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Review

Photosynthetic light reactions – An adjustable hub in basic production and plant immunity signaling

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ABSTRACT

Photosynthetic efficiency is a key trait that influences the sustainable utilization of plants for energy and nutrition. By now, extensive research on photosynthetic processes has underscored important structural and functional relationships among photosynthetic thylakoid membrane protein complexes, and their roles in determining the productivity and stress resistance of plants. Photosystem II photoinhibition-repair cycle, for example, has arisen vital in protecting also Photosystem I against light-induced damage. Availability of highly sophisticated genetic, biochemical and biophysical tools has greatly expanded the catalog of components that carry out photoprotective functions in plants. On thylakoid membranes, these components encompass a network of overlapping systems that allow delicate regulation of linear and cyclic electron transfer pathways, balancing of excitation energy distribution between the two photosystems and dissipation of excess light energy in the antenna system as heat. An increasing number of reports indicate that the above mentioned mechanisms also mediate important functions in the regulation of biotic stress responses in plants. Particularly the handling of excitation energy in the light harvesting II antenna complexes appears central to plant immunity signaling. Comprehensive understanding of the underlying mechanisms and regulatory cross-talk, however, still remain elusive. This review highlights the current understanding of components that regulate the function of photosynthetic light reactions and directly or indirectly also modulate disease resistance in higher plants.

1. Introduction

Photosynthesis is a highly adjustable metabolic process that enables plant productivity for food, feed and energy under a wide range of climatic conditions. However, unforeseen changes in weather conditions that are often combined with infestation by pathogens or pests may, particularly in monocultural farming, lead to significant decreases in photosynthetic activity and cause substantial losses in yield within short periods of time (Strange and Scott, 2005). Elucidation of traits governing photosynthetic efficiency and plant acclimation therefore stands at the forefront of attempts to refine the sustainable utilization of plants in the future.

Besides its direct role in basic production, photosynthesis also fuels and regulates a wide range of defense mechanisms, which may equally well effect plant productivity in terms of biomass and crop yield. Photosynthetic activity as such largely influences the general energy state and redox status by providing NADPH, ATP and carbon skeletons, which support the growth of plants and fuel the initiation and maintenance of responses against external stress factors. In addition, changes in the functional status of photosynthetic components may trigger and fine-tune plant responses to both biotic and abiotic stress agents. To keep growth, metabolism and costly defensive reactions in balance, plants have evolved a plethora of mechanisms that optimize photosynthetic performance and prevent the formation of unnecessary stress signals upon environmental fluctuations. These mechanisms deploy complex self-regulatory feed-back systems that shuttle the information between different cellular compartments thereby ensuring appropriate responses to prevailing environmental cues (Sierla et al., 2013).

Over the past decades, extensive research on photosynthetic light reactions has produced high-resolution structural data for the

Abbreviations: ATP, Adenosine triphosphate; CET, cyclic electron transfer; Cytb6f, cytochrome B6f complex; ETI, effector-triggered immunity; FLG22, conserved peptide of bacterial flagellin; HR, hypersensitive response; LHCII, Light harvesting complex II; LET, linear electron transfer; NADPH, reduced form of Nicotinamide adenine dinucleotide phosphate; NDH, NAD(P)H dehydrogenase-like complex; NPQ, Non-photochemical quenching of excitation energy; $^1O_2$, singlet oxygen; PAMP, pathogen-associated molecular pattern; PQ, plastoquinone; PSI, Photosystem I; PSII, Photosystem II; PTI, PAMP-triggered immunity; ROS, reactive oxygen species.

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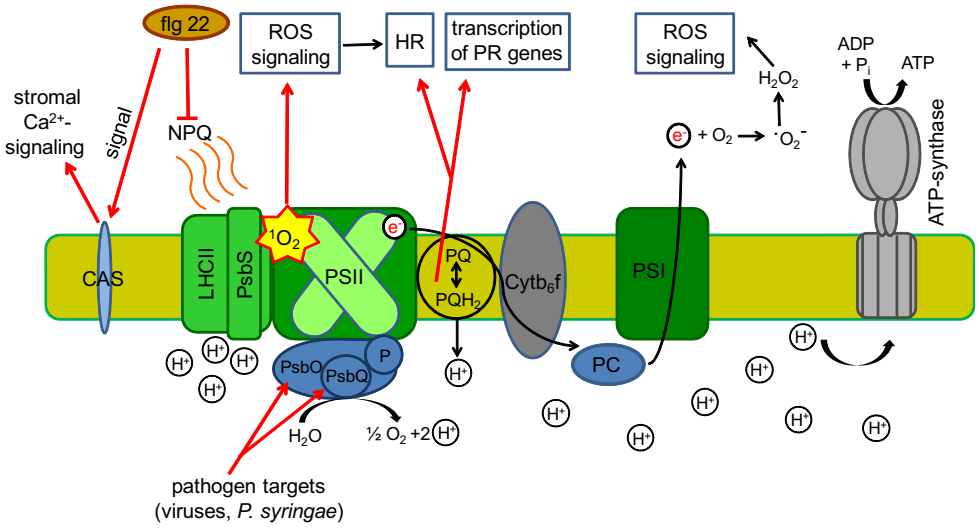

