Chapter 12

Photoinhibition and Recovery in Oxygenic Photosynthesis: Mechanism of a Photosystem II Damage and Repair Cycle

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Summary

This Chapter provides highlights on the mechanism of a photosystem II (PS II) damage and repair cycle in chloroplasts. Photo-oxidative damage to the PS II reaction center is a phenomenon that occurs in every organism of oxygenic photosynthesis. Through the process of evolution, an elaborate repair mechanism was devised, one that rectifies this presumably unavoidable and irreversible photoinhibition and restores the PS II charge separation activity. The repair process entails several enzymatic reactions for the selective removal and replacement of the inactivated D1/32 kD reaction center protein (the chloroplast-encoded *psbA* gene product) from the massive (>1,000 kD) H_2O-oxidizing and O_2-evolving PS II holocomplex. This repair process is unique in the annals of biology; nothing

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analogous in complexity and specificity has been reported in other biological systems. Elucidation of the repair mechanism may reveal the occurrence of hitherto unknown regulatory and catalytic reactions for the selective in situ replacement of specific proteins from within multi-protein complexes. This may not only have significant applications in photosynthesis and agriculture but also in medicine and other fields.

I. Introduction

Life on earth is sustained by oxygenic photosynthesis, a process that begins with the utilization of sunlight for the oxidation of water molecules. The chemical energy stored in this endergonic oxidation is processed through the electron-transport chain of the chloroplast thylakoids and is eventually delivered in the form of reductant (reduced ferredoxin) and high-energy phosphate bond (ATP). The absorption of light and the conversion of excitation energy to chemical energy take place in photosystem II (PS II) and photosystem I (PS I) in the thylakoid membrane (Hill and Bendall, 1960; Duysens et al., 1961). Light energy in PS II specifically facilitates the generation of a strong oxidant capable of oxidizing water molecules. The ability of PS II to utilize water molecules from which to extract electrons and protons was undoubtedly a significant event in the evolution of life on earth. It contributed to the gradual accumulation of oxygen in the atmosphere, thereby permitting the evolution of oxidative phosphorylation. Rightfully so, many scientists refer to PS II as '*the engine of life on earth*'.

From the biochemical point of view, PS II is a specialized H_2O-to-plastoquinone oxidoreductase. This enzyme features a rather sizable holocomplex consisting of more than 25 transmembrane and peripheral proteins. Most of the transmembrane proteins function as chlorophyll-protein light-harvesting complexes. The functional center of the holocomplex is the so-called D1/D2 heterodimer reaction center proteins that perform the light utilization, water oxidation, and primary electron transport reactions in PS II. These highly specialized functions of PS II take place in a protected and isolated microenvironment where oxygen abounds and where photons, in the form of excitation energy, are received by the photochemical reaction center at a rate of up to 5,000 per second. The transient formation of strong oxidants, the abundance of oxygen, and the presence of excitation energy are conditions that may lead to photo-oxidative damage (Barber, 1994). Indeed, such photodamage occurs frequently within the reaction center of PS II. It causes an irreversible inactivation in the PS II electron transport and stops photosynthesis (Powles, 1984).

Through the process of 2–3 billion years of evolution, organisms of oxygenic photosynthesis have not been able to either prevent or avoid this photo-oxidative adverse effect from occurring (Payton et al., 1998). Thus, to date, every oxygen-evolving photosynthetic organism known, from cyanobacteria to C4 plants, is subject to this irreversible photodamage. Nature, however, devised a repair mechanism that restores the functional status of PS II. The PS II damage and repair cycle, as the phenomenon has come to be known (Guenther and Melis, 1990), is of great importance for the maintenance and productivity of photosynthesis. In repair-aberrant mutants, oxygenic photosynthesis cannot be sustained (Zhang et al., 1997). Clearly, life on earth would have been quite different in the absence of the PS II repair process.

The objective of this article is to present highlights on the biochemical and molecular basis of the PS II damage and repair cycle. It examines specific reaction steps of the PS II repair process in chloroplasts and known mechanisms for the regulation of this phenomenon at the molecular and membrane levels. A more in-depth analysis of the PS II structure, function, photodamage, and repair is given below.

II. Photosystem II (PS II) Organization

All PS II electron transport intermediates are contained within the so-called D1/D2 32/34 kD heterodimer protein, coded for by chloroplast genes *psb*A and *psb*D, respectively (Barber et al., 1987; Nanba and Satoh, 1987; Seibert et al., 1988; Deisenhofer and Michel, 1989). Structural information on the architecture of the D1/D2 heterodimer complex at 3.8 Å resolution (Zouni et al., 2001) has shown how each protein binds one of the photochemical reaction center chlorophylls that form P680, one pheophytin, and one quinone binding site. D1 contains the plastoquinone (Q_B) binding site,

Abbreviations: Chl – chlorophyll; HL – High light; LHC – light-harvesting complex; LL – Low light; PS II – photosystem II; PS II-RC – PS II reaction center; D1 – the 32 kD PS II-RC protein; D2 – the 34 kD PSII-RC protein.