_B acceptors [50,51], inactivation of the Mn_4Ca cluster [35,52], as well as the production of various reactive oxygen species, especially of singlet oxygen via Chl triplet formation [53–55] are equally implicated as significant, and often as exclusive mechanisms of light induced damage. The situation is further complicated by the complex interplay of photodamage, photoprotection, as well as repair of damaged PSII centers, whose balance will eventually determine the net loss of PSII activity [2,24]. Mechanisms of photoprotection and PSII repair have been covered in detail by other reviews in this special issue. Therefore, the author concentrates on the mechanisms of photodamage, which take place independent of PSII repair, and discuss photoprotection only when it helps to clarify a particular damaging mechanism.

3.1. Photodamage related to acceptor side events in PSII

Traditionally, photoinhibitory conditions were expected to occur when the capacity of light independent processes is not sufficient to utilize the electrons produced in the primary photoreactions: this situation creates excess excitation, which leads to reduction of the PQ pool and modifies the functioning of the Q_B [50] and Q_A acceptors [53,56] (Fig. 1). However, more recent models challenged this idea and suggested that photodamage depends only on the total amount of incident visible light, which damages the catalytic Mn_4Ca cluster of water oxidation independent of the presence of excess excitation [35,52].

A large body of literature evidence demonstrates that a series of light induced modifications takes place at the acceptor side of PSII under conditions of strong illumination. A light-induced conformational change has been shown to occur in *Chlamydomonas* cells that slows down the $Q_A^{-\bullet}$ to Q_B electron transfer step and leads to an

