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SOME MECHANISMS OF DAMAGE AND ACCLIMATION OF THE PHOTOSYNTHETIC APPARATUS DUE TO HIGH TEMPERATURE *

Katia Georgieva**

Acad. M. Popov Institute of Plant Physiology, Acad. G. Bonchev Str., Bl. 21, 1113 Sofia, Bulgaria

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Summary. A review is made of recent publications concerning the mechanisms of damage and acclimation of the photosynthetic apparatus due to high temperature. Under stress the organisms undergo first of all destabilisation followed by normalisation and stability, when limit of tolerance are not exceeded and the adaptive capacity is not overtaxed. The influence of heat stress on PS II and PS I activity, lipid composition, protein content, CO_2 assimilation and antioxidant enzymes has been described. Besides the processes leading to plant acclimation to high temperature were discussed.

Key words: acclimation ability, heat stress, photosynthetic activity

Abbreviations: PS I and PS II – photosystem I and photosystem II; LHC II – light-harvesting complex II; HSPs – heat shock proteins

Temperature is one of the main factors limiting the productivity of many crop plants. Understanding the mechanisms of heat tolerance is, therefore, important if we are to improve the crop yield under stress conditions.

In most cases the productivity of a plant is directly related to the rate of photosynthesis. Photosynthesis is, like all other physiological processes, temperature dependent. For a specific plant there exists an optimal temperature at which the net rate of carbon dioxide fixation is maximal. It was found that among all cell functions, the photosynthetic activity of chloroplasts is one of the most heat sensitive (Berry and Björkman, 1980; Quinn and Williams, 1985; Yordanov et al., 1986). The damage due

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** E-mail: katya@obzor.bio21.bas.bg

to heat stress includes a wide range of structural and functional changes. Their effect on growth and survival depends on the intensity and duration of heat stress. A long period at a moderately high temperature may be as injurious as a brief exposure to an extreme temperature.

It is considered that the primary site of damage is associated with components of the photosynthetic system located in the thylakoid membranes, most probably photosystem II (PS II) (Berry and Björkman, 1980; Mamedov et al., 1993; Havaux, 1993). The PS II complex is a pigment-protein complex that utilises light energy to drive the transport of electrons and the oxidation of water to oxygen. It is believed that increasing temperature leads first to a blockage of PSII reaction centres and then to a dissociation of the antenna pigment protein complexes from the central core of the PS II (Armond et al., 1978; Gounaris et al., 1984; Sundby et al., 1986). Separation of the light-harvesting complex II (LHC II) from the core centre induces destacking of the grana and temperature-induced migration of the reaction centre (PS IIβ) or LHC II (state transition) to the non-appressed region, which would have consequences for the energy distribution between PS I and PS II. Many investigations have shown that PS I activity appeared to be much more heat stable than PS II (Pearcy et al., 1977; Sayed et al., 1989; Havaux, 1993). It has been found that moderately high temperatures stimulate PS I activity *in vivo* and *in vitro* (Armond et al., 1978; Sayed et al., 1994). This stimulation appears to be associated with an increased capacity for cyclic electron flow around PS I, which could be an adaptive process, producing ATP under conditions when PS II activity is severely diminished. This ATP synthesis could be important for the survival of plants and necessary for repair of stress damaged processes, as suggested by Janssen et al. (1992).

Among partial reactions of PS II the oxygen-evolving process is particularly sensitive to heat (Santarius, 1975; Thomas et al., 1986; Enamy et al., 1994). Oxygen evolving complex involves four Mn per PS II which serve as direct oxidant of water (Hansson and Wydrzynski, 1990). The three extrinsic proteins of 33, 23 and 17 kDa associated with the lumenal surface of the PS II reaction centre complex play important roles in oxygen evolution. Heat inactivation of oxygen evolution is accompanied by separation of Mn and the three extrinsic proteins from the complex (Nash et al., 1985).

