

# How do environmental stresses accelerate photoinhibition?

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**Environmental stress enhances the extent of photoinhibition, a process that is determined by the balance between the rate of photodamage to photosystem II (PSII) and the rate of its repair. Recent investigations suggest that exposure to environmental stresses, such as salt, cold, moderate heat and oxidative stress, do not affect photodamage but inhibit the repair of PSII through suppression of the synthesis of PSII proteins. In particular, production of D1 protein is down-regulated at the translation step by the direct inactivation of the translation machinery and/or by primarily interrupting the fixation of CO<sub>2</sub>. The latter results in the creation of reactive oxygen species (ROS), which in turn block the synthesis of PSII proteins in chloroplasts.**

## Introduction: an excess of light energy enhances photoinhibition

Organisms that perform oxygenic photosynthesis convert light energy into chemical energy (in the form of ATP and NADPH) through the photosynthetic transport of electrons within thylakoid membranes. This energy is then used for the fixation of CO<sub>2</sub> in the Calvin cycle. This process generates organic compounds and molecular oxygen, which are essential for life on earth. The photosynthetic transport of electrons begins with the dehydrogenation of water molecules through a process that uses light energy absorbed by photosynthetic pigments (e.g. chlorophyll and carotenoids). It continues with the reduction of Q<sub>A</sub>, which is a primary acceptor of electrons in photosystem II (PSII). However, when the rate of absorption of light energy by photosynthetic pigments exceeds the rate of its consumption in chloroplasts, the absorbed light energy accelerates the process of photoinhibition [1–3]. Thus, the extent of photoinhibition is enhanced when the consumption of energy during the photosynthetic fixation of CO<sub>2</sub> is limited. Low and high temperatures, drought, and high salinity all strongly limit the photosynthetic fixation of CO<sub>2</sub> and have all been shown to accelerate photoinhibition [1,2,4].

Photodamage to PSII is an indispensable reaction in living organisms that perform oxygenic photosynthesis. To prevent the accumulation of photodamaged PSII, photosynthetic organisms have developed a repair process, which consists of several steps as follows: proteolytic degradation of the D1 protein; synthesis of the precursor to

the D1 protein (pre-D1); insertion of the newly synthesized precursor into the thylakoid membrane concomitant with the assembly of other PSII proteins; maturation of the D1 protein by C-terminal processing of pre-D1; and, finally, assembly of the oxygen-evolving machinery [5,6]. Photoinhibition is, thus, a result of the balance between the rate of photodamage to PSII and the rate of repair.

It was assumed that excess light energy absorbed by photosynthetic pigments was responsible for photodamage to PSII. As such, it was also assumed that environmental stress that limits the photosynthetic fixation of CO<sub>2</sub> would accelerate photodamage to PSII. However, recent studies have revealed that neither interruption of the photosynthetic fixation of CO<sub>2</sub> nor a variety of environmental stresses accelerate photodamage to PSII. In fact, they inhibit the repair of photodamaged PSII. These findings suggest that an excess of light energy absorbed by photosynthetic pigments accelerates photoinhibition through suppression of the repair of photodamaged PSII and not by acceleration of photodamage to PSII [4,7,8]. In this review, we summarize recent progress in our understanding of mechanism of photoinhibition of PSII under environmental stress.

## Rates of photodamage to and repair of PSII

The rate of photodamage can be best studied without the balancing effects of repair. To do this, investigators can create conditions in which repair is inhibited; for example, in the presence of an inhibitor of protein synthesis (e.g. lincomycin or chloramphenicol) [9,10]. The rate of photodamage to PSII, thus determined, is proportional to the intensity of incident light (namely the photon flux density) [11–15], and this is consistent with the two-step model of photodamage [11–15] as described later. Recent studies have revealed that this proportionality is unaffected by environmental stresses, such as oxidative stress [13,16], salt stress [14,17] and cold stress [14,18].