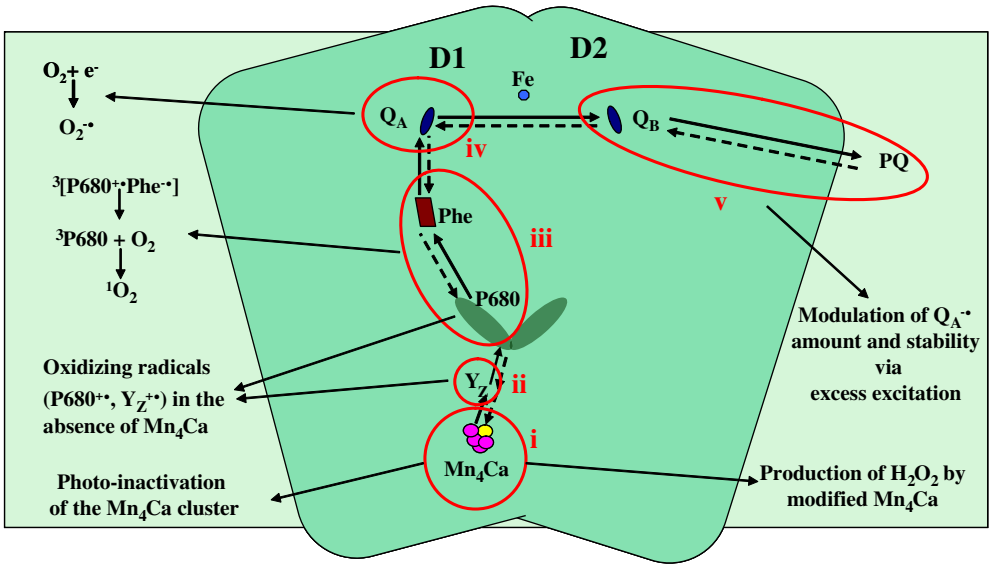

Fig. 1. Sites of photodamage in the PSII complex. The figure shows the electron transport components of PSII, which are involved in light induced damage: (i) The Mn_4Ca cluster of water oxidation is damaged preferentially by UV light, which is absorbed strongly by Mn. However, the Mn absorption extends into the visible range as well. Therefore, visible light has also a potential to damage the Mn_4Ca cluster. Modification of the water oxidizing complex can result in the production of H_2O_2, which is a long lived ROS that acts as precursor of the highly reactive $OH^{-\bullet}$. (ii) In the absence of Mn_4Ca cluster $P680^{+\bullet}$ and Tyr-$Z^{+\bullet}$ ($Y_Z^{+\bullet}$) accumulate. These are highly oxidizing radicals, which damage their protein environment. (iii) Recombination of ^{3}[$P680^{+\bullet}Phe^{-\bullet}$] leads to formation of ^{3}P680, whose interaction with oxygen results in the formation of highly reactive singlet oxygen. ^{3}[$P680^{+\bullet}Phe^{-\bullet}$] can be formed either via spin conversion of the ^{1}[$P680^{+\bullet}Phe^{-\bullet}$] primary charge separated state, or via charge recombination of stable charge separated states. (iv) The reduction state of Q_A is a key factor in the regulation ^{3}P680 formation. In the oxidized state of Q_A stabilization of ^{1}[$P680^{+\bullet}Phe^{-\bullet}$] by electron transfer from $Phe^{-\bullet}$ to Q_A dominates, and consequently the ^{3}P680 yield is low. In the presence of $Q_A^{-\bullet}$ primary radical pair formation is partly suppressed, but not inhibited and the produced ^{1}[$P680^{+\bullet}Phe^{-\bullet}$] can relax only via recombination to P680, or via spin conversion to ^{3}[$P680^{+\bullet}Phe^{-\bullet}$], which yields ^{3}P680. If Q_A is doubly reduced and protonated, or missing from its binding site, forward electron transport is blocked, but primary charge separation is undisturbed. Therefore ^{3}[$P680^{+\bullet}Phe^{-\bullet}$] and ^{3}P680 formation has a high yield. $Q_A^{-\bullet}$ can also serve as reductant for superoxide formation, which is another source of oxidative damage of PSII. (v) Reduction of the PQ molecules in the lipid phase of the membrane and in the Q_B site due to saturation of electron sinks, especially the Calvin–Bensson cycle, is a crucial step in the excess excitation dependent pathway of photodamage. Reduction of the PQ pool and Q_B creates excitation back pressure and stabilizes $Q_A^{-\bullet}$, which leads to enhanced ^{3}P680 and $^1O_2$ formation.

irreversible change of the D1 protein [51]. A modification of the PSII acceptor side at the level of the Q_A to Q_B electron transfer has also been observed in a tobacco mutant, which lacks the key enzyme Rubisco of the Calvin–Bensson cycle due to the deletion of the *rbcL* gene, and is in the state of chronic photoinhibition due to limitation in the lack of final electron sink [57]. Reduction of the PSII acceptor side components is enhanced by anaerobiosis, which made possible to study further mechanistic details of light induced changes at the acceptor side [53,56,58]. These studies revealed that large extent of reduction of the PQ pool leads to a situation in which the binding site of Q_B becomes unoccupied due to the lack of reducible PQ molecules. This leads to the stabilization of $Q_A^{-\bullet}$ [53] (Fig. 1). Under strongly reducing conditions double reduction and protonation of Q_A can also occur followed by the release of Q_AH_2 from its binding site [53,59]. Although the formation of stabilized $Q_A^{-\bullet}$ is an important step of the photoinhibitory process even in the presence of oxygen, double reduction of Q_A has probably a very low yield under physiological conditions. The most important consequence of the changes, which occur at the level of Q_A and Q_B is the blocking of forward electron transport — which is a transient event in case stabilized of $Q_A^{-\bullet}$, and permanent in case of Q_AH_2 formation and release. The blocked or limited forward electron transport facilitates the formation of the triplet excited state of P680 via charge recombination of the ^{3}[$P680^{+\bullet}Phe^{-\bullet}$] state (Fig. 1). The interaction of ^{3}P680 with molecular oxygen leads to the formation of highly reactive singlet oxygen, which damages its protein environment and leads to the actual inhibition of PSII electron transport (see [27,60]), which can be reversed only by D1 protein synthesis dependent PSII repair. Details of the damaging mechanism, which involve the formation of ^{3}P680 and $^1O_2$ are discussed below.

Besides $^1O_2$ other types of ROS, especially superoxide ($O_2^{\bullet-}$) can also be formed at the acceptor side of PSII under illumination. Based on thermodynamic and kinetic limitations $Q_A^{-\bullet}$ appears to be the most likely reductant to O_2 [61] (Fig. 1.). However, under special conditions low yield of $O_2^{-\bullet}$ production could also occur from $Phe^{-\bullet}$, or PQ^- as reductants to O_2 (see Ref. [61]). $O_2^{-\bullet}$ can serve as precursor of H_2O_2 and $OH^{-\bullet}$ formation, which might also contribute to photodamage of the PSII complex (see Ref. [61]). The probability that a reduced PSII acceptor can transfer its electron to O_2 depends on the lifetime of the reduced state, therefore light induced reduction of the electron transport chain under conditions of excess excitation is expected to enhance ROS mediated photodamage.