Dissociation of two of the four Mn atoms from the PS II complex by heat results in full inactivation of oxygen evolution without a significant loss of proteins (Nash et al., 1985). However, the 33 kDa protein is essential for oxygen evolution and its removal with high concentration of $CaCl_2$ or urea always results in a strong inactivation of the activity. In particular, this protein has a stabilising effect on the binding of Mn (Kuwabara et al., 1985). Enami et al. (1994) have shown that heat-inactivation of oxygen evolution is directly related to release of the 33 kDa protein but not that of Mn. Thus, the stabilisation against heat of the oxygen-evolving machinery in the PS II complex seems likely to play an essential role in protecting the entire photosynthetic system from heat inactivation (Nishiyama et al., 1993, 1997).

Since the photosynthetic PS II complex is integrated with thylakoid membranes, several authors hypothesized that physical properties of the membranes may contribute to the thermal stability of photosynthesis (Quinn and Williams, 1985; Webb and Green, 1991). It is believed that thermal denaturation of PS II is directly related to the major changes in the lipid phase of thylakoid membranes that occur at high temperature (Berry and Björkman, 1980; Yordanov et al., 1986). Increasing temperature causes an increase in the fluidity of membrane lipids (Raison et al., 1982) followed by the formation of non-bilayer lipid structures (Gounaris et al., 1984). Presumably, one of the main consequences of those lipid changes is a destabilisation of lipid–protein interactions, perturbing the organisation and function of PS II. It has been suggested that the strength of the hydrophilic interactions which presumably link the light-harvesting antennae with the PS II complexes decreases with increasing temperature, while that of the hydrophobic interactions increases, so that the pigment-protein complexes tend to associate more with the lipids than with each other, resulting in their dissociation (Berry and Björkman, 1980).

The surface charge of thylakoid membrane is the major property determining its stability (Sackman, 1983). If the charge density on the membrane surface increases, the electrostatic repulsion between equally charged molecules grows as well. As a consequence, lipids become more disordered which could lead to a protein lateral diffusion in the membrane plane, accompanied by a decrease in its stability, which makes it more sensitive to heat damage (Barber, 1981; Goltsev et al., 1987).

It has been suggested that leakage (membrane damage) is an early event in a plant's response to heat stress. Electrolyte leakage is an effective means of measuring cell membrane thermostability and has been used as an indicator of direct heat injury in various plants (Ahrens and Ingram, 1988; Inaba and Grandall, 1988).

As it was already mentioned heat effect on the stability of the thylakoid membrane components is the primary cause for irreversible inhibition of photosynthesis (Süss and Yordanov, 1986; Weis and Berry, 1987). For example, photophosphorylation is one process that is inhibited by high temperature due to thermal uncoupling (Weis and Berry, 1987). This will result in a decrease in the supply of ATP necessary for carbon assimilation. In addition, damage to PSII at the level of O_2 evolving complex or the destabilisation of the light-harvesting chlorophyll *a*/*b*-binding complex associated with PS II will decrease the supply of NADPH consumed directly in CO_2 fixation as well as decrease the efficiency of the thioredoxin system responsible for the light activation of key regulatory enzymes of the Calvin cycle (Cseke and Buchanan, 1986). It has been reported that light-dependent activation of Rubisco was inhibited by moderately elevated temperatures and that inhibition was closely correlated with reversible inhibition of CO_2 fixation (Kobza and Edwards, 1987). The diffusion of CO_2 and O_2 and the affinity for carboxylation of the Rubisco enzyme have been proven to be affected by increasing temperatures (Jordan and Ögren, 1984; Brooks and Forquhar, 1985). It has been found that inhibition of carbon assimilation at high tempe-

ratures occurred under both photorespiratory and non-photorespiratory conditions. Thus, the temperature induced inhibition of carbon assimilation cannot be explained solely by increased photorespiration via enhanced specificity of Rubisco for oxygen at high temperatures (Jordan and Ögren, 1984).

Activation of Rubisco in the light is regulated by a stromal enzyme named Rubisco activase (Portis, 1992; Andrews et al., 1995; Salvucci and Ogren, 1996). Several studies have shown that isolated activase is particularly sensitive to inactivation by elevated temperature (Crafts-Brandner et al., 1997; Eckhardt and Portis, 1997). Thus, inactivation of activase provides a probable biochemical explanation for the inactivation of Rubisco at high temperatures (Feller et al., 1998).

