Journal of Plant Biology, March 2008, 51(2) : 85-96

Reaction Centre Quenching of Excess Light Energy and Photoprotection of Photosystem II

Alexander G. Ivanov[1,2]*, Vaughan Hurry[2], Prafullachandra V. Sane[1,2], Gunnar Öquist[2], and Norman P. A. Huner[1]

[1]Department of Biology and the Biotron, University of Western Ontario, 1151 Richmond Street N, London, Ontario, Canada N6A 5B7

[2]Umeå Plant Science Centre, Department of Plant Physiology, Umeå University, Umeå S-901 87, Sweden

In addition to the energy dissipation of excess light occurring in PSII antenna *via* the xanthophyll cycle, there is mounting evidence of a zeaxanthin-independent pathway for non-photochemical quenching based within the PSII reaction centre (reaction centre quenching) that may also play a significant role in photoprotection. It has been demonstrated that acclimation of higher plants, green algae and cyanobacteria to low temperature or high light conditions which potentially induce an imbalance between energy supply and energy utilization is accompanied by the development of higher reduction state of Q_A and higher resistance to photoinhibition (Huner et al., 1998). Although this is a fundamental feature of all photoautotrophs, and the acquisition of increased tolerance to photoinhibition has been ascribed to growth and development under high PSII excitation pressure, the precise mechanism controlling the redox state of Q_A and its physiological significance in developing higher resistance to photoinhibition has not been fully elucidated. In this review we summarize recent data indicating that the increased resistance to high light in a broad spectrum of photosynthetic organisms acclimated to high excitation pressure conditions is associated with an increase probability for alternative non-radiative $P680^+Q_A^-$ radical pair recombination pathway for energy dissipation within the reaction centre of PSII. The various molecular mechanisms that could account for non-photochemical quenching through PSII reaction centre are also discussed.

Keywords: electron transport, energy dissipation, non-photochemical quenching, photoprotection, photosystem II, reaction centre quenching

Changes in irradiance, temperature, nutrients and water availability result in imbalances between the light energy absorbed through photochemistry and energy utilization through photosynthetic electron transport coupled to carbon, nitrogen and sulphur reduction. This leads to photoinhibition of photosynthesis that may result in photodamage to the D1 reaction centre polypeptide of PSII (Aro et al., 1993; Krause, 1988; Long et al., 1994; Powles, 1984). Recovery from photoinhibition in plants, green algae and cyanobacteria is thought to involve a PSII repair cycle in which photodamaged D1 is degraded and the re-synthesised D1 is reinserted to form a functional PSII reaction centre (Aro et al., 1993; Melis, 1999). It has been shown in some chilling-sensitive plant species, green algae and cyanobacteria that protection against photoinhibition may be accounted for, in part, by an increased rate of repair relative to the rate of photodamage to D1 (Melis, 1999; Nishida and Murata, 1996). Alternatively, certain cold tolerant plant species such as winter wheat (*Triticum aestivum* L.), rye (*Secale cereale* L.), barley (*Hordeum vulgare* L.), *Arabidopsis thaliana*, exhibit a minimal dependence on D1 repair in response to cold but instead increase photosynthetic capacity and reprogramme carbon metabolism to increase RuBP regeneration and to recover flux through the Calvin cycle, increasing the capacity for photochemical quenching (Adams et al., 2001; Huner et al., 1993; Hurry et al., 1995; Kim et al., 2005; Stitt and Hurry, 2002; Strand et al., 2003; Strand et al., 1997; Xu et al., 1999). This reprogramming of metabolism results in an increased capacity to keep Q_A oxidized and PSII reaction centres open under high excitation pressure induced by either excessive irradiance or low temperatures (Huner et al., 1998; Öquist and Huner, 2003). Thus, photoprotection in these species is accomplished, in part, through an increase in photochemical quenching (qP) (Krause and Jahns, 2003).

In contrast to the D1 repair cycle and photochemical quenching, the concept of radiationless dissipation of excess energy through antenna quenching was originally developed on the basis of the Butler model for energy transfer and used to account for Chl fluorescence quenching (Butler, 1978). Non-photochemical quenching (NPQ) of excess excitation energy in the antenna pigment bed of PSII is considered to be the major PSII photoprotective mechanism (Demmig-Adams and Adams, 1992; Gilmore, 1997; Gilmore and Ball, 2000; Horton et al., 1996; Ort, 2001).

*Corresponding author; fax +1-519-850-2343
e-mail aivanov@uwo.ca

Abbreviations: Cyt b_{559}, cytochrome b_{559}; D1, photosystem II reaction centre polypeptide; D2, photosystem II reaction centre polypeptide; F_o, minimum yield of chlorophyll fluorescence at open PSII centres in dark-adapted leaves; F_m, maximum yield of fluorescence at closed PSII reaction centres in dark adapted leaves; F_v variable yield of fluorescence in dark adapted leaves; F_v/F_m, maximum PSII photochemical efficiency in dark adapted leaves; LHCII, the major Chl a/b pigment-protein complex associated with PSII; NPQ, non-photochemical quenching; OEC, oxygen evolving complex; Pheo, pheophytin; PSI, photosystem I; PSII, photosystem II; PSIIβ, photosystem β centres; PSIIα, photosystem α centres; PsbS, PSII subunit and gene product of the *PsbS* gene; PQ, plastoquinone; Q_A, primary electron-accepting quinone in PSII reaction centres; Q_B, secondary electron-accepting quinone in PSII reaction centres; qE, ΔpH dependent high energy quenching; quenching coefficient for basal fluorescence; qP, photochemical quenching coefficient; TL, thermoluminescence; T_M, temperature of maximum thermoluminescence emission; V, violaxanthin; Z, zeaxanthin.

There is a historic precedence for the role of reaction centre quenching in the non-photochemical dissipation of excess energy (Weis and Berry, 1987; Krause and Weis, 1991; Walters and Horton, 1993). We summarize the major mechanisms capable of dissipating excess excitation energy within the thylakoid membranes and focus on the reaction centre quenching of excess light and its role in acclimation to changing environmental conditions and photoprotection of the photosynthetic apparatus. We also summarize recent experimental evidence that supports a significant role for reaction centre quenching in the photoprotection of PSII in cyanobacteria, green algae, conifers, and herbaceous plants. Various molecular mechanisms that could account for the increased probability for energy dissipation within the reaction centre of PSII are also discussed. We conclude that, as originally suggested by Krause and Weis (1991), it is probable that both reaction centre and antenna quenching function *in vivo* to different extents, depending on the environmental conditions, to protect PSII from photodamage.

PHOTOPROTECTIVE MECHANISMS OF PSII

Zeaxanthin-dependent Nonphotochemical Quenching of Excess Light Energy

There is broad agreement that the radiationless dissipation of excess excitation energy in the chlorophyll pigment bed of LHCII, associated with the formation of the xanthophyll pigment zeaxanthin (Z), is one of the major protective mechanisms against photoinhibitory damage of PSII (for a reviews see: Demmig et al., 1987; Demmig-Adams, 1990; Demmig-Adams and Adams, 1992; Horton et al., 1996; Niyogi, 1999; Ort, 2001). The role of the pH- and zeaxanthin-dependent shifts in the oligomerisation state of LHCII (Horton et al., 1996) as well as the activation state of zeaxanthin (Ruban et al., 2002; Aspinall-O'Dea et al., 2002) in developing the rapidly relaxing energy dependent component (qE) of NPQ has been well characterized, and this mechanism is considered to reflect the indirect, allosteric mechanism for antenna quenching.

More recently, a possible involvement in NPQ has been suggested for the *psbS* gene product (Funk et al., 1995a), which belongs to the LHC protein superfamily (Wedel et al., 1992). Indeed, a *psbS* deletion mutant (Li et al., 2000) and various *psbS*-defective mutants (Peterson and Havir, 2001; Peterson and Havir, 2003; Grasses et al., 2002) are impaired in the development of the major component of NPQ. Based on the original observation that PsbS binds chlorophylls and xanthophylls (Funk et al., 1995b), and its role in the development of qE, Li et al. (2000) suggested that this protein is the site of ΔpH and xanthophyll-dependent NPQ. More detailed biochemical analysis suggested that the PsbS protein does not bind pigments (Dominici et al., 2002) and most of the highly conserved amino acids that form the ligands for chlorophyll in most of the LHC proteins (Kühlbrandt et al., 1994; Bassi et al., 1999) are not found in PsbS. However, isolated PsbS protein was shown to bind exogenous zeaxnathin (Aspinall-O'Dea et al., 2002), making the precise function and specific mode of action of the PsbS protein still controversial.

Inactive PSII Reaction Centres as Quenchers of Excess Energy

In addition to ΔpH and zeaxanthin-dependent nonphotochemical quenching, it has been demonstrated that fluorescence quenching might result from a conversion of PSIIα-centres (dimers) to PSIIβ–centres (monomers) in a low fluorescence state (Delrieu, 1998). The monomerization of PSII centres would effectively decrease the absorption cross-section of PSII (Delrieu, 1998) and can be triggered by high light (Kruse et al., 1997). The relative proportion of active PSII centres versus inactive centres is dependent both on the intrathylakoid ΔpH as well as the proportion of closed reaction centres measured as the relative reduction state of Q_A (Weis and Berry, 1987; Krause and Weis, 1991; Krause and Jahns, 2003). Furthermore, photoinactivated PSII complexes may also effectively dissipate excess excitation energy as heat (Krause, 1988) and the role of photoinactivated centres as quenchers increases with the severity of photoinactivation (Lee et al., 2001). It has been proposed that the conversion of photochemically active, fluorescent, closed PSII reaction centres into photochemically inactive, nonfluorescent PSII reaction centres may serve as an effective mechanism for energy dissipation (Krause, 1988; Krause and Weis, 1991) and prevent further damage not only to the photoinactivated reaction centres themselves but also to neighbouring active PSII reaction centres (Lee et al., 2001; Matsubara and Chow, 2004).