Although a large body of literature data supports the role of the reduction state of the PSII acceptors in photodamage (see Refs. [2,25,62]) it was proposed recently that limitation of electron transport at the level of electron sinks, especially the suppression of CO_2 fixation inhibits the repair of PSII, but does not affect the rate of photodamage itself (see Ref. [24]). This idea was developed to a model in which photodamage does not depend on the rate of electron transport and the redox state of PSII acceptor components, instead the damage is caused exclusively by the inactivation of the Mn_4Ca cluster by visible light [24,63,64]. The question of excess excitation, or excitation pressure, in the modulation of photodamage has recently been revisited by Konyeyev et al. [65] who concluded on the basis of extensive survey of literature data that parameters derived from excitation pressure correlate with the level of PSII photoinactivation, whereas the amount of incident photons do not. This finding confirms the role of the reduction state of PSII acceptors in the induction of photodamage of PSII and does not support the idea that photo-inactivation of PSII electron transport is caused exclusively by impairment of the Mn_4Ca cluster.

3.2. Photodamage related to triplet Chl formation and singlet oxygen production in PSII

An important consequence of light absorption and following light reactions in PSII is the formation of the triplet excited state of

chlorophylls. This process may occur via intersystem crossing from the singlet excited state of antenna Chls (i.e. ^{1}Chl–>^{3}Chl). Another pathway of triplet Chl formation in PSII is the recombination of the triplet form of the primary radical pair (^{3}[P680$^+$•Pheo$^-$•]–>^{3}P680) as discussed below. The interaction of triplet chlorophylls with molecular oxygen, which also has triplet spin configuration in its ground state, leads to the formation of highly reactive singlet oxygen (see Refs. [27,60,66,67]).

Although the Chls in the light harvesting systems are protected against ^{3}Chl formation by carotenoids, which also act as $^1O_2$ quenchers [68], $^1O_2$ production has been demonstrated in isolated LCHII complexes [69]. The involvement of weakly coupled Chl pigments in photoinhibition PSII has also been shown [70,71]. In addition, destruction of a single Chl in PSII other than P680 has been demonstrated to occur during photoinhibition of PSII with transiently impaired donor side [72].

In contrast to ^{3}Chl formation in the light harvesting antenna, which does not depend directly on electron transport reactions, ^{3}P680 formation is influenced by electron transfer events through excitation back pressure. Therefore, ^{3}P680 plays a dynamic, electron transport regulated role in the photoinhibitory process. After the excitation reaches the PSII reaction center charge separation occurs and the P680$^+$•Pheo$^-$• state is formed in the singlet spin configuration (^{1}[P680$^+$•Pheo$^-$•]). Although ^{1}[P680$^+$•Pheo$^-$•] is rapidly stabilized by acceptor and donor side electron transfer events spin conversion may occur during the lifetime of the primary radical pair, which can convert ^{1}[P680$^+$•Pheo$^-$•] to the triplet ^{3}[P680$^+$•Pheo$^-$•] state, whose recombination results in ^{3}P680 [73]. Backflow of electrons from stabilized charge separated states can also restore the primary radical pair in the triplet configuration without the need for spin conversion of ^{1}[P680$^+$•Pheo$^-$•] [74,75]. The conditions, which have been demonstrated to lead to the formation of ^{3}P680 include the complete lack of Q_A in isolated PSII reaction center particles, double reduction of Q_A under anaerobic conditions [53,58], as well as stabilization of Q_A^-• during illumination [76].

Several lines of experimental evidence (reviewed in Refs. [27,60,67]) shows that singlet oxygen is formed under photoinhibitory conditions, and also that this is the most damaging reactive oxygen species for plant cells [27,77,78]. The importance of singlet oxygen in photoinhibitory processes has also been supported by $^1O_2$ specific lipid peroxidation [77], as well as by photoprotection due to $^1O_2$ quenching compounds, such as α-tocopherol [64,79] and plastoquinone [80,81]. However, the exact role of $^1O_2$ in the process of PSII photodamage has not been fully clarified. Earlier studies indicated that $^1O_2$ can be directly involved in specific damage and cleavage of the D1 and D2 reaction center subunits [82,83]. This idea was further supported by demonstrating the involvement of $^1O_2$ as an intermediate in the triggering of D1 protein degradation [79]. In contrast, from more recent studies in which $^1O_2$ was generated by exogenous sensitizer (Rose Bengal) in the cyanobacterium *Synechocystis* 6803 [63], or by a mutation which eliminates the $^1O_2$ quencher α-tocopherol [64], it was concluded that $^1O_2$ induces net photodamage only by inhibiting the repair cycle of the PSII without damaging directly the PSII complex. In contrast to these findings, direct damage of PSII has been demonstrated in intact tobacco leaves, which were infiltrated with Rose Bengal [84].