Fig. 1. Photosynthetic light reactions as a source of immunity signals in plants. Light-induced reduction of the plastoquinone (PQ) pool, formation of singlet oxygen ($^1O_2$) in Photosystem II (PSII) and/or generation of superoxide ($^{\bullet}O_{2-}$) and hydrogen peroxide (H_2O_2) through Photosystem I (PSI) may trigger the induction of pathogenesis related genes and hypersensitive reaction (HR) upon biotic challenges. To elicit such redox signals, plants may intentionally modulate the activation state of photoprotective mechanisms. Perception of a conserved peptide of bacterial flagellin (flg22) in the apoplast triggers calcium-dependent signals and down-regulation of non-photochemical energy quenching (NPQ) in chloroplasts. Weakening of NPQ may promote reduction of the PQ-pool and formation of $^1O_2$ in PSII. Acidification of thylakoid lumen promotes NPQ but also activates photosynthetic control, which limits electron transport through the Cytochrome b6f (Cytb6f) complex, promotes reduction of the PQ-pool, but alleviates the formation of ROS in PSI. Different plant pathogens attempt to annul ROS production and the consequent formation of defense signals in chloroplasts. Both bacterial and viral pathogens are known to deteriorate the oxygen evolving complex of PSII in this purpose.

major thylakoid membrane protein complexes, and facilitated functional analyses of electron transfer reactions as well as photophosphorylation by Photosystem II (PSII), cytochrome b6f complex (Cytb6f), Photosystem I (PSI) and the ATPase complex of photosynthetic organisms. As a result, the structure-function relationships of photosynthetic light reactions have become relatively well established (Amunts et al., 2010; Rochaix, 2011; Umena et al., 2011; Barber and Horton, 2012). Also the key mechanisms underlying dynamic regulation of the photosynthetic apparatus, and its interconnections with the metabolic and regulatory networks of the entire plant have started to emerge (Anderson et al., 2012; Grieco et al., 2012; Tikkanen and Aro, 2013). Comprehensive understanding of the regulatory networks, however, still awaits future discoveries. This review highlights the current understanding of components that delicately regulate photosynthetic light reactions on the thylakoid membrane and contribute to the modulation of biotic stress responses in higher plants.

1.1. Photosystem II carries a safety valve that protects the photosynthetic machinery from extensive photodamage

Susceptibility of photosynthetic structures to light-induced damage has been one of the major targets for interdisciplinary research on photosynthetic processes. Illumination under high irradiance levels favors the formation of highly reactive redox intermediates and photoinhibition of PSII, a damaging event commonly associated with light-induced formation of singlet oxygen ($^1O_2$) in the reaction center of PSII (Barber and Andersson, 1992; Aro et al., 1993; Macpherson et al., 1993; Hideg et al., 1998). The exact mechanism of photoinhibition, however, still remains controversial and likely involves a combination of damaging effects in PSII (Nath et al., 2013; Tyystjärvi, 2013). This intrinsic vulnerable property of PSII manifests itself in a targeted damage to the reaction center protein D1, which is generally considered to limit the damage to a single component that can be degraded and replaced by *de novo* protein synthesis in the so called repair cycle of PSII (Mulo et al., 2008). This biochemically complex sequence of events involves movement of the macromolecular PSII complexes between different compartments of the thylakoid membrane, also prone to structural alterations during light stress (Herbstova et al., 2012). Along the way, sequential functions of numerous protein kinases, protein phosphatases, proteases and auxiliary proteins have been found to assist the repair process (Mulo et al., 2008; Rochaix, 2011).

The entire PSII repair machinery is delicately regulated according to the redox status and metabolic state of the chloroplast, and provides plants with a mechanism for tuning the amount of active PSII centers to a level that corresponds to the metabolic capacity in the chloroplast. For example, counteracting activities of the STATE TRANSITION 8 (STN8)/PHOTOSYSTEM II CORE PHOSPHATASE (PBCP) kinase/phosphatase pair most likely regulate the repair process through reversible phosphorylation of the PSII core proteins D1, D2 and CP43 (Bonardi et al., 2005; Tikkanen et al., 2008; Samol et al., 2012; Tikkanen and Aro, 2012). Such tight regulation is crucial, since electron transfer through PSII may also compromise the function of PSI. Although PSI is relatively tolerant against absorption of excess light energy, it is remarkably prone to oxidative damage upon excess electron flow from PSII under high light conditions (Sonoike, 2011; Suorsa et al., 2012). Recently, down-regulation of the PSII turnover was shown to protect PSI from permanent photodamage (Tikkanen et al., 2014). It is thus conceivable that controlled containment of PSII repair has far-reaching effects on the stress resistance of plants.

1.2. Photoprotective mechanisms are coming to light

To cope with the potentially detrimental effects of light, higher plants have evolved numerous protective mechanisms that prevent over-accumulation of redox intermediates and formation of reactive oxygen species (ROS) in the photosynthetic electron transfer chain. Additionally, chloroplasts are well equipped with a multilayered antioxidant system that can efficiently prevent uncontrolled oxidative damage by ROS. These scavenging systems include carotenoids and tocopherol that are embedded in the light-harvesting antenna of PSII (LHCII), as well as the low molecular