PSII Cyclic Electron Transport

Reduction of $P680^+$ by $Cytb_{559}$ *via* the cyclic electron pathway: $Cytb_{559} \rightarrow dChl_z \rightarrow$ β-Car $\rightarrow P^+_{680}$, represented by dotted lines in Figure 2, has been also suggested as an alternative energy dissipating mechanism operating within the PSII reaction centre (Barber and De Las Rivas, 1993; Falkowski et al., 1986; Telfer et al., 1991; Allakhverdiev et al., 1997). The conversion of $Cytb_{559}$ from its high potential form (HP) to the low-potential form (LP) (Prasil et al., 1996), may play a key role as a molecular switch allowing it to act as an electron donor to $P680^+$ (HP form) or an electron acceptor (LP form) (Barber and De Las Rivas, 1993). The role of the high potential form of $Cytb_{559}$ in photoprotection of PSII has been also discussed (Stewart et al., 1998; Thompson and Brudvig, 1988). It has been suggested that $Cytb_{559}$ may act as a secondary donor to $P680^+$ *via* the $dChl_z$ chlorophyll molecule (Barber and De Las Rivas, 1993; Nield et al., 2000; Thompson and Brudvig, 1988). In support of this, the reversible oxidation of $dChl_Z$ was shown to play a significant role in fluorescence quenching in PSII (Schweitzer and Brudvig, 1997). Additionally, β-carotene can be photooxidized and it was suggested to facilitate the electron flow from $Cytb_{559}$ and $dChl_Z$ to $P680^+$ (Telfer et al., 1991, 1994). Although the involvement of β-carotene and $dChl_Z$ in cyclic electron flow from $Cytb_{559}$ to $P680^+$ has been ambiguous, the recently published structure of the oxygen evolving complex placed one of the β-carotene molecules of the PSII reaction centre in direct contact with Chl_{ZD2} and

between Cytb_{559} and P680 chlorophylls (Ferreira et al., 2004), supporting a role for β-carotene in PSII cyclic electron flow.

More recently, cyclic electron transport around PSII in intact chloroplasts was demonstrated to be almost equal to the water-water cycle driven electron flow, implying that it can effectively dissipate excess light energy and contribute to photoprotection of PSII under conditions that limit photosynthesis (Miyake and Yokota 2001; Miyake et al. 2002). The accumulation of Chl_z^+, as a result of over-oxidation of P680, has also been suggested as a site for photoprotection (Schweitzer and Brudvig, 1997; Stewart et al., 1998).

State I – State II Transitions

State transitions have been also well known as an adaptive mechanism for short-term redistribution of excitation energy between the two photosystems, which may play substantial role in protecting PSII from overexcitation in all photosynthetic organisms (Anderson, 1986; Allen, 1995; Lunde et al., 2000; Haldrup et al., 2001). It is well established that state transitions involve reversible phosphorylation/dephosphorylation of the major LHCII by a thylakoid-bound kinase, which is activated by the redox state of the PQ pool and regulated by the imbalance of PSII excitation and the capacity of the acceptor side of PSI to utilize the electron flow. The phosphorylation of LHCII induces lateral migration of P-LHCII from PSII towards PSI increasing the antenna cross-section of PSI at the expense of PSII (Anderson, 1986; Allen, 1995; Haldrup et al., 2001) resulting in a redistribution of the energy in favour of PSI. Cyt b_6/f has been identified as the redox sensor of the PQ pool and has been involved in controlling the phosphorylation of LHCII (Anderson, 1992). Recently, the small PsaH subunit of the PSI complex has been demonstrated to play a crucial role for state transition in *Arabidopsis thaliana* (Lunde et al., 2000).

Photorespiration, the Water-water Cycle and Chlororespiration

Photorespiration can also effectively dissipate excess light energy and can serve as an alternative sink for photosynthetic electrons by consumption of NADPH and ATP *via* the photorespiratory pathway (Osmond, 1981; Kozaki and Takeba, 1996). It has been demonstrated that the photorespiratory pathway may play significant role in protecting plants from photoinhibition (Osmond, 1981; Kozaki and Takeba, 1996). In addition, the role of the water-water cycle as an alternative photon and electron sink, in suppressing photoinhibition by decreasing the production of reactive oxygen species is also well established (Asada, 1999).

Chlororespiration, a light independent process that can maintain a trans-thylakoid proton gradient, has been also suggested as an effective alternative electron sink in alleviating over-reduction of the PQ pool and protecting PSII reaction center from photo-damage under excessive light conditions (Field et al., 1998). It has been demonstarted that the contribution of the chlororespiratory electron flux involving the NDH-complex and PTOX to total electron flow in the chloroplast and its photoprotective role as an alternative electron sink is rather limited under optimal growth conditions (Ort and Baker, 2002; Rosso et al., 2006). However, chlororespiration has been shown to play an important photoprotective role in the high alpine plant species *Ranunculus glacialis* acclimated to low temperature (Streb et al., 2005). Furthermore, up-regulation of PTOX and the chloroplast NDH-complex have been reported in oat plants subjected to heat and high light stresses (Quiles, 2006).

REACTION CENTRE QUENCHING

Although a major focus of recent research on photoprotection has been on the contribution of antenna quenching to NPQ (for a reviews see: (Horton et al., 1996; Niyogi, 1999), there is historical precedence for alternative mechanisms for the dissipation of excess light and of photoprotection of PSII reaction centres (Krause, 1988; Krause and Weis, 1991; Walters and Horton, 1993). Considerable experimental evidence for non-radiative energy dissipation within the reaction centre of PSII has been published (Briantais et al., 1979; Weis and Berry, 1987; Krause, 1988; Bukhov et al., 2001; Sane et al., 2002; Vavilin and Vermaas, 2000; Ivanov et al., 2003, 2005, 2006; Finazzi et al., 2004; Matsubara and Chow, 2004; Zulfugarov et al., 2007).

At physiological temperatures, *in vivo* chlorophyll fluores-

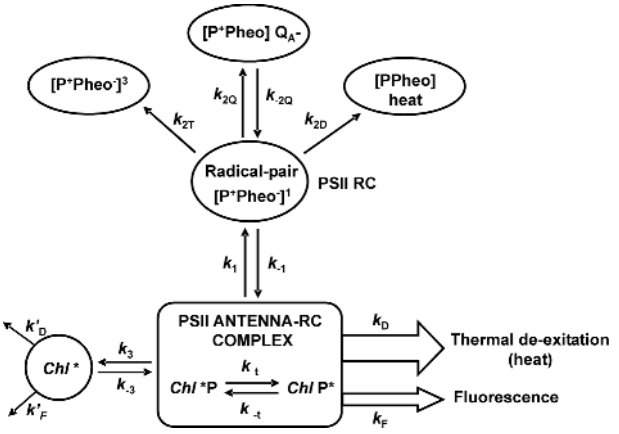

Figure 1. A schematic kinetic model of energy migration in PS II modified from the original reversible radical pair model (Schatz et al., 1988) including additional kinetic compartment of loosely connected PSII antenna (Moise and Moya, 2004). The rapid excitation equilibrium between the antenna of PSII and the primary donor P (P680) represented by the trapping and detrapping rate constants k_t and k_{-t} are not explicitly considered since the model assumes trap-limited exciton decay. The apparent rate constant for the primary charge separation which results in the primary singlet radical pair $[P^+Pheo^-]^1$ is represented by k_1. The primary singlet radical pair may recombine back to the excited state of the primary donor (k_{-1}) or by three separate reactions to recombine (nonradiative decay) back to the ground state (k_{2D}), to the triplet (spin dephasing) excited state (k_{2T}), and under Fo conditions by photochemical conversion (charge stabilization) to transfer an electron to Q_A (k_{2Q}) (Schreiber and Krieger, 1996). k_3 and k_{-3} describe the excitation energy exchange between the main PSII antenna-RC complex and the loosely connected PSII antenna. k_D, k_F and k'_D, k'_F represent the apparent rate constants for nonradiative (thermal deexitation) and radiative (fluorescence) deactivation processes in the main PSII antenna-RC complex and the loosely connected PSII antenna, respectively.

cence originates from the light harvesting antenna of PSII and its magnitude reflects the competing processes of photochemistry and non-photochemical dissipation of the absorbed light energy (Butler, 1978; Krause and Weiss, 1991). As postulated in the bipartite model (Butler, 1978) there are three pathways for available dissipation of an exciton within the PSII antenna (Figure 1): 1) thermal de-excitation (k_D); 2) it can be re-emitted as fluorescence (k_F) and 3) it can be transferred to the reaction centre of PSII (k_t). It is considered that if an exciton is transferred to an open reaction centre (primary quinone acceptor Q_A is oxidized) it can be used for photochemistry by transferring an electron from water to Q_A (k_1). Based on fluorescence lifetime measurements a reversible radical pair model, featuring rapid excitation equilibrium between the PSII antenna and the reaction centre P680 (P) was proposed by Schatz et al. (1988). In this model, excitation trapping is described by the apparent rate constant k_1 for the primary charge separation, resulting in the primary radical pair $[P^+Pheo^-]^1$ in a singlet state. This process is considered to be reversible and the rate constant k_{-1} describes the recombination of $[P^+Pheo^-]$ back to the excited state of the donor P* (Fig. 1). When the reaction centre is open under F_o conditions, three further deactivation pathways could be considered: 1) photochemical energy conversion by charge stabilization reactions (k_{2Q}); 2) spin dephasing processes resulting in a triplet radical pair state (k_{2T}); and 3) recombination of the singlet radical pair by nonradiative decay to ground state (k_{2D}) (Schatz et al., 1988; Wagner et al., 1996; Schreiber and Krieger, 1996). Although the original model proposed by Schatz et al. (1988) does not assume the formation of a triplet state of the primary radical pair, later nanosecond flash absorption spectroscopy analysis of charge recombination reactions in PSII has demonstrated that the yield of the triplet state ^{3}P680 was high in preparations with both open or closed reaction centres (Van Mieghem et al., 1995; Hillmann et al., 1995). However, if the reaction centre is closed (when Q_A is reduced under F_m conditions) the reversible radical pair model predicts that only radiative and non-radiative recombinations to the ground state are possible. Assuming all of the above, it is clear that the fluorescence yield (k_F) reflects the excitation density in PSII antenna-reaction centre complex, determined by light absorption, primary radical pair recombination (k_{-1}) and competing nonradiative decay (k_D). The increase in fluorescence yield when the primary stable electron acceptor is reduced is defined as variable fluores-