An interesting observation shows the enhancement of $^1O_2$ production under low oxygen conditions, which effect is eliminated by ferricyanide [85]. In thylakoids, oxygen functions as electron acceptor of PSI. Therefore, oxygen limitation leads to enhanced reduction of the intersystem electron carrier PQ, and also of PSII acceptors that facilitates charge recombination to produce ^{3}P680. The source of oxygen for $^1O_2$ generation would be the $^3O_2$ evolved by the water oxidizing enzyme in the vicinity of P680 that cannot be fully removed from a thylakoid system. The disappearance of $^1O_2$ in the presence of the electron acceptor ferricyanide confirms that an increased production of $^1O_2$ by ^{3}P680 under low oxygen conditions is caused by the over reduction of the intersystem carriers and supports the role of electron acceptor limitation in singlet oxygen mediated photodamage [85].

^{3}P680 forming charge recombination can occur not only during continuous illumination, but also in the dark after backflow of electrons from stable charge separated states ($S_2Q_A^-$•,$S_2Q_B^-$•, $S_3Q_B^-$•). This mechanism of photoinhibition was first explored by Keren and coworkers [55,86–88], who demonstrated that D1 protein degradation was correlated with the amount of $S_2Q_B^-$• and $S_3Q_B^-$• charge pairs, as well as with the dark interval allowed for charge recombination. PSII activity measurements in isolated thylakoids under flashing light conditions performed by Szilard et al. [89] also showed that the extent of PSII photodamage is most pronounced in the S_2 and S_3 states of the water oxidizing complex, which are capable of charge recombination, and increases with increasing delay time between the subsequent flashes. In addition, the amount of singlet oxygen detected under the same conditions was also increasing with increasing flash spacing, which allow more time for the recombination process [89]. In contrast to these findings Sarvikas et al. [90] did not find a dependence of photoinhibitory damage on the flash interval in lincomycin treated pumpkin leaves. The reasons for the discrepancy between these findings and the earlier data, which were performed by two independent groups, are not fully clear, but most likely related to different ways of assessing photodamage, i.e. initial rate of the photoinhibitory damage in Ref. [90], which required illumination of pumpkin leaves with 1800 flashes, in contrast to total photodamage, which could be detected already after the application of 80–360 flashes both in isolated thylakoids [55,89] and *Chlamydomonas* cells [55]. It is likely that the rate of photoinhibition is not a sensitive measure of photodamage as detection of accumulated damage, therefore the rate measurements might have left the flash interval dependence undetected. It is also possible that the efficiency of the photodamaging process cannot be directly compared in thylakoids or cell suspensions, which are uniformly exposed to light during continuous stirring, and in leaves in which the complex leaf architecture makes the evaluation of the photoinhibitory damages more complicated [91].

An important aspect of PSII photodamage is that its efficiency linearly increases with light intensity even above the saturation of photosynthetic electron transport [92]. It is often argued that among the proposed mechanisms of photoinhibition only direct photodamage of the Mn_4Ca cluster, but not the acceptor side mediated, or charge recombination dependent inactivation of PSII is compatible with the linear light intensity dependence [35,52]. According to the recently extended model of charge recombination dependent and $^1O_2$ mediated model of photodamage [66,67] this is not the case, and the charge recombination model can also explain linear light intensity dependence of photodamage above the saturation level of electron transport. This is because primary charge separation and formation of ^{3}[P680$^+$•Phe$^-$•], which is the precursor of ^{3}P680 and $^1O_2$, occurs also after the saturation of electron transport, either via spin conversion of ^{1}[P680$^+$•Phe$^-$•], or via recombination of P680$^+$•Q_A^-•. Since the rate of primary charge separation in closed PSII centers linearly depends on light intensity, the yield of ^{3}[P680$^+$•Phe$^-$•], and therefore the produced amount of $^1O_2$, as well as the induced photodamage is expected to increase linearly with increasing light intensity even after reaching saturation of photosynthetic electron transfer (see Refs. [66,67] for details).

3.3. Photodamage related to donor side events in PSII

3.3.1. Donor side mediated photodamage in the presence of functionally impaired water oxidizing complex

Increased light sensitivity of PSII in the presence of partly or fully inactivated donor side function is well documented and characterized (see Ref. [2]). Donor-side induced inactivation of PSII can take place under illumination conditions when electron donation from the