The rate of repair also depends on the intensity of incident light, but it reaches a maximum under relatively weak light [14]. In contrast to photodamage, repair was severely inhibited by oxidative stress [13,14,16], salt stress [14,17] and low-temperature stress [14]. An early study suggested that oxidative stress inhibits the elongation of peptides during the translation step [13,14]. In an *in vitro* study of cyanobacterial translation, hydrogen peroxide oxidized elongation factor G (EF-G), causing it to change conformation and, as a result, inactivate translation [19]. Various studies have demonstrated that exposure of plants to stress conditions increases the intracellular level of

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reactive oxygen species (ROS), which are supposed to inhibit translation of factors required for the repair of PSII. Alternatively, environmental stress, such as salt stress and low temperature, might destabilize the translation machinery, resulting in the inhibition of repair.

### CO<sub>2</sub> fixation involved in the regulation of PSII repair

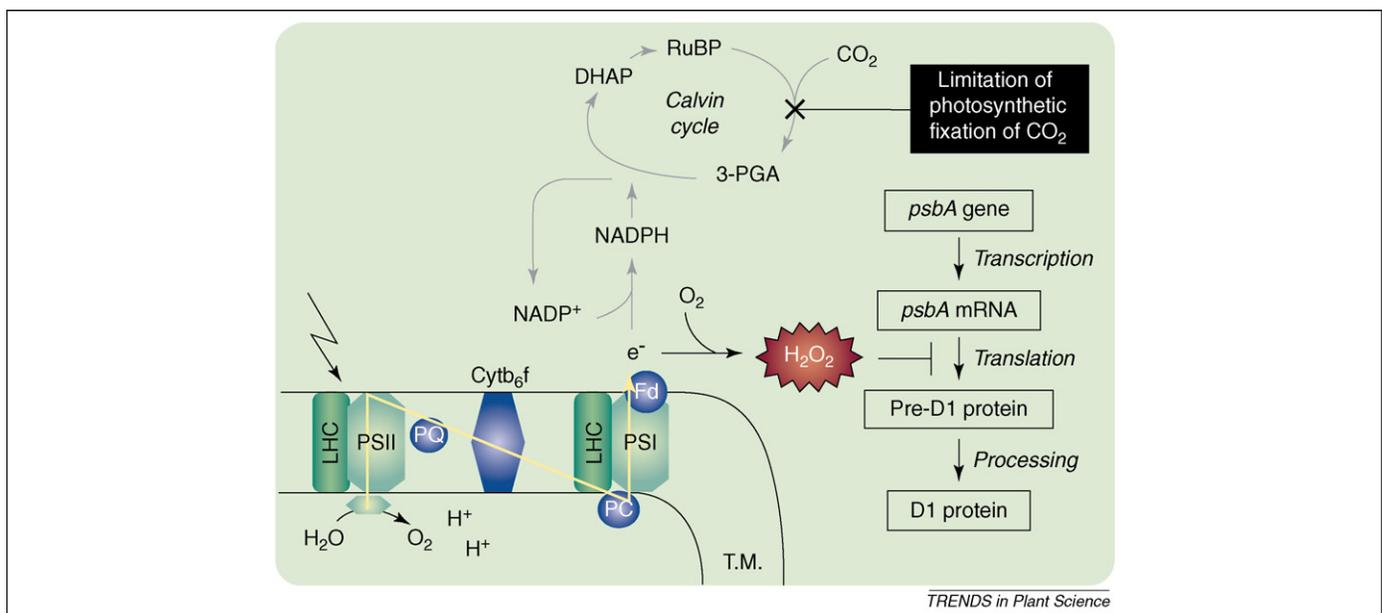
If the inhibition of PSII repair by various types of stress is mediated by a common mechanism, ROS might be involved in such a mechanism. The photosynthetic fixation of CO<sub>2</sub> can regulate the generation of ROS; namely, limitation of the CO<sub>2</sub> fixation decreases the consumption of ATP and NADPH, resulting in an excess of NADPH, especially under strong light. In a recent study in *Chlamydomonas reinhardtii*, applying exogenous glycolaldehyde, an inhibitor of phosphoribulokinase, or generating a missense mutation in the gene encoding the large subunit of ribulose-1,5-bisphosphate (RuBP) carboxylase/oxygenase (Rubisco) interrupted photosynthetic fixation of CO<sub>2</sub>. This failed to accelerate photodamage to PSII but did inhibit the repair of photodamaged PSII [20]. Inhibition of the repair of photodamaged PSII upon interruption of the photosynthetic fixation of CO<sub>2</sub> is attributable to inhibition of the synthesis of PSII proteins – in particular, the D1 protein – at the translation step [20,21].