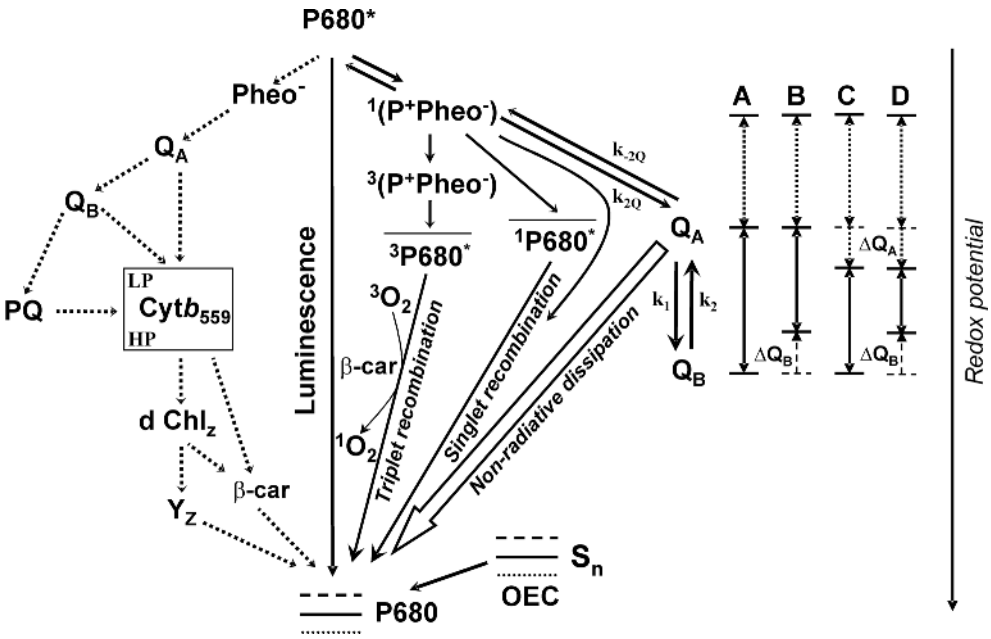

Figure 2. Simplified model of possible pathways associated with non-photochemical chlorophyll fluorescence quenching localised within the reaction centre of PSII. The primary electron donor P680 accepts light excitation energy and the charge separation *via* the first singlet exited state results in the radical pair $P680^+Pheo^-$ followed by the electron transfer from $Pheo^-$ to Q_A. If the water splitting complex is inhibited the probability for charge recombination will arise due to reversible back electron transfer from Q_A^- to Pheo resulting in a singlet $^1(P680^+Pheo^-)$, or by spin dephasing to triplet $^3(P680^+Pheo^-)$ radical pair. Both radical pairs are unstable and will recombine *via* ***singlet recombination*** $[^1(P680^+Pheo^-)]$ to the ground state P680 or the first singlet excited state ^{1}P680* and *via* ***triplet recombination*** $[^3(P680^+Pheo^-)]$ resulting in the triplet excited state ^{3}P680*, which may be quenched by β-carotene *via* singlet oxygen. When forward electron transfer from Q_A^- to Q_B is favoured ($k_1 >> k_2$) radiative energy dissipation pathway probably involves the back-reaction *via* the $P680^+Pheo^-$ radical pair (**A**). Shifting the redox potential of Q_B toward Q_A favours the k_2 rate constant and would result in increased steady state proportion of reduced Q_A (**B**). Shifting the redox potential of Q_A to more positive values will decrease the k_{-2Q} rate constant and increase the free energy gap between $P680^+$ and Q_A^-. This will decrease the probability for a charge recombination pathway involving $P680^+Pheo^-$ and will also cause stabilization of $S_2Q_A^-$ pair (**C**). It is proposed that this will increase the probability for direct recombination of Q_A^- with $P680^+$ *via* ***non-radiative dissipation***, thus avoiding triplet recombination and production of singlet oxygen. Shifting the redox potentials of Q_A and Q_B in opposite directions (**D**) would cause further stabilization of $S_2Q_A^-$ and further increase the probability for non-radiative energy dissipation. The cyclic electron pathway: Cytb_{559} → dChl$_z$ → β-Car → P^+_{680} is represented by dotted lines. The key role of Cytb_{559} as one electron protectant against donor or acceptor side photoinhibition is emphasized by the presence of its high potential (HP) and low potential (LP) forms, which may allow it to act as an electron acceptor (for acceptor side protection) or as an electron donor (for donor side protection) (Barber and De Las Rivas, 1993).

cence (F_V) and it is assumed to reflect a decrease in k_1 and an increase in k_{-1}. Schreiber and Krieger (1996) proposed that an increase in the rate of nonradiative energy dissipation processes (k_{2D} and k_{2T}) in the presence of reduced Q_A (Q_A-) would also result in a decrease in [P^+Pheo^-] recombination (k_{-1}), thus modulating the magnitude of F_v. In support of this hypothesis, a picosecond time-resolved study showed that zeaxanthin-independent ΔpH non-photochemical quenching (Wagner et al., 1996) and the slow reversible qI type nonphotochemical quenching in photoinhibited leaves (Richter et al., 1999) does involve nonradiative decay of the primary charge separated state to its ground state and/or triplet excited state (k_{2D} and k_{2T}) localized within the reaction centre of PSII.

A simplified model of possible pathways associated with non-photochemical chlorophyll fluorescence quenching localised within the reaction centre of PSII is presented in Figure 2. The primary electron donor P680 accepts excitation energy and charge separation *via* the first singlet exited state results in formation of the radical pair $P680^+Pheo^-$, followed by electron transfer from $Pheo^-$ to Q_A. If the water splitting complex is inhibited by Ca^{2+} depletion under increased ΔpH, changing the redox properties of both the donor and acceptor sides of PSII (Johnson et al., 1995; Krieger et al., 1993, 1995; Krieger and Weis, 1993), conditions will be right for alternative pathways for $P680^+$ reduction and the probability for charge recombination will increase. The release of Ca^{2+} cations from the donor side has been shown to shift the redox state of Q_A towards higher values (Johnson et al., 1995; Krieger et al., 1993, 1995; Krieger and Weis, 1993), inhibiting electron transfer from Q_A to Q_B and promoting recombination of the radical pair $P680^+Pheo^-$. Bruce et al. have also proposed that, in addition to this reaction centre quenching based on charge recombination between $P680^+$ and Q_A-, nonphotochemical energy dissipation may occur *via* direct quenching by $P680^+$ (Bruce et al., 1997). $P680^+$ can be also reduced by reverse electron transfer from Q_A- to Pheo *via* singlet $^1(P680^+Pheo^-)$, or by spin dephasing of pair electrons, by triplet $^3(P680^+Pheo^-)$ radical pair resulting in the formation of singlet $^1P680^*$ or a triplet $^3P680^*$, respectively (Schreiber and Neubauer, 1990). Both radical pairs are unstable, and the singlet radical pair $^1(P680^+Pheo^-)$ will recombine *via* singlet recombination to ground state P680 or the first singlet excited state $^1P680^*$ (Liu et al., 1993; Takahashi et al., 1987). The triplet recombination of $^3(P680^+Pheo^-)$ results in the triplet excited state $^3P680^*$ (Takahashi et al., 1987), which is quenched by β-carotene directly or indirectly *via* singlet oxygen (Takahashi et al., 1987; Telfer et al., 1994).

Under conditions, when forward electron transfer from Q_A- to Q_B is restricted ($k_1 << k_2$), the radiative energy dissipation pathway probably involves the back-reaction *via* the $P680^+Pheo^-$ radical pair (Fig. 2). The possible back reaction of reduced Q_A with $P680^+$ has been suggested previously (Krieger-Liszkay and Rutherford, 1998; Prasil et al., 1996) and this may be enhanced when Q_A remains reduced (Vavilin and Vermaas, 2000). Shifting the redox potential of Q_B toward Q_A favours the k_2 rate constant and would result in an increase in the steady state proportion of reduced Q_A. In addition, an increased free energy gap between $P680^+$ and Q_A^- would decrease the probability for a charge recombination pathway involving $P680^+Pheo^-$ and will also cause stabilization of $S_2Q_A^-$ pair. The accumulation of Q_A^- has been shown to inhibit the formation of radical pair $P680^+Pheo^-$, preventing P680 triplet formation (Schatz et al., 1988; Vass et al., 1992). Furthermore, it has been suggested that there is a non-radiative pathway of charge recombination between Q_A^- and the donor side of PSII (Briantais et al., 1979; Vavilin and Vermaas, 2000; Weis and Berry, 1987). Such a pathway would increase the probability for non-radiative dissipation of excitation energy within the reaction centre of PSII (Bukhov et al., 2001; Weis and Berry, 1987), avoiding triplet recombination and production of singlet oxygen.