Mn_4Ca cluster of water oxidation is unable to keep up with the rate of withdrawal of electrons by $P680^{+\bullet}$. This results in the accumulation of long-lived, oxidizing radicals at the PSII donor side leading to rapid inactivation of electron transport and protein damage. This donor-side induced photoinhibition can be observed in isolated thylakoid membrane systems in which the water-oxidizing complex of PSII is artificially inhibited by the removal of Mn by Tris washing or NH_2OH treatment [93–95], or by the removal of Cl^- [96], and also in isolated PSII reaction center particles which do not contain the Mn_4Ca cluster [97]. Other conditions, which disturb the integrity of the PSII donor side, such as modification of the protein environment around the Mn_4Ca cluster also lead to increased light sensitivity. Increased PSII photoinactivation and D1 protein degradation are also observed in intact systems, which have modified donor side, such as the *Scenedesmus* LF-1 mutant which is unable to evolve oxygen [98], and *Synechocystis* 6803 deletion mutants lacking the PsbO (Mn stabilizing) protein [99]. Single amino acid changes at the C-terminal part of the D1 protein, which inhibit largely or completely the oxygen evolving activity also lead to increased light sensitivity of PSII in *Synechocystis* 6803 [100–103]. The main mechanism of photoinactivation of PSII with impaired electron transport at the donor side is the generation of long lived $P680^{+\bullet}$ and $Tyr\text{-}Z^{+\bullet}$ radicals that could not be reduced in the absence of sufficient electron flow from the Mn_4Ca cluster [93,96,104]. These radicals, especially $P680^{+\bullet}$, have high oxidizing power and induce damage of their protein environment leading to the impairment of electron transport between Tyr-Z and P680 [93,104], and destruction of PSII reaction center proteins [96]. The Mn_4Ca cluster is very sensitive for structural changes at the PSII donor side, e.g. partial release or disturbance of the 17, 23, or 33 kDa peripheral proteins, and its proper function can easily be disturbed even if the cluster is not fully inactivated. Under such conditions electron transfer towards Tyr-Z and P680 can take place, but instead of complete oxidation of water H_2O_2 can be formed [105,106]. H_2O_2 is a precursor of the highly reactive $OH^{-\bullet}$, which can induce protein damage and can contribute to enhanced light sensitivity of PSII with disturbed donor side. A particular condition, which disturbs the integrity of the PSII donor side is heat stress, which leads to partial impairment of water oxidation and significant increase in light sensitivity. According to a recent report ascorbate can act as an alternative electron donor under such conditions, and can provide partial protection against photodamage of heat inactivated PSII under low light conditions [107].

3.3.2. Donor side mediated photodamage in the presence of initially functional water oxidizing complex

The most important question regarding the significance of donor side induced modifications in the photoinhibitory events is whether light itself is able to damage preferentially the Mn_4Ca cluster of water oxidation in initially intact systems. Such light dependent impairment of the Mn_4Ca cluster is clearly induced by UV-B and UV-A irradiation [31,33,34,37]. Light induced inactivation of the Mn_4Ca cluster has also been suggested to occur in the blue and green spectral range of visible light [52]. Based on these data the two-step mechanism of photoinhibition, which was initially put forward to explain the interaction of light in the UV-A/UV-B plus visible range [108], was extended by the proposal that blue and green light can also inactivate the water oxidizing complex, followed by red light driven destruction of the PSII reaction center [52]. The idea of direct photodamage of the Mn_4Ca cluster has been extended even to red light based on the small extent of absorption observed in model Mn compounds [26,35].

Considering that in natural sunlight a significant part of photodamage is caused by the UV photons, especially UV-A, [38,45–47,109], inactivation of the Mn_4Ca cluster by short wavelength UV light is clearly an important pathway of photoinhibition under physiologically relevant conditions. In addition, the models which are based on the direct inactivation of the Mn_4Ca cluster can explain the increased light sensitivity of PSII under conditions of disturbed integrity of the PSII donor side, and also the linear light intensity dependence of photoinhibition. However, the idea that the exclusive mechanism for the inhibition of electron transfer through PSII, i.e. from H_2O to Q_A and Q_B, by visible light is the inactivation of the Mn_4Ca cluster, as suggested by the proponents of the Mn-inactivation mechanism [26,35,52], contradicts a number of well founded experimental observations, which are discussed below.