At the molecular level, the negative effect of high temperature stress on leaves may be partly a consequence of the oxidative damage to important molecules as a result of the imbalance between production of activated O_2 and antioxidant defences (Foyer et al., 1994). This hypothesis is very plausible because chloroplasts are a major source of activated O_2 in plants (Asada and Takahashi, 1987; Asada et al., 1998), and because antioxidants, which may play a critical role in preventing oxidative damage, are greatly affected by environmental stresses (Bowler et al., 1994). In chloroplasts the superoxide radical ($O_2^{\bullet -}$) is produced by photoreduction of O_2 at PSI and PSII, and singlet O_2 is formed by energy transfer to O_2 from triplet excited state chlorophyll (Asada and Takahashi, 1987). H_2O_2 can originate, in turn, from the spontaneous or enzyme catalysed dismutation of $O_2^{\bullet -}$. Fortunately, in optimal conditions leaves are rich in antioxidant enzymes and metabolites and can cope with activated O_2, thus minimising oxidative damage. An increase of the active O_2 forms in plant tissue has been found at high temperature stress (Foyer et al., 1997; Dat et al., 1998). High temperatures can also influence the antioxidant enzymes (Paolacci et al., 1997). Superoxide dismutase (EC 1.15.1.1), the first enzyme in the detoxifying process, converts $O_2^{\bullet -}$ radicals to H_2O_2. In chloroplasts, H_2O_2 is reduced by ascorbate peroxidase (EC 1.11.1.11) using ascorbate as an electron donor. Oxidised ascorbate is then reduced by reactions that are catalysed by monodehydroascorbate reductase (EC 1.8.5.1) and glutatione reductase (EC 1.6.4.2) in a series of reactions known as the Halliwell–Asada pathway (Bowler et al., 1992).

For a given plant material, the temperature at which the above-described denaturation events occur appears to vary. It is also well-known that increasing the temperature at which plants are grown causes an upward shift of the optimal temperature of photosynthesis in numerous species and renders the photosynthetic apparatus more tolerant to heat stress (Berry and Björkman, 1980; Yordanov et al., 1986). This phenomenon is termed acclimation. Acclimation to a new growth temperature is not instant but requires a certain time period. Under stress organisms undergo first of all destabilisation followed by normalisation and stability enhancement when limits of tolerance are not exceeded and adaptive capacity is not overtaxed (Larcher, 1987). The ability of plants to withstand high temperatures is due to a variety of mechanisms acting at

different level of plant organisation (Levitt, 1980) including: increased evapotranspiration to maintain lower leaf temperatures (Upchurch and Mahan, 1988); modification of the photosynthetic system to avoid photoinhibition and free radical damage (Burke, 1990; Steffen, 1991) and synthesis of heat shock proteins (HSPs) in plant cells (Vierling, 1991). In most cases the adaptive mechanisms to different thermal regimes could be considered as compensatory, because they enable plants to buffer the effect of the temperature shift on their metabolic system (Berry and Raison, 1981).

Havaux (1995) has shown that chloroplasts possess an adaptive mechanism that senses a moderate elevation of environmental temperature and triggers the rapid conversion of PS II from its "normal" heat-sensitive state to a heat-resistant state. The molecular basis of this short-term adaptation to heat is unknown. The phenomenon does not seem to involve *de novo* protein synthesis since it is not perturbed by chloramphenicol or cycloheximide (Havaux, 1994). Significant changes in the lipid composition of thylakoid membrane are also improbable because they are known to occur much more slowly than the above mentioned increase in PS II thermostability induced by moderately elevated temperatures. Consequently, the rapid adjustment of PS II thermostability has been interpreted in terms of temperature-induced conformational changes of PS II (Havaux, 1994).

Some oxygenated carotenoids (xanthophylls) are known to be important factors controlling and stabilising the conformation of LHCII which contains about 14 chlorophyll molecules per polypeptide of the apoprotein and three to four xanthophylls (Thornber et al., 1993). It has been proposed that deepoxidation of violaxanthin to zeaxanthin favours ΔpH-induced conformational changes in the light harvesting system of PS II, converting PS II to a state of high thermal energy dissipation (Horton et al., 1994). A xanthophyll-modulated increase in heat losses is believed to protect the PS II reaction centres from overexcitation and subsequent photoinhibition.