PHOTOPROTECTION THROUGH REACTION CENTRE QUENCHING

Over-reduction of Q_A has been suggested to be a major prerequisite for efficient dissipation of the excess light within the reaction centre of PSII (Bukhov et al., 2001; Krause, 1988; Öquist and Huner, 2003). Based on a theoretical assessment of alternative mechanisms for NPQ in *Hordeum vulgare*, Walters and Horton (1993) concluded that reaction centre quenching is operative only when reaction centres are closed, that is, when Q_A is in the reduced state. Nonradiative charge recombination between Q_A^- and the donor side of PSII has been suggested as a mechanism for dissipating excitation energy *via* PSII reaction centre quenching (Briantais et al., 1979; Krieger et al., 1992; Vavilin and Vermaas, 2000; Weis and Berry, 1987). There is now a mounting body of experimental evidence for zeaxanthin-independent dissipation of excess light energy within the reaction centre of PSII, involving several possible pathways for nonradiative Q_A^- decay. In an early study, Jursinic and Govindjee (1982) estimated that more than 90% of recombinations between Q_A^- and the oxidized primary donor P^+ occurred *via* a nonradiative pathway in thylakoids with an impaired oxygen-evolving system. Similarly, charge recombination between Q_A^- and the oxidized primary donor P^+ was reported to occur predominantly *via* a nonradiative pathway in bacterial reaction centres (Woodbury et al., 1986). Donor side induced shifts in the redox properties of Q_A have also been reported during photoactivation of the Mn cluster in PSII (Johnson et al., 1995) and Ca depletion of the oxygen evolving complex (Krieger and Rutherford, 1997; Krieger et al., 1995). From these studies, it was proposed that the increase in E_m of Q_A/Q_A^- could result in photoprotection of the reaction centre against excess light by inducing a nonradiative charge recombination pathway that did not involve the formation of the $P680^+Pheo^-$ radical pair, and decreased the yield of P680 triplet and singlet oxygen (Krieger and Rutherford, 1997; Krieger-Liszkay and Rutherford, 1998).

Although, the concept for nonradiative dissipation of excitation energy within the reaction centre of PSII (reaction centre quenching) was developed mostly from *in vitro* experiments, *in vivo* Stern-Volmer analyses of NPQ in a marine diatom *Phaeodactylum tricornitum* also suggested that changes in NPQ resulted from thermal dissipation in both the PSII antenna and the reaction centre of PSII

(Olaizola et al., 1994). It was demonstrated that the contribution of reaction centre quenching to NPQ may affect the relationship between antenna quenching and changes in photochemical efficiency, resulting in a decrease in fluorescence yield that may not necessarily be accompanied by a decrease in photochemical efficiency (Olaizola and Yamamoto, 1994). Experimental analysis of fluorescence quenching based on a model involving three separate quenching mechanisms, two based in the reaction centre and one in the antenna of PSII, clearly demonstrated that reaction centre quenching is important, if not predominant, in spinach and *Arabidopsis* (Bukhov et al., 2001). Additionally, as distinct from higher plants, ΔpH-dependent qE was shown to be independent of the xanthophyll cycle in *Euglena gracilis* (Doege et al., 2000). Moreover, no evidence was found that the qE was localised in the PSII antenna in *Euglena gracilis* and reaction centre quenching of excess energy was suggested to be the major mechanism involved (Tschiersch et al., 2002). Similar ΔpH-dependent energy quenching occurring in the reaction centre of PSII, but not in the antenna, was proposed to be predominant in the red alga *Rhodella violacea* (Delphin et al., 1996). The possible involvement of enhanced charge recombination of Q_A- and $P680^+$, favouring nonradiative reaction centre dissipation of excess light, was suggested recently from an *in vivo* global analysis of PSII lifetime distributions in photoinhibited *Capsicum annuum* L. leaves (Matsubara and Chow, 2004). In addition, a zeaxanthin-independent non-photochemical quenching identified as a form of high-energy state quenching mechanism localized in the PSII core complex was shown in low light illuminated barley plants (Finazzi et al., 2004). This reaction centre quenching was suggested to be a common transient characteristic during illumination depending on the balance between light and carbon fixation fluxes (Finazzi et al., 2004).

Our recent data also implicate reaction centre quenching as an alternative mechanism for nonradiative dissipation of excess light energy (Ivanov et al., 2003). From this work it is evident that exposure to low temperature results in major alterations in the redox properties of the acceptor side of PSII in photosynthetic organisms as diverse as the cyanobacterium *Synechococcus* sp. PCC 7942 (Sane et al., 2002), the conifer *Pinus sylvestris* (Ivanov et al., 2001, 2002), the model plant species *Arabidopsis thaliana* (Sane et al., 2003) and barley (Ivanov et al., 2006). The shifts in the characteristic T_M of $S_2Q_A^-$ and $S_2Q_B^-$ recombinations in cold-acclimated *Arabidopsis* and barley plants with the Q_A^- and Q_B^- associated peaks appearing at higher and lower temperatures respectively (Sane et al. 2003; Ivanov et al., 2006), imply substantial changes in the activation energies associated with de-trapping of the electron from reduced Q_A and Q_B (Fig. 2). Similar changes in the redox properties of PSII associated with a downshift in the T_M of the B-band ($S_2Q_B^-$) have been reported in cold acclimated spinach (Briantais et al., 1992) and more recently in low temperature grown maize (Janda et al., 2000). Because the activation energies have been shown to be directly related to the redox potentials of the participating species (Devault and Govindjee, 1990), narrowing the temperature gap between the characteristic T_M for Q_A and Q_B reflects a narrowing of the redox potential gap between Q_A and Q_B as a result of cold acclimation (Fig. 2). Furthermore, the high temperature shift in the T_M of $S_2Q_A^-$ corresponding to increased activation energy of Q_A/Q_A^- (Sane et al., 2003; Ivanov et al., 2006) would increase the free energy gap between Q_A^- and $P680^+$. This could cause stabilization of $S_2Q_A^-$ and decrease the probability for the back reaction through $P680^+Pheo^-$ (Minagawa et al., 1999; Vavilin and Vermaas, 2000). Moreover, the preferential localization of the electron on Q_A in cold acclimated *Arabidopsis* could also result from a change in the redox potential of Q_B. Lowering the redox potential of Q_B will narrow the gap between the redox potentials between Q_A and Q_B even further and will decrease the probability for electron transfer between the two quinone acceptors by shifting the redox equilibrium between Q_A^- Q_B and $Q_AQ_B^-$ towards $Q_A^-Q_B$ (Minagawa et al., 1999). The retention of electrons preferentially on Q_A through a modification of the redox potentials of Q_A and Q_B in opposite directions would inhibit the reoxidation of Q_A^- by either forward or back electron flow (Mäenpää et al., 1995). This would ensure that the Q_B site remains occupied by a quinone, which would protect PSII from photoinhibition and D1 degradation (Ohad and Hirschberg, 1992). More detailed studies of DCMU effects on the redox properties of PSII acceptor side have demonstrated that DCMU binding increases the free energy gap between $P680^+Pheo^-$ and $P680^+Q_A^-$ by raising the redox potential of Q_A (Krieger-Liszkay and Rutherford, 1998; Fufezan et al., 2002). It was suggested that this would favour a direct non-radiative charge recombination pathway without the formation of singlet oxygen (Krieger-Liszkay and Rutherford, 1998; Fufezan et al., 2002). Supporting evidence for this argument comes from experiments in which the addition of DCMU had a protective effect on D1 turnover under photoinhibitory conditions (Komenda and Masojidek, 1998). When the Q_B site is occupied in the presence of DCMU and Q_A is in a reduced state, PSII shows increased resistance to photoinhibition.

In this regard, it is important to note that acclimation to low temperatures is strongly correlated with an increased proportion of reduced Q_A at the given growth temperature (Huner et al., 1993; Huner et al., 1998). Hence, it seems very likely that the increased population of Q_A^- due to the altered redox potentials of Q_A and Q_B during the shift and acclimation to low temperature may enhance the dissipation of excess light within the reaction centre of PSII via non-radiative $P680^+Q_A^-$ recombination, protecting the Q_A site from excessive excitation pressure (Huner et al., 1998; Öquist and Huner, 2003). However, upon extension of our results on low temperature acclimation in cyanobacteria, *Arabidopsis* and pine, our recent results on the energy partitioning in barley indicated that the probability for non-radiative dissipation of excess energy (reaction centre quenching) increases not only in cold acclimated plants, but also in plants acclimated to high growth irradiance. Since either low temperature or high light induced a comparable reduction state of Q_A, this implies that reaction centre quenching is correlated with the excitation pressure (Ivanov et al., 2006). Thus, it would now appear that any environmental condition which increases the reduction state of Q_A will enhance the probablility of photoprotection through reaction centre quenching.

MOLECULAR MECHANISMS REGULATING REACTION CENTRE QUENCHING

Protein Modifications

Considering that conditions favouring nonradiative pathway of energy dissipation in PSII reaction centres could be induced by changes in the redox properties of both the donor and acceptor sites of PSII, it is clear that certain structural modifications to the molecular organization and/or local electrostatic environment of the water splitting complex and the binding sites of Q_A and Q_B electron acceptors could be involved. It has been well documented that donor side dependent shifts in the redox properties of Q_A could be induced by modification in structural and/or functional integrity of the donor side during photoactivation of the Mn cluster in PSII (Johnson et al., 1995) and by Ca-depletion of the oxygen evolving complex (Krieger and Rutherford, 1997; Krieger et al., 1995). Furthermore, a single change in a crucial amino acid residue on the donor side of D1 (Minagawa et al., 1999; Ohad and Hirschberg, 1992) or a deletion of the PEST-like sequence of D1 (Nixon et al., 1995) has been shown to result in shifts of the $S_2Q_B^-$ TL peak towards lower temperatures. In addition, over-reduction of the S-states *in vitro* might also influence the $S_2Q_A^-$ recombination (Messinger et al., 1997; Schmid et al., 1994). Interestingly, in addition to these earlier *in vitro* reports, formation of super-reduced states (S_{-1} and S_{-2}) was recently reported to occur *in vivo* (Quigg et al., 2003). The generation and accumulation of the over-reduced state (S_{-1}) and super-reduced state (S_{-2}) due to backward transition of the S-states (Messinger et al., 1997; Quigg et al., 2003) would increase the population of PSII reaction centres retaining reduced Q_A, which is believed to be prerequisite for the induction of nonradiative reaction centre quenching (Bukhov et al., 2001).