(i) Earlier studies have shown that under conditions of simultaneous exposure to visible and short wavelength UV-B light, whose ability to impair the Mn_4Ca cluster has been clearly demonstrated, PSII electron transport is inactivated via non-interacting mechanisms. The UV-B and visible spectral ranges affect separate target sites both at the level of oxygen evolving activity and PSII reaction center integrity [108]. As a consequence, there should be a mechanism for the inactivation of PSII electron transfer in the visible range, which does not include the inactivation of the Mn_4Ca cluster. (ii) Singlet oxygen production, which is a common phenomenon under illumination by visible light, is incompatible with the lack of functional Mn_4Ca cluster. When electron transport from the water oxidizing complex is inactive only the Tyr-Z donor is functional, which can transfer only one electron to the acceptor side of PSII. The electron will end up in the PQ pool leaving the reaction center in the $Tyr\text{-}Z^{+\bullet}P680PheQ_A$ state in the majority of the PSII complexes, which is not capable of $^3P680$ forming charge recombination. This idea is fully supported by experimental results, which show the lack of singlet oxygen formation in donor side inhibited PSII [42,110]. In the absence of functional Mn_4Ca cluster singlet oxygen can be formed only in such PSII centers, which lack also Q_A, as occurs in isolated D1D2 reaction center complexes [54], or contain Q_A in the double reduced inactive form. Therefore, the well documented singlet oxygen formation during photoinhibition of initially intact systems demonstrates that inhibition of PSII electron transport occurs in the presence of functional Mn_4Ca cluster. (iii) Inactivation of the Mn_4Ca cluster depends only on light intensity and is not influenced by the reduction state of the PQ pool. This is in contrast with the well documented and recently reconfirmed role of excitation pressure [65] in the photoinhibitory process.

4. Photoprotection mechanisms

All oxygenic photosynthetic organisms possess an array of photoprotection mechanisms, which help to alleviate the harmful effects of light (see Ref. [111] for a recent review). Here these protection mechanisms are discussed with the aim to obtain information about the damaging mechanisms against which they provide protection. The so called non-photochemical quenching (NPQ) mechanisms dissipate absorbed light energy in the antenna system before it can reach the PSII reaction center (see Ref. [112]). These mechanisms include LHCII dependent NPQ, which depends on light induced lumen acidification and mediated by the PsbS protein in higher plants [113], or the LHCSR protein in green algae [114], as well as the phycobilisome dependent NPQ in cyanobacteria, which is mediated by the OCP protein [115]. The common feature of the NPQ mechanisms is that they help to decrease the excitation pressure of PSII and provide protection against electron transport dependent and/or triplet chlorophyll dependent photodamage.

Another strategy for photoprotection is the elimination of the potentially harmful radical states, especially $^3[P680^{+\bullet}Phe^{-\bullet}]$, after they are formed during illumination. This can happen via non-radiative charge recombination, which can be modulated via redox potential changes of Phe and Q_A [66,67,116]. Cyanobacterial species, which live under changing light environment utilize different D1 forms in the PSII reaction center under low and high light conditions [117]. Recent data demonstrate that the light dependent exchange of D1 protein forms is related to optimal photoprotection. In this process the modulation of non-radiative charge recombination via changing the redox potential of Phe through the exchange Gln and Glu residues at the 130th amino acid position in the D1 protein of

plays an important role [116,118–121]. It has to be noted however, that this effect is more pronounced in the mesophylic *Synechococcus* 7942 [118] than in the thermopylic *Thermosynechococcus elongatus* species [120,121].

Charge recombination dependent modulation of photodamage and photoprotection can also be achieved via changing the Q_A and Q_B redox potential. In the presence of bromoxynil, which shifts the redox potential of $Q_A/Q_A^{-\bullet}$ to more negative values $^1O_2$ production and PSII photodamage is increased [122]. Whereas, the opposite effect is observed in the presence of DCMU, which shifts of $Em(Q_A/Q_A^{-\bullet})$ to more positive values [122,123]. The involvement of changing Q_A redox potential in photoprotection has been reported in the cyanobacterium *Synechococcus* PCC7942 [124] and in *Arabidopsis* [125], in which cold acclimation and increased phototolerance are accompanied with decreased redox gap between Q_A and Q_B. This effect was proposed to enhance the non-radiative protective recombination pathway from $P680^{+\bullet}Q_A^{-\bullet}$. Decreasing the redox potential of Q_A in the cyanobacterium *Thermosynechococcus elongatus* [126], and of Q_B in the green alga *Chlamydomonas reinhardtii* [127] by site directed mutaganesis increased the susceptibility to photodamage, most likely by enhancing the ^{3}P680 producing charge recombination pathway as supported by the increased yield of $^1O_2$ production in case of the *T. elongatus* mutant with decreased E_m $(Q_A/Q_A^{-\bullet})$ [126].

Photoprotection of PSII via non-radiative charge recombination reactions has also been demonstrated in the filamentous cyanobacterium *Microcoleus vaginatus* in which a reversible light induced decrease of radiative charge recombination, which might be related to a conformational change in PSII, confers protection against photooxidative stress [128]. Further examples of protective charge recombination pathways occurring in the PSII reaction are reviewed in Ref. [129].

