REVIEW

Photoinhibition of photosynthesis: Role of carotenoids in photoprotection of chloroplast constituents

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Abstract

Exposure of plants to irradiation, in excess to saturate photosynthesis, leads to reduction in photosynthetic capacity without any change in bulk pigment content. This effect is known as photoinhibition. Photoinhibition is followed by destruction of carotenoids (Cars), bleaching of chlorophylls (Chls), and increased lipid peroxidation due to formation of reactive oxygen species if the excess irradiance exposure continues. Photoinhibition of photosystem 2 (PS2) in vivo is often a photoprotective strategy rather than a damaging process. For sustainable maintenance of chloroplast function under high irradiance, the plants develop various photoprotective strategies. Cars perform essential photoprotective roles in chloroplasts by quenching the triplet Chl and scavenging singlet oxygen and other reactive oxygen species. Recently photoprotective role of xanthophylls (zeaxanthin) for dissipation of excess excitation energy under irradiance stress has been emphasised. The inter-conversion of violaxanthin (Vx) into zeaxanthin (Zx) in the light-harvesting complexes (LHC) serves to regulate photon harvesting and subsequent energy dissipation. De-epoxidation of Vx to Zx leads to changes in structure and properties of these xanthophylls which brings about significant structural changes in the LHC complex. This ultimately results in (1) direct quenching of Chl fluorescence by singlet-singlet energy transfer from Chl to Zx, (2) trans-thylakoid membrane mediated, ΔpH-dependent indirect quenching of Chl fluorescence. Apart from these, other processes such as early light-inducible proteins, D1 turnover, and several enzymatic defence mechanisms, operate in the chloroplasts, either for tolerance or to neutralise the harmful effect of high irradiance.

Additional key words: chlorophyll fluorescence; high irradiance; photosystem 1; photosystem 2; violaxanthin; xanthophyll cycle; zeaxanthin.

Introduction

The absorption of photons by chlorophyll (Chl) molecules is a physical process with no biological control over it. However, the biochemistry of photosynthesis and subsequently the utilisation of reducing power and chemical energy in CO₂ fixation are always under the control of various enzymes and metabolic intermediates in a highly co-ordinated manner. At irradiance limiting photosynthesis, photons are captured and utilised with the highest possible efficiency. Photosynthetic apparatus is capable of absorption of radiant energy over a wide range of photon flux density (PFD). However, with increasing PFD, the rate of photosynthesis initially increases linearly and above a certain PFD, the process is incapable of utilising all the absorbed energy (Demmig-Adams 1990) and hence declines.

The primary charge separation at PS2 reaction centre (RC) occurs much faster than the electron transport. When the rate of transfer of excitation energy from the antennae to RCs exceeds the rate of transfer from the RCs to the electron transport chain, photoinhibition is resulted. Typical manifestation of photoinhibition in leaves includes sustained decrease in photon yield and often a reduction in maximum photosynthetic capacity. Photoinhibition is often associated with damage to the photosynthetic apparatus under prolonged high irradiance (HI). One of the components most frequently suggested to be

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Abbreviations: Ax – antheraxanthin; Car – carotenoids; Chl – chlorophyll; HI – high irradiance; LHC – light-harvesting complex; OEC – oxygen evolving complex; PFD – photon flux density; PS – photosystem; RC – reaction centre; VDE – violaxanthin de-epoxidase; Vx – violaxanthin; ZE – zeaxanthin epoxidase; Zx – zeaxanthin.

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Photoinhibition may also result not only from some form of damage to PS2 but also from an increased thermal energy dissipation, which is a photoprotective process and does not reflect damage (Demming-Adams 1990, Demming-Adams and Adams 1992). It is now evident that photoinhibition, which results from the conjunction of high irradiance stress with other stress factors such as drought, chilling, or high temperature, has an important impact on plants under natural conditions. Plants have evolved photoadaptive and photoprotective mechanisms at levels ranging from the whole plant to leaves and thylakoid membrane of chloroplasts, to avoid excessive radiant energy interception, and have developed several mechanisms to minimise the energisation of the thylakoid under high irradiance.

Cars are a diverse group of lipophilic pigment molecules that are widely distributed in nature and present in all photosynthetic organisms. They act as light-harvesting antennae and also protect the photosynthetic apparatus from photodestruction in strong irradiance (Siefermann-Harms 1985, Bartley and Scolnik 1995). Cars protect photosynthetic apparatus through two important ways: (1) β-carotene (β-Car) directly quenches both triplet chlorophyll (Chl') and singlet oxygen (O2). (2) The xanthophylls lower the Chl' formation by quenching excited singlet state of Chl (Chl').

The review focuses current views on photoinhibition and the primary photoprotective mechanism responsible for dissipation of excess absorbed photon energy in the light-harvesting antennae of PS2.

**Photoinhibition of photosynthesis**

Photoinhibition of photosynthesis generally denotes a decrease in photosynthetic activity when plants are exposed to HI that exceeds the ability of the light-harvesting system to dissipate the energy not used for photochemistry. Powles (1984) considered the photoinhibition as a first stage of HI-induced thylakoid damage leading to reduction of photosynthetic capacity. The second stage, pigment photooxidation, commences after a long-term exposure of the plants to strong irradiance and concerns the bleaching of antennae pigments. The later process requires oxygen.

**Photoinhibition at PS2**

The primary target of HI causing photoinhibition of photosynthesis is PS2. In this process, two mechanisms are involved which may affect either the acceptor side or the donor side of PS2. The two mechanisms are distinguished on the basis of differences in the primary site of electron transport malfunctioning, the subsequent D1 protein degradation, and the oxygen requirement of the process (Fig. 1).

![Fig. 1. Scheme showing the two routes of damage due to acceptor and donor side photoinhibition (Minkov et al. 1999). Bold arrows indicate site of photoinhibition.](image)

Acceptor side-induced photoinhibition of PS2 occurs under HI when it exceeds the saturation of photosynthetic electron transport (Barber and Andersson 1992). Excess exposure causes non-physiological over-reduction of the first quinone electron acceptor in PS2. This brings about sequential modifications at the level of QA and QB acceptors (Keren et al. 1997). These conditions lead to the recombination of the radical pair, P680*Pheo* (Vass et al. 1992) and the production of the triplet state of P680 (P680*). Under aerobic conditions, this Chl may be quenched by oxygen and O2 is thus produced (Fig. 1). The addition of O2 scavengers such as histidine (Mishra et al. 1994, Telfer et al. 1994), diazobicyclooctane (Barényi and Krause 1985, Miyao 1994), azid (Macpherson et al. 1993) as well as free radical scavengers such as uric acid or propylgallate (Sopory et al. 1990) provide partial photoprotection against the acceptor side-induced photo-inhibition of PS2 in an isolated system. The O2* initiates and also triggers degradation of the RC protein D1, probably by promoting a special conformational change which makes the protein susceptible to proteolytic cleavage (Fig. 1). One possibility is that, in complex in vivo systems, the D1 protein may be cleaved by the direct action of active oxygen. The possible cleavage site is on the stroma side of the thylakoid membrane and the characteristic degradation products of the D1 protein are 23 kDa.