Figure 1 shows a scheme for the relationship among the transport of electrons, the fixation of CO<sub>2</sub> in the Calvin cycle, and the inhibition of the synthesis of D1 protein. Limitation of the photosynthetic fixation of CO<sub>2</sub> decreases the utilization of NADPH, with a resultant decline in the level of NADP<sup>+</sup>. Given that NADP<sup>+</sup> is a major acceptor of electrons in photosystem I (PSI), depletion of NADP<sup>+</sup> accelerates the transport of electrons from PSI to molecular oxygen with generation of H<sub>2</sub>O<sub>2</sub> via <sup>-</sup>O<sub>2</sub> [22]. Consistent with this proposed scheme, interruption of the photosynthetic fixation of CO<sub>2</sub> accelerates the production of H<sub>2</sub>O<sub>2</sub>

[23–26]. Normally, in chloroplasts, H<sub>2</sub>O<sub>2</sub> is rapidly scavenged through the water–water cycle – namely, the reduction of H<sub>2</sub>O<sub>2</sub> by electrons from PSI [22]. However, if the water–water cycle fails to scavenge all available H<sub>2</sub>O<sub>2</sub>, the remaining H<sub>2</sub>O<sub>2</sub> inhibits the synthesis of PSII proteins – in particular, the D1 protein (Figure 1) [13,21]. Thus, excessive absorbed light energy causes the production of H<sub>2</sub>O<sub>2</sub>, which oxidizes EF-G and thus inhibits the synthesis of D1 protein at the step of elongation [19]. This results in inhibition of the repair of PSII and thus accelerates photo-inhibition [7,8].

Fixation of CO<sub>2</sub> in the Calvin cycle is sensitive to environmental stress [4,27]. For example, high temperatures inactivate Rubisco activase [28–30], which is essential for the activity of Rubisco. Furthermore, the carboxylation reaction catalyzed by Rubisco is also suppressed by increases in temperature, through a decrease in the specificity of Rubisco for CO<sub>2</sub> [31]. Under such environmental stresses, inhibition of the repair of photodamaged PSII upon interruption of the fixation of CO<sub>2</sub> might be expected to accelerate photoinhibition. Consistent with this hypothesis, high-temperature stress (Box 1) [32–34], low-temperature stress [14,33,35] and salt stress [17,36] have all been shown to accelerate photoinhibition by inhibiting the repair of photodamaged PSII. Although these various stresses might inhibit the repair of photodamaged PSII directly, their inhibitory effects might be attributable, in part, to limitation of the fixation of CO<sub>2</sub> in the Calvin cycle.

The photosynthetic fixation of CO<sub>2</sub> occurs through the Calvin cycle and the photorespiratory pathway. When the carboxylation reaction of Rubisco is suppressed, the photorespiratory pathway helps to sustain the photosynthetic fixation of CO<sub>2</sub>. It does so by providing an alternative supply of 3-phosphoglycerate (3-PGA) through the oxygenation reaction of Rubisco