The results concerning the photoprotective effects of redox potential changes at the PSII acceptor side can be easily explained by the decreased efficiency of thermally activated back reaction of $P680^{+\bullet}Q_A^{-\bullet}$ to $^3[P680^{+\bullet}Phe^{-\bullet}]$, which depends on the redox gap between Phe and Q_A, as well as by enhanced non-radiative charge recombination from $P680^{+\bullet}Q_A^{-\bullet}$ and $^1[P680^{+\bullet}Phe^{-\bullet}]$, which also compete with $^3[P680^{+\bullet}Phe^{-\bullet}]$ and ^{3}P680 formation (for details see Refs. [67,130]). On the other hand, it would be very difficult to assume that the redox potential change of either Phe, Q_A, or Q_B could have any direct or indirect effect on the efficiency of visible light to inactivate the Mn_4Ca cluster. Therefore, the photoprotection mechanisms, which utilize redox potential changes of PSII acceptor side components provide strong evidence for the existence of photodamaging mechanism(s) in the visible spectral range, which are related to $^1O_2$ formation but do not involve the inactivation of the Mn_4Ca cluster.

Photosynthetic electron transport is accompanied by the formation of not only singlet oxygen, but also of other reactive oxygen species especially superoxide, which can also mediate photodamage (see Ref. [61]). The overall reaction for scavenging of superoxide is the so called water–water cycle which proceeds through photoreduction of dioxygen to water via superoxide and hydrogen peroxide in PSI by the electrons derived from water in PSII, as reviewed by Ref. [131]. This mechanism of photoprotection is again inefficient against the direct light-induced inactivation of the Mn_4Ca cluster.

The photoprotective mechanisms, which are described above dissipate the light energy, which is absorbed by the light harvesting complexes, or protect against harmful ROS production. On the other hand they have no effect on direct light absorption by the Mn_4Ca cluster, therefore their existence would be an evolutionary nonsense if only the Mn-inactivation based photodamage mechanism would exist. On the other hand, there is apparently no mechanism which could protect the Mn_4Ca cluster against visible light induced inactivation, other than the rather unspecific leaf- and chloroplast movement [111], which decreases overall light intensity inside cells or chloroplasts. This is surprising considering that the Mn_4Ca cluster is well protected against light damage in the UV range by the UV-regulated synthesis of UV absorbing compounds (flavonoids in higher plants and microsporin like amino acids in photosynthetic microorganisms). Therefore, the lack of specific photoprotection against visible light induced damage of the Mn_4Ca cluster may indicate that the physiological significance of this effect is below the threshold that could induce selection pressure for the evolution of relevant protective mechanisms other than the complete repair of damaged PSII complexes.

5. Concluding remarks

As a result of active research during the last three decades important cornerstones of the photoinhibitory phenomenon have been identified. These concern: (i) the increase of photoinhibition under conditions of increased excitation pressure due to limitation of electron sinks (see Ref. [132]). (ii) The involvement of PSII acceptor side components and singlet oxygen in inducing photoinactivation of PSII (see Refs. [27,60]) and photodamage of other cell components (see Ref. [78]). (iii) The involvement of light induced inactivation of the Mn_4Ca cluster of water oxidation in photodamage ([26,28]; (iv) the role of the PSII repair cycle in regulating the overall photoinhibitory damage (see Ref. [2,24]).

The large body of available literature data clearly shows that photodamage of PSII is a complex process in which different mechanisms participate. These damaging events can proceed in parallel with each other. However, their efficiency depends on the environmental conditions, especially the spectral distribution of light. Parallel pathways of PSII photodamage are demonstrated to occur in the UV range. Besides the inactivation of the Mn_4Ca cluster which represents the main pathway of UV-induced inactivation of PSII [26,28], destruction of plastoquinone electron acceptors and tyrosine electron donors also take place [33,36,37].

In the visible range the main damaging mechanisms are the formation of singlet oxygen and other types of ROS, as well as the potential inactivation of the Mn_4Ca cluster due to the weak Mn absorption that extends up to the red part of the spectrum. The main arguments in favor of the $^1O_2$/ROS based mechanism are: (i) clear experimental support for the formation of ^{3}P680 under conditions of limited forward electron transport at the level of Q_A [53,58]. (ii) Detection of $^1O_2$ under photoinhibitory conditions including the correlated occurrence with ^{3}P680 ([110,133] see also Refs. [27,60]). (iii) Correlated changes of $^1O_2$ production and photoinhibitory damage in the absence of PSII repair [84,123,130,134]. (iv) Existence of photoprotection mechanisms, which provide protection by dissipating excess excitation in the antenna system (see Refs. [111,135]), by oxidation of intersystem electron carriers (see Ref. [85]), or by decrease of ^{3}P680 and $^1O_2$ production via modulation of the redox potential of PSII acceptors and non-radiative charge recombination (see Refs. [66,130]). (v) Existence of light regulated $^1O_2$ quenching mechanisms by α-tocopherol [136] and PQ [80,81]. The main argument against the direct damaging role of $^1O_2$/ROS is based on the results, which show that $^1O_2$ inhibits only the PSII repair process, but does not induce functional inactivation of the PSII electron transport in *Synechocystis* cells [63,64]. However, this result does not agree with data obtained in intact tobacco leaves [84] and in isolated PSII membrane particles [137].