High-temperature acclimation involves a considerable reorganisation of thylakoid membrane, including adaptive changes of lipid composition. They contribute to the optimum physical state of the membrane (microviscosity, permeability, etc.). During heat acclimation the threshold temperature at which fluidity still maintains the native membrane structure and function, rises (Raison et al., 1982). The more saturated lipid species decrease noticeably the thylakoid membrane mobility at elevated temperatures thus keeping a well arranged lateral movement of electron carries between the photosystems. The achieved thermotolerance of the majority of thermosensible light reactions is probably the result of both the lipid-induced more thermostable conformations of the membrane connected thylakoid protein subunits and the adjustment of membrane lipid fluidity. *In vivo* interaction of xanthophyll-cycle pigments with the membrane lipid matrix is supported by a series of experimental facts (reviewed by Sarry et al., 1994). It has been reported that zeaxanthin synthesis modulate the fluidity (Gruszecki and Strzalka, 1991) and the lipid peroxidation status (Sarry et al., 1994) of thylakoid membranes.

The protein synthesising system plays a crucial role in plant acclimation processes. If the growth temperature of a plant is increased beyond a particular point, a sudden decline in the ability to synthesise protein occurs. On the other hand, elevated temperatures initiate changes in transcription and selective translation of heat shock mRNA encoding heat shock proteins (HSPs), thereby enhancing thermotolerance of treated plants (Nover, 1991; Vierling, 1991; Howarth and Ougham, 1993; Waters et al., 1996). HSPs are generally divided into two classes: high-molecular-mass HSPs (60–110 kDa) and low-molecular-mass HSPs (15–30 kDa) (Vierling, 1991; O'Connell, 1994). The low-molecular-mass HSPs, which are encoded by a large gene family, are the most abundant HSP class found in plants (Vierling, 1991; Waters et al., 1996). Depending on the plant species, there can be more than 30 different plant small HSPs belonging to at least five different classes. Three of them specify proteins localised to the chloroplast, endoplasmic reticulum, or mitochondrion. The other small HSP families, designated class I and class II, are cytosolic proteins. In mature vegetative tissue, plant small HSPs are normally undetectable under optimal growing conditions, but they accumulate rapidly in response to heat stresses 10 to 20°C above normal (Süss and Yordanov, 1986; Vierling, 1991). Indeed, small HSPs can accumulate to more than 1% of the total protein in heat stressed pea leaves and they persist long after the cessation of heat stress, with half-lives of more than 24 h (De Rocher et al., 1991; Waters et al., 1996).

The accumulation of HSPs has been temporally correlated with the development of thermotolerance in many organisms (Nover, 1991), however little is known about the HSP function in plants. Several of the low-molecular-weight HSPs have been shown to be transported into the chloroplast of pea, soybean and maize (Vierling et al., 1986) and some have been demonstrated to have become associated with the photosynthetic membranes, potentially protecting the photosynthetic apparatus from damage at high temperatures (Schuster et al., 1988; Süss and Sainis, 1996). Low-molecular-weight HSPs have also been found to form cytoplasmic aggregates during heat stress (Nover et al., 1983). These "heat-shock-granules" have been found to contain mRNAs coding for normal cellular proteins and possibly have a role in protecting these mRNAs which are not translated during high temperature stress (Nover et al., 1989). Considerable recent data indicate that several HSPs function as "molecular chaperones". Molecular chaperones are proteins that bind to partially folded or denaturated substrate proteins and thereby prevent irreversible aggregation or promote correct folding of the substrate (Landry and Gierasch, 1994; Hartl et al., 1996). Binding of substrate to chaperones can also maintain the substrate in an unfolded conformation in order to facilitate protein translocation across membranes and other processes. It is proposed that during high temperature stress HSPs prevent accumulation of heat denaturated protein aggregates or facilitate protein reactivation following stress (Parsell et al., 1994; Morimoto et al., 1994).

In conclusion, the mechanisms for plant acclimation to high temperatures have been little understood although much data are available in literature (Pearcy, 1978; Armond et al., 1978; Raison et al., 1982). It is not clear yet whether the expression of HSPs is an adaptive response to the stress applied and in addition, to what extent the conformational changes in lipids, proteins or pigment-protein complexes determine plant acclimation to heat shock.

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