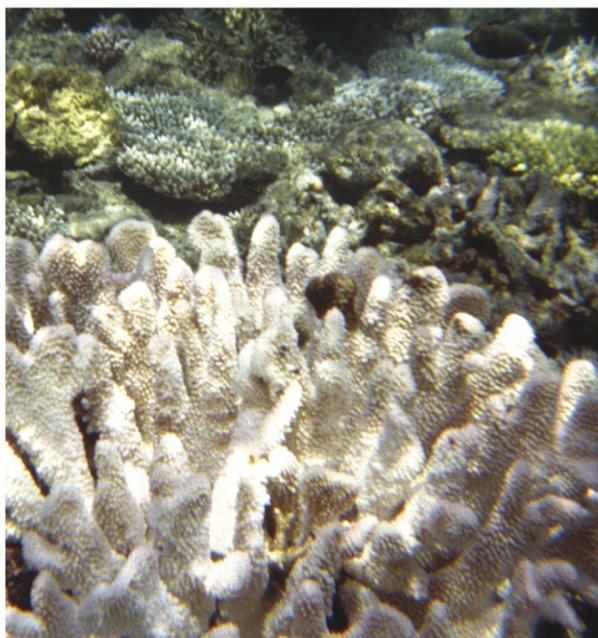
**Figure 1.** A hypothetical scheme for inhibition of the synthesis of the D1 protein upon limitation of the photosynthetic fixation of CO<sub>2</sub>. When the photosynthetic fixation of CO<sub>2</sub> is limited, electrons from PSI tend to be transferred to molecular oxygen. This results in the production of superoxide, which is converted to H<sub>2</sub>O<sub>2</sub> by superoxide dismutase. H<sub>2</sub>O<sub>2</sub> inhibits the repair of photodamaged PSII by inhibiting the synthesis of the precursor to the D1 protein (pre-D1) at the translation step. Abbreviations: cytb<sub>6</sub>f, cytochrome b<sub>6</sub> complex; DHAP, dihydroxyacetone phosphate; e<sup>-</sup>, electron; LHC, light-harvesting complex; 3-PGA, glycerate-3-phosphate; PC, plastocyanin; PQ, plastoquinone; pre-D1 protein, precursor to D1 protein; RuBP, ribulose-1,5-bisphosphate; TM, thylakoid membrane.

### Box 1. Photoinhibition during the bleaching of coral

Reef-building corals are associated with photosynthetic microalgal endosymbionts (i.e. *Symbiodinium* spp.) known as zooxanthellae. Their brownish color is due to their photosynthetic pigments, such as chlorophylls *a* and *c*, and peridinin, a major xanthophyll. In seawater at moderately high temperature, when the mean maximum temperature in summer exceeds the norm by as little as 1°C to 2°C, bleaching of corals occurs as a result of loss of coral pigmentation (see Figure 1). This phenomenon is becoming more frequent with ongoing global changes in climate [53]. Massive bleaching kills corals and destroys their ecosystems. The sensitivity of corals to thermal stress differs among coral species and is consistent with the sensitivity of their endosymbiotic zooxanthella cells to photoinhibition at elevated temperatures [54,55]. Thus, it has been proposed that photoinhibition triggers coral bleaching [53,56].

Photosynthesis in cultured cells of zooxanthellae, which was originally isolated from the jellyfish *Cassiopeia xamachana*, was inhibited to a moderate extent at 30°C and completely at 34°C to 36°C [57]. The depressed photosynthesis in thermally stressed zooxanthellae is attributable to photoinhibition of PSII [54,55]. In zooxanthellae within the coral *Acropora digitifera*, moderate heat stress accelerated photoinhibition by inhibiting the repair of photodamaged PSII and not by accelerating photodamage to PSII [32]. The sensitivity of the repair process to thermal stress differs among coral species [32], suggesting that the sensitivity of zooxanthella cells to thermal stress is strongly influenced by differences in the susceptibility of the repair of PSII to elevated temperature.

Previously, it had been assumed that photoinhibition caused by thermal stress broke down the symbiotic association between zooxanthellae and corals and caused the expulsion of zooxanthellae from their host corals [57]. However, recent experimental evidence suggested that zooxanthella cells that had been expelled from corals were photosynthetically competent [58]. Furthermore, in zooxanthellae within corals, moderate heat stress that caused photoinhibition induced coral bleaching through loss of photosynthetic pigments in zooxanthella cells, but it did not cause expulsion of zooxanthella cells [32]. Therefore, it is likely that photoinhibition is associated with loss of photosynthetic pigments through photobleaching after photoinhibition in zooxanthella but that it is not associated with expulsion of zooxanthellae from corals. However, photoinhibition might eventually cause loss of zooxanthellae from corals, owing to mortality of zooxanthellae, because severe photoinhibition abolishes the photosynthetic production of energy, which is indispensable for survival.