In higher plants, the D1 polypeptide of PSII is subject to at least five post-translational modifications: C-terminal processing in the conversion of 34 kDa precursor polypeptide to the 32 kDa mature polypeptide; removal of the initiating methionine residue; N-acetylation of the N-terminal threonine residue; covalent palmitoylation mapped to the N-terminal two thirds of the polypeptide and, finally, reversible phosphorylation of the N-terminal threonine catalyzed by a light-dependent, redox-regulated kinase (Mattoo et al., 1993; Rintamärki and Aro, 2001). Although the functional role of D1 palmitoylation remains unknown, palmitoylation has been shown to regulate signal transduction through G-protein linked receptors by regulating protein-protein interactions (Milligan et al., 1995). There is no evidence for the role of D1 palmitoylation in altering the T_M for $S_2/S_3 - Q_A^-/Q_B^-$ recombinations. However, alterations in protein-protein interactions within PSII may be important since *Arabidopsis thaliana npq4* mutants lacking only the PsbS protein exhibit significant downshifts in the T_M for $S_2/S_3 - Q_A^-/Q_B^-$ recombinations under normal growth conditions (Huner et al., 2006). Recent studies demonstrating that decreased oligomerization of LHCII proteins in *Costata 2/133* pea mutant correlates with increased probability for reaction center quenching (Ivanov et al., 2005) and reaction center-type energy quenching depends on PSII antenna size (Zulfugarov et al., 2007) also support the importance of protein-protein interactions within PSII complex in modulating the redox properties of PSII acceptor side.

During the PSII damage-repair cycle in higher plants, both the D1 and D2 reaction centre polypeptides undergo reversible phosphorylation. The extent of D1 phosphorylation appears to be regulated by excitation pressure (Rintamärki and Aro, 2001) as well as by an endogenous circadian rhythm (Booij-James et al., 2002). Site-directed mutagenesis of *PsbA* in *Synechocystis* PCC 6803 indicates that alterations in a single amino acid can result in significant changes in the T_M for $S_2/S_3 - Q_A^-/Q_B^-$ recombinations (Minagawa et al., 1999; Vavilin and Vermaas, 2000). Thus, it is conceivable that post-translational modification of D1 and/or D2 PSII reaction centre polypeptides by either palmitoylation or phosphorylation may alter the local conformation and/or the electrostatic environment of the Q_A and Q_B binding sites localised on these polypeptides. This, in turn, may result in shifts in the T_M for $S_2/S_3 - Q_A^-/Q_B^-$ recombinations and hence the changes in the redox potentials of Q_A and Q_B. Unlike seed plants, no phosphorylation of the D1 polypeptide has been detected in *Chlamydomonas reinhardtii* (Rintamärki and Aro, 2001) but the decrease in HCO_3^- concentrations in the chloroplast under saturating irradiance has also been shown to affect the redox potentials of Q_A and Q_B (Demeter et al., 1995; Govindjee, 1993). Thus, limitation of chloroplastic HCO_3^- concentrations may also contribute to modulating the redox potentials of Q_A and Q_B through its regulatory role in the electron flow from Q_A to Q_B, acting as a fifth ligand to the nonheme Fe in PSII (Hienerwadel and Berthomieu, 1995). In the recent structural model of the PSII reaction centre, the bicarbonate anion is positioned close to $D1Tyr^{246}$ and $D2Lys^{264}$, which could stabilize it by hydrogen bonding (Ferreira et al., 2004).

Lipid Environment

While environmental modulation of D1 protein exchange (Sane et al., 2002) and various post-translation modifications of PSII polypeptides mentioned above may well be responsible for alteration of the redox properties of the acceptor site of PSII, changes in the lipid environment surrounding D1/D2 protein complex may also affect the redox characteristics of the electron accepting quinones Q_A and Q_B. Growth temperature and growth irradiance have a significant impact on the lipid and fatty acid composition of thylakoid membranes of cyanobacteria and higher plants (Harwood, 1998; Los and Murata, 2002; Nishida and Murata, 1996). The contribution of specific lipid classes and the unsaturation of membrane lipids to enhance the tolerance of the photosynthetic machinery of cyanobacteria and higher plants towards chilling and high light stress have been well established and reviewed (Nishida and Murata, 1996).

Crystallographic X-ray diffraction analysis (at 2.55 Å resolution revealed close interactions between three lipid molecules (cardiolipin, phosphatidylcholine, glucosylgalactosyl diacylglycerol) and the reaction centre of *Rhodobacter sphaeroides* (Camara-Artigas et al., 2002). The lipid molecules were localized on the surface of the protein at three distinct sites: the glycolipid chains make contact with the isoprenoid chain of Q_A; the phosphatidylcholine binds at

the interface between L and M subunit and comes in contact with the isoprenoid chain of Q_B which lies 9.5 Å apart from the lipid; the polar group of the cardiolipin is over 15 Å from any of the electron accepting quinones. Based on the structural model, it has been suggested that specific lipid-protein interactions may affect the functioning of the electron accepting quinones within the reaction centre (Camara-Artigas et al., 2002). In support of this, an earlier study of the charge recombination process in PSII demonstrated that lipid rigidity modulates $P680^+Q_A^-$ recombination in *Rhodopseudomonas viridis* and *Rhodobacter sphaeroides* (Sebban et al., 1991). In addition, a very recent study demonstrated that the redox midpoint potential (E_m) of Q_A in bacterial reaction centres was significantly lowered by the native lipid cardiolipin (Rinyu et al., 2004). This effect was discussed in terms of a specific lipid induced modification of the structure of the Q_A binding site (Rinyu et al., 2004). In addition, lipid phase transitions also affect the second electron transfer reaction in bacterial reaction centres by modifying the free energy gap between Q_A and Q_B, most probably *via* an electrostatic effect due to the segregation of the reaction centre proteins (Taly et al., 2002).

Precise information of the specific lipid binding sites within the PSII reaction centre complex of higher plants are still not available, but it is well known that chloroplast lipids play a key role in the assembly and functioning of PSII (Nishida and Murata, 1996; Pali et al., 2003). For example, the phospholipid phosphatidylglycerol (PG) has been suggested to be a functional effector and membrane anchor of the D1 protein in the PSII core complex (Kruse and Schmid, 1995). It has been also shown that PG is directly involved in the dimerization of the D1/D2 heterodimer of PSII (Kruse et al., 2000) and in the function of the secondary electron acceptor, Q_B, in the PSII reaction centres (Gombos et al., 2002). In a recent study Sakurai et al. (2003) demonstrated that the absence of PG increased the susceptibility of *Synechocystis* sp. PCC6803 mutant (*pgsA*) cells to photoinhibition due to impaired dimerization of PSII core monomers and the reactivation of photoinhibited PSII core complexes. Furthermore, Dobrikova et al. (1997) showed that the asymmetric surface charge distribution and electric polarizability of thylakoid membranes are significantly altered in the *fad*B and the *fad*C mutants of *Arabidopsis* that are deficient in lipid fatty acid desaturases. In addition, the *dgd1* mutant of *Arabidopsis*, deficient in digalactosyldiacylglycerol (DGDG), exhibits modified properties for the oxygen evolving complex, providing direct evidence that specific lipids may play an important role for the functional and/or structural integrity of the water oxidizing complex (Reifarth et al., 1997). Thus, it is conceivable that light- and temperature-induced changes in the thylakoid lipid and fatty acid composition could also alter the microenvironment of PSII reaction centres by altering lipid-protein interactions causing a shift in the T_M for the Q_A and Q_B recombinations.

SUMMARY

We conclude that, as originally suggested by Krause and Weis (1991), both reaction centre and antenna quenching function *in vivo* to different extents to protect PSII from photodamage depending on the species as well as the environmental conditions. Reaction centre quenching may replace antenna based non-photochemical quenching and enhance the protection of PSII against photoinhibition induced either by high light or low temperature in organisms such as cyanobacteria which lack xanthophyll-cycle-dependent antenna quenching and complement the capacity for antenna quenching under conditions where the enzyme-dependent xanthophyll cycle is thermodynamically restricted (Öquist and Huner, 2003). Any condition which increases the reduction state of Q_A, enhances the probability of reaction centre quenching. However, further research is required not only to assess the contribution of any one of these mechanisms to the shifts in the T_M for $S_2/S_3 - Q_A^-/Q_B^-$ recombinations and hence reaction centre quenching but also to assess the timing for the onset of reaction centre quenching versus antenna quenching associated with NPQ during exposure to increased excitation pressure.

ACKNOWLEDGEMENTS

This work was financially supported by the Natural Science and Engineering Research Council of Canada to NPAH, the Swedish Foundation for International Cooperation in Research and Higher Education (STINT) to GÖ and NPAH, and by the Swedish Research Council to GÖ and the Swedish Council for Forestry and Agricultural Research to VH.

Received December 10,2007; accepted January 15, 2008.

