Chapter 13

Regulation by Environmental Conditions of the Repair of Photosystem II in Cyanobacteria

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Summary

The activity of photosystem II (PS II) is severely restricted by a variety of environmental factors and, under environmental stress, is determined by the balance between the rate of damage to PS II and the rate of the repair of damaged PS II. The effects of environmental stress on damage and repair can be examined separately and it appears that, while light can damage PS II directly, most types of environmental stress act primarily by inhibiting the repair of PS II. Studies in cyanobacteria have demonstrated that repair-inhibiting conditions include oxidative stress, salt stress, and low-temperatures stress, each of which suppresses the de novo synthesis of proteins, in particular the D1 protein, which is required for the repair of PS II. The synergistic effects of combinations of different types of environmental stress suggest that it is the repair process that determines the sensitivity of PS II to specific environmental conditions.

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I. Introduction

In natural environments, photosynthetic organisms are often exposed to unfavorable environmental conditions, such as strong light, high concentrations of salt, and low and high temperatures. Photosystem II (PS II) is very sensitive to changes in the environment and, under unfavorable or stressful environmental conditions, the activity of PS II declines more rapidly than many other physiological activities (Berry and Björkman, 1980; Demmig-Adams and Adams, 1992; Aro et al., 1993; Andersson and Aro, 2001; and references therein). Since the efficiency of photosynthesis is largely a reflection of the activity of PS II, considerable attention has been paid to the effects of environmental stress on PS II.

Initial studies directed toward an understanding of the mechanisms of the inhibition of PS II by environmental stress suggested that environmental stress might damage PS II directly (Jones and Kok, 1966a,b; Boyer and Bowen, 1970; Jones, 1973; Keck and Boyer, 1974; Björkmann and Powles, 1984; see also reviews by Powles, 1984; Aro et al., 1993; Ohad et al., 1994; Keren and Ohad, 1998; Melis, 1999; Andersson and Aro, 2001; Adir et al., 2003). This conclusion was based on the results of experiments in vitro in which isolated thylakoid membranes or PS II complexes were exposed to the conditions associated with environmental stress. Nonetheless, this approach revealed that damage to PS II was not repaired under the conditions of the experiments. Evidence for direct damage to PS II by environmental stress was also obtained from in vivo studies in which whole organisms were exposed to environmental stress. Again, conclusions were often derived from observations that failed to distinguish the process of damage to PS II from the repair of PS II.

In living photosynthetic cells, PS II is damaged by light and is repaired simultaneously (Kyle et al., 1984; Mattoo et al., 1984; Ohad et al., 1984). The rate of repair of PS II is coordinated with the rate of damage under non-stress conditions but the delicate balance is perturbed under stressful conditions. Environmental stress reduces the rate of repair and, as a result, the activity of PS II decreases. Thus, the activity of PS II that is detected under a given stress is determined by the balance between the rate of damage to PS II and the rate of repair. In order to clarify in full detail the nature of the inhibition of PS II, we must examine separately the effects of environmental stress on damage and on repair. Methods for monitoring the two processes separately have been established (Gombos et al., 1994; Wada et al., 1994) and their application has revealed that light can damage PS II directly, while a variety of other forms of environmental stress act primarily by inhibiting the repair of PS II. This review provides a summary of recent progress in this area and focuses on the elucidation of the mechanisms responsible for the regulation by environmental factors of the repair of PS II.

II. Effects of Light

A. Photodamage and Repair of PS II

Light is a prerequisite for photosynthesis but it is harmful to the photosynthetic machinery. Exposure of photosynthetic organisms to strong light results in severe inhibition of the activity of PS II (Powles, 1984; Aro et al., 1993; Ohad et al., 1994; Melis, 1999; Andersson and Aro, 2001; Adir et al., 2003). This phenomenon is referred to as photodamage to PS II or the photoinhibition of PS II. Although full details of mechanisms responsible for the photodamage to PS II remain unclear, there is general agreement that the primary target of photodamage is the photochemical reaction center. It is hypothesized that a primary event in photoinhibition causes damage to the D1 protein, which triggers the rapid degradation of the damaged D1 protein by several proteases (Prásil et al., 1992; Aro et al., 1993; Andersson and Aro, 2001).

In living photosynthetic cells, a system exists for the repair of photodamaged PS II (Aro et al., 1993; Andersson and Aro, 2001). The damaged D1 protein is replaced by a newly synthesized precursor to the D1 protein, which is encoded by the *psbA* gene (Ohad et al., 1984; Mattoo et al., 1984, 1989). The carboxy-terminal region of the precursor protein is removed by specific proteases (Anbudurai et al., 1994; Inagaki et al., 2001) and PS II is reactivated.

B. Dissection of Photodamage and Repair

Photosystem II normally undergoes photodamage and repair simultaneously in living cells. Thus, if we want to monitor the process of damage exclusively, it is necessary to block the repair process by exposure of cells to an inhibitor of protein synthesis, such as chloramphenicol or lincomycin. Figure 1A shows typical kinetics of the photodamage to PS II in the cyanobacterium *Synechocystis* sp. PCC 6803. Transformable cyanobacteria are useful model organisms for studies of the effects of environmental stress on photosynthesis. The incubation of *Synechocystis* cells for 2 h in light at