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Figure 1. Bleached coral.

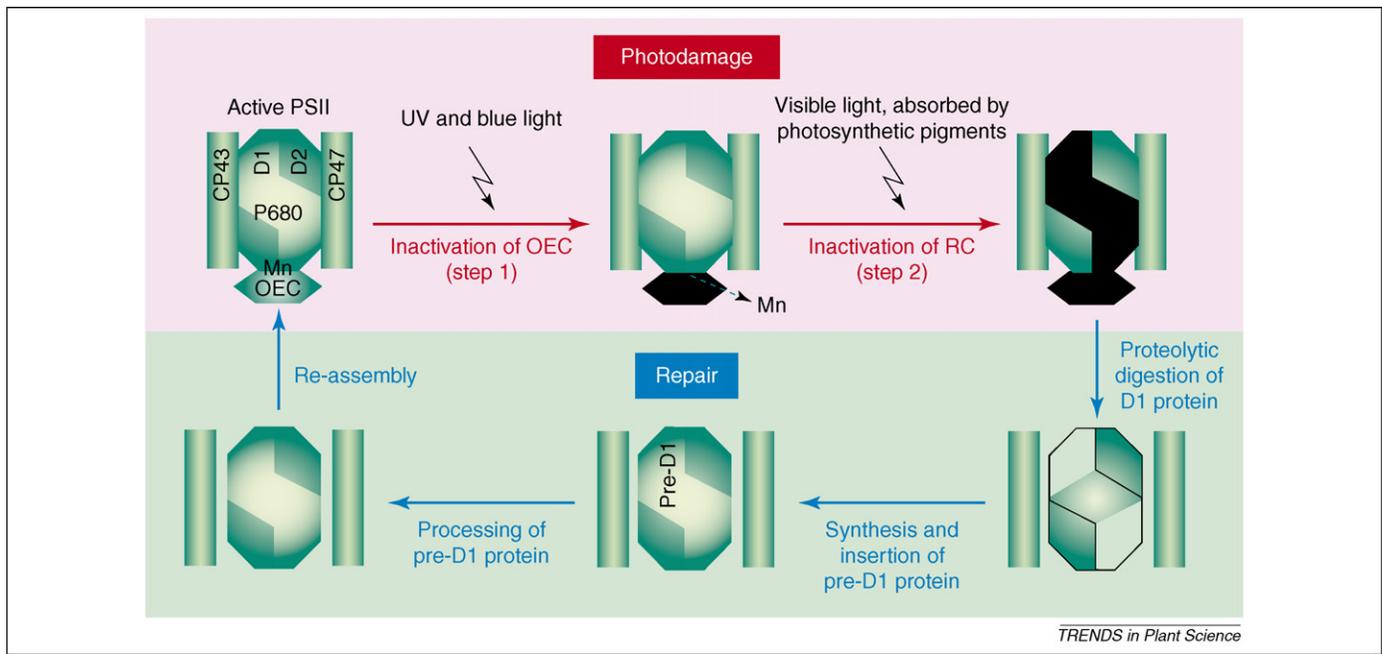
(RuBP→3-PGA + glycolate-2-phosphate) and the recycling of glycolate-2-phosphate to 3-PGA (two molecules of glycolate-2-phosphate produce one molecule of 3-PGA) [37]. Recent experiments have demonstrated that mutations in genes encoding proteins in the photorespiratory pathway accelerate photoinhibition by suppressing the repair of photodamaged PSII, but that they do not accelerate photodamage to PSII [38]. In *Arabidopsis thaliana* mutants in which the photorespiratory pathway was impaired, synthesis of the D1 protein was inhibited at the translation step [38]. These results indicate that an alternative supply of 3-PGA and CO<sub>2</sub> through the photorespiratory pathway helps to mitigate inhibition of the synthesis of the D1 protein, which is essential for the repair of photodamaged PSII [21,38].