LITERATURE CITED

Adams WW, Demmig-Adams B, Rosenstiel TN, Ebbert V (2001) Dependence of photosynthesis and energy dissipation activity upon growth form and light environment during the winter. Photosynth Res 67: 51-62

Allakhverdiev SI, Klimov VV, Carpentier R (1997) Evidence for the involvement of cyclic electron transport in the protection of photosystem II against photoinhibition: Influence of a new phenolic compound. Biochemistry 36: 4149-4154

Allen JF (1995) Thylakoid protein phosphorilation, state 1- state 2 transitions, and phosystem stoichiometry adjustment: Redox control at multiple levels of gene expression. Physiol Plant 93: 196-205

Anderson JM (1986) Photoregulation of the composition, function and structure of thylakoid membranes. Annu Rev Plant Physiol 37: 93-136

Anderson JM (1992) Cytochrome b_6/f complex: Dynamic molecular organization, function and acclimation. Photosynth Res 34: 341-357

Aro E-M, Virgin I, Andersson B (1993) Photoinhibition of photosystem II. Inactivation, protein damage and turnover. Biochim Biophys Acta 1143: 113-134

Asada K (1999) The water-water cycle in chloroplasts: scavenging of active oxygens and dissipation of excess photons. Annu Rev Plant Physiol Plant Mol Biol 50: 601-639

Aspinall-O'Dea M, Wentworth M, Pascal A, Robert B, Ruban A, Horton P (2002) *In vitro* reconstitution of the activated zeaxan-

thin state associated with energy dissipation in plants. Proc Natl Acad Sci USA 99: 16331-16335

Barber J, De Las Rivas J (1993) A functional model for the role of cytochrome-b_{559} in the protection against donor and acceptor side photoinhibition. Proc Natl Acad Sci USA 90: 10942-10946

Bassi R, Croce R, Cugini D, Sandona D (1999) Mutational analysis of a higher plant antenna protein provides identification of chromophores bound in multiple sites. Proc Natl Acad Sci USA 96: 10056-10061

Booij-James IS, Swegle WM, Edelman M, Mattoo AK (2002) Phosphorylation of the D1 photosystem II reaction center protein Is controlled by an endogenous circadian rhythm. Plant Physiol 130: 2069-2075

Briantais J-M, Dueruet J-M, Hodges M, Krause GH (1992) The effects of low temperature acclimation and photoinhibitory treatments on Photosystem 2 studied by thermoluminescence and fluorescence decay kinetics. Photosynth Res 31: 1-10

Briantais J-M, Vernotte C, Picaud M, Krause GH (1979) A quantitative study of the slow decline of chlorophyll fluorescence in isolated chloroplasts. Biochim Biophys Acta 548: 128-138

Bruce D, Samson G, Carpenter C (1997) The origins of nonphotochemical quenching of chlorophyll fluorescence in photosynthesis. Direct quenching by $P680^+$ in photosystem II enriched membranes at low pH. Biochemistry 36: 749-755

Bukhov NG, Heber U, Wiese C, Shuvalov VA (2001) Energy dissipation in photosynthesis: Does the quenching of chlorophyll fluorescence originate from antenna complexes of photosystem II or from the reaction center? Planta 212: 749-758

Butler WL (1978) Energy distribution in the photochemical apparatus of photosynthesis. Annu Rev Plant Physiol 29: 345-378

Camara-Artigas A, Brune D, Allen JP (2002) Interactions between lipids and bacterial reaction centers determined by protein crystallography. Proc Nat Acad Sci USA 99: 11055-11060

Delphin E, Duval JC, Etienne AL, Kirilovsky D (1996) State transitions or ΔpH-dependent quenching of photosystem II fluorescence in red algae. Biochemistry 35: 9435-9445

Delrieu MJ (1998) Regulation of thermal dissipation of absorbed excitation energy and violaxanthin deepoxidation in the thylakoids of *Lactuca sativa*. Photoprotective mechanism of a population of photosystem II centers. Biochim Biophys Acta 1363: 157-173

Demeter S, Janda T, Kovacs L, Mende D, Wiessner W (1995) Effects of *in vivo* CO_2 depletion on electron transport and photoinhibition in the green algae, *Chlamydobotrys stellata* and *Chlamydomonas reinhardtii*. Biochim Biophys Acta 1229: 166-174

Demmig B, Winter K, Krüger A, Czygan F-C (1987) Photoinhibition and zeaxanthin formation in intact leaves: a possible role of the xanthophyll cycle in the dissipation of excess light. Plant Physiol 84: 218-224

Demmig-Adams B (1990) Carotenoids and photoprotection: a role for the xanthophyll zeaxanthin. Biochim Biophys Acta 1020: 1-24

Demmig-Adams B, Adams WW (1992) Photoprotection and other responses of plants to high light stress. Annu Rev Plant Physiol Plant Mol Biol 43: 599-626

Devault D, Govindjee (1990) Photosynthetic glow peaks and their relationship with the free-energy changes. Photosynth Res 24: 175-181

Dobrikova A, Taneva SG, Busheva M, Apostolova E, Petkanchin I (1997) Surface electric properties of thylakoid membranes from *Arabidopsis thaliana* mutants. Biophys Chem 67: 239-244

Doege M, Ohmann E, Tschiersch H (2000) Chlorophyll fluorescence quenching in the alga *Euglena gracilis*. Photosynth Res 63: 159-170

Dominici P, Caffarri S, Armenante F, Ceoldo S, Crimi M, Bassi R (2002) Biochemical properties of the psbS subunit of photosystem II either purified from chloroplast or recombinant. J Biol Chem 277: 22750-22758

Falkowski PG, Fujita Y, Ley A, Mauzerall D (1986) Evidence for cyclic electron flow around photosystem II in *Chlorella pyrenoidosa*. Plant Physiol 81: 310-312

Ferreira KN, Iverson TM, Maghlaoui K, Barber J, Iwata S (2004) Architecture of the photosynthetic oxygen-evolving center. Science 303: 1831-1838

Field TS, Nedbal L, Ort DR (1998) Nonphotochemical reduction of the plastoquinone pool in sunflower leaves originates from chlororespiration. Plant Physiol 116: 1209-1218

Finazzi G, Johnson GN, Dallosto L, Joliot P, Wollman F-A, Bassi R (2004) A zeaxanthin-independent nonphotochemical quenching mechanism localized in the photosystem II core complex. Proc Natl Acad Sci USA 101: 12375-12380

Fufenzan C, Rutherford AW, Krieger-Liszkay A (2002) Singlet oxygen production in herbicide-treated photosystem II. FEBS Lett 532: 407-410

Funk C, Adamska I, Green BR, Andersson B, Renger G (1995a) The nuclear-encoded chlorophyll-binding photosystem II-S protein is stable in the absence of pigments. J Biol Chem 270: 30141-30147

Funk C, Schröder WP, Napiwotzki A, Tjus SE, Renger G, Andersson B (1995b) The PSII-S protein of higher plants: a new type of pigment-binding protein. Biochemistry 34: 11133-11141

Gilmore AM (1997) Mechanistic aspects of xanthophyll cycle-dependent photoprotection in higher plant chloroplasts and leaves. Physiol Plant 99: 197-209

Gilmore AM, Ball MC (2000) Protection and storage of chlorophyll in overwintering evergreens. Proc Natl Acad Sci USA 97: 11098-11101

Gombos Z, Varkonyi Z, Hagio M, Iwaki M, Kovacs L, Masamoto K, Itoh S, Wada H (2002) Phosphatidylglycerol requirement for the function of electron acceptor plastoquinone Q_B in the photosystem II reaction center. Biochemistry 41: 3796-3802

Govindjee (1993) Bicarbonate-reversible inhibition of plastoquinone reductase in photosystem-II. Z Naturforsch 48C: 251-258

Grasses T, Pesaresi P, Schiavon F, Varotto C, Salamini F, Jahns P, Leister D (2002) The role of pH-dependent dissipation of excitation energy in protecting photosystem II against light-induced damage in *Arabidopsis thaliana*. Plant Physiol Biochem 40: 41-49

Greer DH, Berry JA, Björkman O (1986) Photoinhibition and photosynthesis in intact bean leaves: role of light and temperature, and requirement for chloroplast-protein synthesis during recovery. Planta 168: 253-260

Haldrup A, Jensen PE, Lunde C, Scheller HV (2001) Balance of power: a view of the mechanism of photosynthetic state transitions. Trends Plant Sci 6: 301-305

Harwood JL (1998) Involvement of chloroplast lipids in the reaction of plants submitted to stress. *In* P-A Siegenthaler, ed, Advances in photosynthesis and respiration. Lipids in photosynthesis: structure, function and genetics, Vol. VI. Kluwer Academic Publishers, Dordrecht, pp 287-302

Hienerwadel R, Berthomieu C (1995) Bicarbonate binding to the non-heme iron of photosystem II investigated by Fourier transform infrared difference spectroscopy and ^{13}C-labeled bicarbonate. Biochemistry 34: 16288-16297

Hillmann B, Brettel K, van Mieghem F, Kamlowski A, Rutherford AW, Schlodder E (1995) Charge recombination reactions in photosystem-II. 2. Transient absorbency difference spectra and their temperature-dependence. Biochemistry 34: 4814-4827

Horton P, Ruban AV, Walters RG (1996) Regulation of light harvesting in green plants. Annu Rev Plant Physiol Plant Mol Biol 47:

655-684

Huner NPA, Ivanov AG, Sane PV, Pocock T, Krol M, Balseris A, Rosso D, Savitch LV, Hurry VM, Öquist G (2006) Photoprotection of photosystem II: reaction centre quenching versus antenna quenching. *In* B Demming-Adams, WW AdamsIII, AK Mattoo, eds, Advances in Photosynthesis and Respiration: Photoprotection, Photoinhibition, Gene Regulation and Environment, Vol. 21 Springer, Dordrecht, pp 155-173

Huner NPA, Öquist G, Hurry VM, Krol M, Falk S, Griffith M (1993) Photosynthesis, photoinhibition and low temperature acclimation in cold tolerant plants. Photosynth Res 37: 19-39

Huner NPA, Öquist G, Sarhan F (1998) Energy balance and acclimation to light and cold. Trends Plant Sci 3: 224-230

Hurry VM, Huner NPA (1992) Effect of cold hardening on sensitivity of winter and spring wheat leaves to short-term photoinhibition and recovery of photosynthesis. Plant Physiol 100: 1283-1290

Hurry VM, Strand A, Tobiaeson M, Gardeström P, Öquist G (1995) Cold hardening of spring and winter wheat and rape results in differential effects on growth, carbon metabolism, and carbohydrate content. Plant Physiol 109: 697-706

Ivanov AG, Sane P, Hurry V, Krol M, Sveshnikov D, Huner NPA, Öquist G (2003) Low-temperature modulation of the redox properties of the acceptor side of photosystem II: photoprotection through reaction centre quenching of excess energy. Physiol Plant 119: 376-383

Ivanov AG, Sane PV, Zeinalov Y, Malmberg G, Gardeström P, Huner NPA, Öquist G (2001) Photosynthetic electron transport adjustments in overwintering Scots pine (*Pinus sylvestris* L.). Planta 213: 575-585

Ivanov AG, Sane PV, Zeinalov Y, Simidjiev I, Huner NPA, Öquist G (2002) Seasonal responses of photosynthetic electron transport in Scots pine (*Pinus sylvestris* L.) studied by thermoluminescence. Planta 215: 457-465