The main arguments in favor of the Mn-based photodamage mechanism are: (i) the known inactivation of the Mn_4Ca cluster by UV light (see Refs. [28,29]) combined with the idea that only one mechanism of PSII electron transport inhibition should exist independent of light quality [26,52,92]. (ii) The presence of Mn absorption in the visible (mainly the blue) range combined with the apparent similarity of the action spectrum of photoinhibition with those of synthetic Mn complexes [35,52]. However, the action spectra, which are obtained by different groups show significant differences from

each other and also from the reference Mn spectra [35,52,138,139]. (iii) Release of Mn during photoinhibition [35], which effect however, has been shown earlier to be correlated with D1 protein degradation [140] and can easily be a consequence rather than the cause of photodamage. (iv) The linear light intensity dependence of photodamage above the saturation of photosynthetic electron transport [92], which is claimed to be explainable only by the Mn-inactivation mechanism [35,52].

There are no really strong arguments against the existence of the Mn-based photodamage mechanism in the visible range, although the pro-arguments are based mostly on circumstantial evidence, which indicate the possibility that this mechanism may exist rather than proving its existence. However, there are strong arguments against the suggestion that inactivation of the Mn_4Ca cluster would be the exclusive mechanism of PSII electron transfer impairment by visible light, such as: (i) indication for the existence of different target sites by UV-B and visible light [108]. (ii) Incompatibility of $^1O_2$ production with the lack of electron transport from the Mn_4Ca cluster [110]. (iii) The existence of excitation back pressure dependent modulation of photodamage, which is incompatible with the strictly light intensity dependent inactivation of the Mn_4Ca cluster (see Ref. [65]). (iv) The existence of various photoprotective mechanisms, which protect against excess reduction of the electron transport chain or ^{3}P680 formation, but have no effect on Mn absorption (see Refs. [66,85,111,135]). (v) The claim that only the Mn-based mechanism could explain the linear light intensity dependence of the rate of photodamage [35,52] is clearly a misunderstanding since ^{3}Chl formation in the antenna also linearly dependent on light intensity, and similar feature is expected for ^{3}P680 production [67].

Based on the above discussed arguments it can be safely concluded that inactivation of the Mn_4Ca cluster cannot be the exclusive mechanism of photodamage in the visible spectral range. However, the parallel occurrence of $^1O_2$/ROS mediated and Mn-dependent photodamage mechanisms is a possibility. Unfortunately very few studies have been performed, which could make possible to compare the efficiency of these damaging pathways under identical experimental conditions. This is due to the lack of specific signatures for the occurrence of one or the other type of damaging mechanisms. Preferential inactivation of the Mn_4Ca cluster in the UV range can be easily detected by the appearance of a fast phase in the relaxation of flash induced Chl fluorescence in the presence of DCMU, which arises form charge recombination of the Tyr-$Z^{+\bullet}Q_A^{-\bullet}$ state in the absence of functional Mn_4Ca cluster [141]. However, this signature cannot be observed when photoinhibition is induced by visible light [108], which could be due to the absence, or insignificant extent of Mn-based damage mechanism in the visible range. On the other hand, the lack of accumulation of PSII centers with impaired donor side could also be caused by the much higher quantum yield for the destruction of donor side impaired PSII centers by visible photons as compared to the quantum yield of destruction of centers with intact donor side [104]. Although light induced peroxidation of thylakoid lipids is clearly assigned to $^1O_2$ by mass spectrometry [77], $^1O_2$/ROS induced inactivation of PSII has no clearly identified signatures. In spite of these uncertainties recent studies in which the photoinhibitory effects of different light intensity and spectral regimes were compared provided support for the parallel existence of excess excitation dependent and Mn-based photodamage mechanisms [142,143].

Based on the data, which are summarized here the recently proposed “new paradigms” of photoinhibition, which claim that the exclusive mechanism of inactivation of PSII electron transfer in the visible range is the impairment of the Mn_4Ca cluster [35,52], and also that $^1O_2$/ROS affects only the repair process without damaging the structure and function of PSII [24,63,64] appear to project over simplified views without being sufficiently supported by experimental data.

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