### A new model for the photodamage to PSII

Photoinhibition is often observed when the amount of light available for photosynthesis is excessive. Thus, it was assumed that an excess of absorbed light energy would cause photodamage to PSII primarily through acceptor-side photoinhibition [3]. However, recent studies have demonstrated that inhibition of the transport of electrons from Q<sub>A</sub> to Q<sub>B</sub> [39–41] have no effects on the rate of photodamage. This indicates that the rate of photodamage to PSII does not depend on the photosynthetic transport of electrons. It was assumed that the rate of photodamage to PSII is controlled by the redox state of Q<sub>A</sub> [3]. However, the over-reduction of Q<sub>A</sub> upon interruption of electron transport from Q<sub>A</sub> to Q<sub>B</sub> has no effect on the rate of photodamage [39–41]. Thus, photodamage to PSII might not be related to the redox state of Q<sub>A</sub>.

Recent studies of photodamage to PSII have suggested a new mechanism of photodamage, the two-step photodamage model [8,42–44]. This is different from previous models, such as those that invoke acceptor-side photoinhibition. Studies of the effect of monochromatic light on photodamage have suggested that photodamage to PSII occurs in two steps: primary damage by UV and blue light, as well as red light to lesser extents, occurring at the oxygen-evolving complex; and secondary damage by light absorbed by photosynthetic pigments at the reaction center of PSII (Figure 2) [42,43]. The action spectrum of photodamage to PSII is very different from the absorption spectra of chlorophylls and carotenoids [39,43,45–47] but resembles those of model manganese compounds [42,48,49]. Release of manganese ions (Mn<sup>2+</sup>) from the oxygen-evolving complex is accompanied by photodamage to PSII [42,50], suggesting that disruption of the manganese cluster upon absorption of light might be a primary event in photodamage (Figure 2). Inactivation of manganese enzymes, such as Mn superoxide dismutase and Mn catalase, by UV light strongly supports this hypothesis [42,49].

Once the oxygen-evolving complex has been inactivated, the supply of electrons from water to P680<sup>+</sup>, a primary electron donor of PSII, is blocked and, as a result, levels of P680<sup>+</sup> remain high. Given that P680<sup>+</sup> is a strong oxidant [51], high levels of P680<sup>+</sup> might damage the reaction center (Figure 2) [51,52]. Thus, impairment of the oxygen-evolving complex is likely to be the rate-limiting step in the overall process of photodamage to PSII. The rate of



**Figure 2.** A two-step model for photodamage to PSII. Ultraviolet (UV) light and strong blue light primarily inactivate the oxygen-evolving complex (OEC) through the release of manganese (Mn) from the PSII complex. Inactivation of the oxygen-evolving complex prolongs the lifetime of P680\*, the oxidized form of P680, and accumulated P680\* damages the reaction center (RC) of PSII. Photodamaged PSII is repaired through the replacement of PSII proteins – in particular, the D1 protein – by newly synthesized proteins.

photodamage to PSII is proportional to the intensity of incident light, and this is consistent with the two-step model of photodamage [11–15] but not with previous photodamage models. Although the two-step photodamage model has not yet been completely agreed upon, it explains why various kinds of environmental stress and excessive light energy enhance the extent of photoinhibition of PSII.

### Conclusion

The rate of photodamage to PSII is proportional to the intensity of incident light. By contrast, repair of PSII is strongly regulated by light absorbed by photosynthetic pigments. However, under light-excess conditions in which the rate of absorption of light energy by photosynthetic pigments exceeds the rate of consumption of such energy in the cell, the translation of *psbA* mRNA is inhibited as a result of the production of H<sub>2</sub>O<sub>2</sub> [7,8,13]. Thus, under environmental conditions that are unfavorable for the photosynthetic fixation of CO<sub>2</sub>, an excess of light energy absorbed by photosynthetic pigments suppresses the repair of photodamaged PSII and, thus, increases the extent of photoinhibition.

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