Ivanov AG, Krol M, Apostolova EL, Morgan-Kiss RM, Naydenova N, Huner NPA, Sane PV (2005) Oligomerization state of LHCII modulates the redox properties of the acceptor side of photosystem II in *Costata-2/133* mutant of pea. Physiol Mol Biol Plants 11: 199-207

Ivanov AG, Sane PV, Krol M, Gray GR, Balseris A, Savitch LV, Öquist G, Hüner NPA (2006). Acclimation to temperature and irradiance modulates PSII charge recombination. FEBS Lett 580: 2797-2802

Janda T, Szalai G, Paldi E (2000) Thermoluminescence investigation of low temperature stress in maize. Photosynthetica 38: 635-639

Johnson GN, Rutherford AW, Krieger A (1995) A change in the midpoint potential of the quinone Q_a in photosystem-II associated with photoactivation of oxygen evolution. Biochim Biophys Acta 1229: 202-207

Jursinic P, Govindgee (1982) Effects of hydroxylamine and silicomolybdate on the decay in delayed light emission in the 6-100 microsecond range after a single 10 ns flash in pea thylakoids. Photosynth Res 3: 161-177

Kim J-H, Kim S-J, Cho SH, Chow WS, Lee C-H (2005) Photosystem I acceptor side limitation is a prerequisite for the reversible decrease in the maximum extent of P700 oxidation after short-term chilling in the light in four plant species with different chilling sensitivities. Physiol Plant 123: 100-107

Komenda J, Masojidek J (1998) The effect of photosystem II inhibitors DCMU and BNT on the high-light induced D1 turnover in two cyanobacterial strains *Synechocystis* PCC 6803 and *Synechococcus* PCC 7942. Photosynth Res 57: 193-202

Kozaki A, Takeba G (1996) Photorespiration protects C_3 plants from photoinhibition. Nature 384: 557-560

Krause GH (1988) Photoinhibition of photosynthesis. An evaluation of damaging and protective mechanisms. Physiol Plant 74: 566-574

Krause GH, Jahns P (2003) Pulse amplitude modulated chlorophyll fluorometry and its application in plant science. *In* BR Green, WW Parson, eds, Advances in photosynthesis and respiration. Light harvesting antennae in photosynthesis, Vol. XIII. Kluwer Academic Publishers, Dordrecht, pp 373-399

Krause GH, Weis E (1991) Chlorophyll fluorescence and photosynthesis: The basics. Annu Rev Plant Physiol Plant Mol Biol 42: 313-349

Krieger A, Moya I, Weis E (1992) Energy-dependent quenching of chlorophyll a fluorescence - effect of pH on stationary fluorescence and picosecond-relaxation kinetics in thylakoid membranes and photosystem-II preparations. Biochim Biophys Acta 1102: 167-176

Krieger A, Rutherford AW (1997) Comparison of chloride-depleted and calcium-depleted PSII: The midpoint potential of Q_A and susceptibility to photodamage. Biochim Biophys Acta 1319: 91-98

Krieger A, Rutherford AW, Johnson GN (1995) On the determination of redox midpoint potential of the primary quinone electron-acceptor, Q_a, in Photosystem-II. Biochim Biophys Acta 1229: 193-201

Krieger A, Weis E (1993) The role of calcium in the pH-dependent control of photosystem II. Photosynth Res 37: 117-130

Krieger A, Weis E, Demeter S (1993) Low-pH induced Ca^{2+} ion release in the water-splitting system is accompanied by a shift in the midpoint redox potential of the primary quinone acceptor Q_A. Biochim Biophys Acta 1144: 411-418

Krieger-Liszkay A, Rutherford AW (1998) Influence of herbicide binding on the redox potential of the quinone acceptor in photosystem - II. Relevance to photodamage and phytotoxicity. Biochemistry 37: 17339-17344

Kruse O, Hankamer B, Konczak C, Gerle C, Morris E, Radunz A, Schmid GH, Barber J (2000) Phosphatidylglycerol is involved in the dimerization of photosystem II. J Biol Chem 275: 6509-6514

Kruse O, Schmid GH (1995) The role of phosphatidylglycerol as a functional effector and membrane anchor of the D1 core peptide from photosystem II particles of the cyanobacterium *Oscillatoria chalybea*. Z Naturforsch 50C: 380-390

Kruse O, Zheleva D, Barber J (1997) Stabilization of photosystem two dimers by phosphorylation: Implication for the regulation of the turnover of D1 protein. FEBS Lett 408: 276-280

Kühlbrandt W, Wang DN, Fujiyoshi Y (1994) Atomic model of plant light-harvesting complex by electron crystallography. Nature 367: 614-621

Lee HY, Hong YN, Chow WS (2001) Photoinactivation of photosystem II complexes and photoprotection by non-functional neighbours in *Capsicum annuum* L. leaves. Planta 212: 332-342

Li X-P, Björkman O, Shin C, Grossman A, Rosenquist M, Jansson S, Niyogi KK (2000) A pigment-binding protein essential for regulation of photosynthetic light harvesting. Nature 403: 391-395

Liu B, Napiwotzki A, Eckert H-J, Eichler HJ, Renger G (1993) Studies on the recombination kinetics of the radical pair $P680^+Pheo^-$ in isolated PSII core complexes from spinach. Biochim Biophys Acta 1142: 129-138

Long SP, Humphries S, Falkowski PG (1994) Photoinhibition of photosynthesis in nature. Annu Rev Plant Physiol Plant Mol Biol 45: 633-642

Los DA, Murata N (2002) Sensing and responses to low temperature in cyanobacteria. *In* KB Storey, JM Storey, eds, Sensing, signalling and cell adaptation, Elsevier Science BV, Amsterdam, pp 139-153

Lunde C, Jensen PE, Haldrup A, Knoetzel J, Scheller HV (2000) The PSI-H subunit of photosystem I is essential for state transitions in plant photosynthesis. Nature 408: 613-615

Mäenpää P, Miranda T, Tyystjarvi E, Tyystjarvi T, Govindjee, Ducruet JM, Etienne AL, Kirilovsky D (1995) A mutation in the D-loop of D1 modifies the stability of the $S_2Q_A^-$ and $S_2Q_B^-$ states in photosystem-II. Plant Physiol 107: 187-197

Matsubara S, Chow WS (2004) Populations of photoinactivated photosystem II reaction centers characterized by chlorophyll a fluorescence lifetime *in vivo*. Proc Natl Acad Sci USA 101: 18234-18239

Mattoo AK, Elich TD, Ghirardi ML, Callahan FE, Edelman M (1993) Post-translational modification of chloroplast proteins and the regulation of protein turnover. *In* NH Battey, HG Dickinson, AM Hetherington, eds, Post-translational modification in plants, Vol. 53. Cambridge University Press, Cambridge, pp 65-78

Melis A (1999) Photosystem II damage and repair cycle in chloroplasts: what modulates the rate of photodamage *in vivo*? Trends Plant Sci 4: 130-135

Messinger J, Seaton G, Wydrzynski T, Wacker U, Renger G (1997) S-3 state of the water oxidase in photosystem II. Biochemistry 36: 6862-6873

Milligan G, Parenti M, Magee AI (1995) The dynamic role of palmitoylation in signal transduction. Trends Biochem Sci 20: 256-256

Minagawa J, Narusaka Y, Inoue Y, Satoh K (1999) Electron transfer between Q_A and Q_B in photosystem II is thermodynamically perturbed in phototolerant mutants of *Synechocystis* sp. PCC 6803. Biochemistry 38: 770-775

Miyake C, Yokota A (2001) Cyclic flow of electrons within PSII in thylakoid membranes. Plant Cell Physiol 42: 508-515

Miyake C, Yonekura K, Kobayashi Y, Yokota A (2002) Cyclic electron flow within PSII functions in intact chloroplasts from spinach leaves. Plant Cell Physiol 43: 951-957

Nield J, Funk C, Barber J (2000) Supermolecular structure of photosystem II and location of the PsbS protein. Phil Trans Royal Soc London Ser B-Biol Sci 355: 1337-1343

Nishida I, Murata N (1996) Chilling sensitivity in plants and cyanobacteria: the crucial contribution of membrane lipids. Annu Rev Plant Physiol Plant Mol Biol 47: 541-568

Nixon PJ, Komenda J, Barber J, Deak Z, Vass I, Diner BA (1995) Deletion of the Pest-like region of photosystem 2 modifies the Q_B-binding pocket but does not prevent rapid turnover of D1. J Biol Chem 270: 14919-14927

Niyogi KK (1999) Photoprotection revisited: Genetic and molecular approaches. Annu Rev Plant Physiol Plant Mol Biol 50: 333-359

Ohad I, Hirschberg J (1992) Mutations in the D1 subunit of photosystem II distinguish between quinone and herbicide binding sites. Plant Cell 4: 273-282

Olaizola M, Laroche J, Kolber Z, Falkowski PG (1994) Nonphotochemical fluorescence quenching and the diadinoxanthin cycle in a marine diatom. Photosynth Res 41: 357-370

Olaizola M, Yamamoto HY (1994) Short-term response of the diadinoxanthin cycle and fluorescence yield to high irradiance in *Chaetoceros muelleri* (Bacillariophyceae). J Phycol 30: 606-612

Öquist G, Chow WS, Anderson JM (1992) Photoinhibition of photosynthesis represents a mechanism for long-term regulation of photosystem II. Planta 186: 450-460

Öquist G, Huner NPA (2003) Photosynthesis of overwintering evergreen plants. Annu Rev Plant Biol 54: 329-355

Ort DR (2001) When there is too much light. Plant Physiol 125: 29-32

Ort DR, Baker NR (2002) A photoprotective role for O_2 as an alternative electron sink in photosynthesis? Curr Opin Plant Biol 5: 193-198

Osmond, B (1981) Photorespiration and photoinhibition: some implications for the energetics of photosynthesis. Biochim Biophys Acta 639: 77-98

Pali T, Garab G, Horvath LI, Kota Z (2003) Functional significance of the lipid-protein interface in photosynthetic membranes. Cell Mol Life Sci 60: 1591-1606

Peterson RB, Havir EA (2001) Photosynthetic properties of an *Arabidopsis thaliana* mutant possessing a defective PsbS gene. Planta 214: 142-152

Peterson RB, Havir EA (2003) Contrasting modes of regulation of PSII light utilization with changing irradiance in normal and PsbS mutant leaves of *Arabidopsis thaliana*. Photosynth Res 75: 57-70

Powles SB (1984) Photoinhibition of photosynthesis induced by visible light. Annu Rev Plant Physiol 35: 15-44

Prasil O, Kolber Z, Berry JA, Falkowski PG (1996) Cyclic electron flow around photosystem II *in vivo*. Photosynth Res 48: 395-410

Quigg A, Beardall J, Wydrzynski T (2003) Photoacclimation involves modulation of the photosynthetic oxygen-evolving reactions in *Dunaliella tertiolecta* and *Phaeodactylum tricornutum*. Funct Plant Biol 30: 301-308

Quiles MJ (2006) Stimulation of chlororespiration by heat and high light intensity in oat plants. Plant Cell Environ 29: 1463-1470

Reifarth F, Christen G, Seeliger AG, Dormann P, Benning C, Renger G (1997) Modification of the water oxidizing complex in leaves of the *dgd1* mutant of *Arabidopsis thaliana* deficient in the galactolipid digalactosyldiacylglycerol. Biochemistry 36: 11769-11776

Richter M, Goss R, Wagner B, Holzwarth AR (1999) Characterization of the fast and slow reversible components of non-photochemical quenching in isolated pea thylakoids by picosecond time-resolved chlorophyll fluorescence analysis? Biochemistry 38: 12718-12726

Rintamärki E, Aro E-M (2001) Phosphorylation of photosystem II proteins. *In* E-M Aro, B Andersson, eds, Advances in photosynthesis and respiration, Vol. XI. Kluwer Academic Publishers, Dordrecht, pp 395-418

Rinyu L, Martin EW, Takahashi E, Maroti P, Wraight CA (2004) Modulation of the free energy of the primary acceptor (Q_A) in reaction centers from *Rhodobacter sphaeroides*: contributions from protein and protein-lipid (cardiolipin) interactions. Biochim Biophys Acta 1655: 93-101

Rosso D, Ivanov AG, Fu A, Geisler-Lee J, Hendrickson1 L, Geisler M, Stewart G, Krol M, Hurry V, Rodermel SR, Maxwell DP, Hüner NPA (2006) IMMUTANS does not act as a stress-induced safety valve in the protection of the photosynthetic apparatus of *Arabidopsis thaliana* during steady state photosynthesis. Plant Physiol 142: 574-585

Ruban AV, Pascal AA, Robert B, Horton P (2002) Activation of zeaxanthin is an obligatory event in the regulation of photosynthetic light harvesting. J Biol Chem 277: 7785-7789

Sakurai I, Hagio M, Gombos Z, Tyystjarvi T, Paakkarinen V, Aro EM, Wada H (2003) Requirement of phosphatidylglycerol for maintenance of photosynthetic machinery. Plant Physiol 133: 1376-1384

Sane PV, Ivanov AG, Hurry V, Huner NPA, Öquist G (2003) Changes in the redox potential of primary and secondary electron-accepting quinones in photosystem II confer increased resistance to photoinhibition in low-temperature-acclimated *Arabidopsis*. Plant Physiol 132: 2144-2151

Sane PV, Ivanov AG, Sveshnikov D, Huner NPA, Öquist G (2002) A transient exchange of the photosystem II reaction center protein D1:1 with D1:2 during low temperature stress of *Synechococcus* sp PCC 7942 in the light lowers the redox potential of Q_B. J Biol Chem 277: 32739-32745

Schatz GH, Brock H, Holzwarth AR (1988) Kinetic and energetic model for the primary processes in photosystem II. Biophys J 54: 397-405

Schmid GH, Bader KP, Schulder R (1994) A study on the life time

of the S-3-state in the filamentous cyanobacterium *Oscillatoria chalybea*. Z Naturforsch 49C: 108-114

Schreiber U, Krieger A (1996) Two fundamentally different types of variable chlorophyll fluorescence *in vivo*. FEBS Lett 397: 131-135

Schreiber U, Neubauer C (1990) O_2-dependent electron flow, membrane energization and the mechanism of non-photochemical quenching of chlorophyll fluorescence. Photosynth Res 25: 279-293

Schweitzer RH, Brudvig GW (1997) Fluorescence quenching by chlorophyll cations in photosystem II. Biochemistry 36: 11351-11359

Sebban P, Parot P, Baciou L, Mathis P, Vermeglio A (1991) Effects of low temperature and lipid rigidity on the charge recombination process in *Rps. Viridis* and *Rb. sphaeroides* reaction centres. Biochim Biophys Acta 1057: 109-114

Shatz GH, Brock H, Holzwarth AR (1988) Kinetic and energetic model for the primary processes in photosystem II. Biophys J 54: 397-405

Somersalo S, Krause GH (1990) Reversible photoinhibition of unhardened and cold-acclimated spinach leaves at chilling temperatures. Planta 180: 181-187

Stewart DH, Cua A, Chisholm DA, Diner BA, Bocian DF, Brudvig GW (1998) Identification of histidine 118 in the D1 polypeptide of photosystem II as the axial ligand to chlorophyll Z. Biochemistry 37: 10040-10046

Stitt M, Hurry V (2002) A plant for all seasons: alterations in photosynthetic carbon metabolism during cold acclimation in *Arabidopsis*. Curr Opin Plant Biol 5: 199-206

Strand Å, Foyer CH, Gustafsson P, Gardeström P, Hurry V (2003) Altering flux through the sucrose biosynthesis pathway in transgenic *Arabidopsis thaliana* modifies photosynthetic acclimation at low temperatures and the development of freezing tolerance. Plant Cell Environ 26: 523-535

Strand Å, Hurry V, Gustafsson P, Gardeström P (1997) Development of *Arabidopsis thaliana* leaves at low temperatures releases the suppression of photosynthesis and photosynthetic gene expression despite the accumulation of soluble carbohydrates. Plant J 12: 605-614

Streb P, Josse E-M, Gallouet E, Baptist F, Kuntz M, Cornic G (2005) Evidence for alternative electron sinks to photosynthetic carbon assimilation in the high mountain plant species *Ranunculus glacialis*. Plant Cell Environ 28: 1123-1135

Takahashi Y, Hansson O, Mathis P, Satoh K (1987) Primary radical pair in the photosystem II reaction center. Biochim Biophys Acta 893: 49-59

Taly A, Baciou L, Sebban P (2002) The DMPC lipid phase transition influences differently the first and the second electron transfer reactions in bacterial reaction centers. FEBS Lett 532: 91-96

Telfer A, Dhami S, Bishop SM, Phillips D, Barber J (1994) β-carotene quenches singlet oxygen formed by isolated photosystem II reaction centers. Biochemistry 33: 14469-14474

Telfer A, Rivas JD, Barber J (1991) β-carotene within the isolated photosystem-II reaction center - photooxidation and irreversible bleaching of this chromophore by oxidized P680. Biochim Biophys Acta 1060: 106-114

Thompson LK, Brudvig GW (1988) Cytochrome b_{559} may function to protect photosystem II from photoinhibition. Biochemistry 27: 6653-6658

Tschiersch H, Ohmann E, Doege M (2002) Modification of the thylakoid structure of *Euglena gracilis* by norflurazon-treatment: consequences for fluorescence quenching. Environ Exp Bot 47: 259-270

van Mieghem F, Brettel K, Hillmann B, Kamlowski A, Rutherford AW, Schlodder E (1995) Charge recombination reactions in photosystem II. 1. Yields, recombination pathways, and kinetics of the primary pair. Biochemistry 34: 4798-4813

Vass I, Styring S, Hundal T, Koivuniemi A, Aro E-M, Andersson B (1992) Reversible and irreversible intermediates during photoinhibition of photosystem II: stable reduced Q_A species promote chlorophyll triplet formation. Proc Natl Acad Sci USA 89: 1408-1412

Vavilin DV, Vermaas WFJ (2000) Mutations in the CD-loop region of the D2 protein in Synechocystis sp PCC 6803 modify charge recombination pathways in photosystem II *in vivo*. Biochemistry 39: 14831-14838

Wagner B, Goss R, Richter M, Wild A, Holzwarth AR (1996) Picosecond time-resolved study on the nature of high-energy-state quenching in isolated pea thylakoids - Different localization of zeaxanthin dependent and independent quenching mechanisms. J Photochem Photobiol B 36: 339-350

Walters RG, Horton P (1993) Theoretical assessment of alternative mechanisms for non-photochemical quenching of PSII fluorescence in barley leaves. Photosynth Res 36: 119-139

Wedel, N, Klein R, Ljungderg U, Andersson B, Herrmann RC (1992) The single copy gene PsbS codes foe a phylogenetically intriguing 22 kDa polypeptide of photosystem II. FEBS Lett 314: 61-66

Weis E, Berry JA (1987) Quantum efficiency of photosystem II in relation to 'energy'-dependent quenching of chlorophyll fluorescence. Biochim Biophys Acta 894: 198-207

Woodbury NW, Parson WW, Gunner MR, Prince RC, Dutton PL (1986) Radical-pair energetics and decay mechanisms in reaction centers containing anthraquinones, naphtoquinones or benzoquinones in place of ubiquinone. Biochim Biophys Acta 851: 6-22

Xu CC, Jeon YA, Lee C-H (1999) Relative contributions of photochemical and non-photochemical routes to excitation energy dissipation in rice and barley illuminated at a chilling temperature. Physiol Plant 107: 447-453

Zulfugarov IS, Ham O-K, Misra SR, Kim J-Y, Nath K, Koo H-Y, Kim H-S, Lee C-H (2007) Dependence of reaction center-type energy-dependent quenching on photosystem II antenna size. Biochim Biophys Acta 1767: 773-780