Photosynthesis: Limitations in Response to High Temperature Stress

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Photosynthesis: Response to high temperature stress

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ABSTRACT

Global warming has led to increased temperature of the earth which is a major abiotic stress posing a serious threat to the plants. Photosynthesis is amongst the plant cell functions that is highly sensitive to high temperature stress and is often inhibited before other cell functions are impaired. The primary sites of targets of high temperature stress are Photosystem II (PSII), ribulose-1,5-bisphosphate carboxylase/oxygenase (Rubisco) while Cytochrome b559 (Cytb559) and plastoquinone (PQ) are also affected. As compared to PSII, PSI is stable at higher temperatures. ROS production, generation of heat shock proteins, production of secondary metabolites are some of the consequences of high temperature stress. In this review we have summarized the physiological, biochemical and molecular aspects of high temperature stress on the process of photosynthesis, as well as the tolerance and adaptive mechanisms involved.

1. Introduction

Temperature is continually increasing due to global warming and has become a serious threat to crop yield. Temperatures above the normal optimum are termed as heat stress (HS) which disturbs the normal cellular homeostasis leading to retardation in growth and development and even death in extreme conditions. As sessile organisms, plants are constantly exposed to changes in temperature and other abiotic factors. High temperature stress is one of the major abiotic stresses that limit plant productivity in a number of ways [1]. Although the plant growth is controlled by a multitude of physiological, biochemical, and molecular processes, photosynthesis is a key phenomenon, which contributes substantially to the crop yield. Photosynthetic pigments, the two photosystems, electron transport system, and CO_2 reduction pathways are a few important components of photosynthesis and damage to any of them is enough to inhibit overall photosynthetic mechanism of a plant [2].

Photosynthesis serves as a global sensor of environmental stress that induces cellular energy imbalance as reflected in the distinct alteration in redox state associated with thylakoid membranes [3]. Photosynthesis is highly sensitive to high temperature stress and is often inhibited before other cell functions are impaired [4]. Maximum plants show extensive competence to fine-tune their photosynthetic characteristics to their growth temperatures. The most distinctive observable fact is a shift in the optimum temperature of photosynthesis as the growth temperature changes or with seasonal temperature shifts, which allows the plant to enhance photosynthetic efficiency at their new growth temperature [4,5].

It has been a matter of discussion that a new "green revolution" is essential in world agriculture to increase crop yields for increasing food demands [5,6], and ornamentation of photosynthesis will be a promising approach for increasing crop yield. However, to accomplish this target, it is essential to be aware of the processes that limit photosynthesis under a series of growth conditions, and how fine photosynthesis can acclimate to predicted changes in temperature. High temperature stress is invariably associated with high light intensities. It is difficult to differentiate effects of each of them under natural conditions. However mechanistic aspects of photoinhibition have been reviewed extensively recently [7–9]. Hence, in this review we have focussed more on the major biochemical, physiological and molecular changes occurring in the photosynthetic apparatus in response to high temperature stress.

1.1. High temperature stress and photoinhibition

Other major abiotic stress which is often accompanied with high temperature is light intensity. Light varies in intensity both temporally (as a result of the diurnal cycle) and spatially (as a result of shading by clouds and other organisms and objects) throughout the day. Excess light leads to photoinhibition, a sustained decline in photosynthetic efficiency, associated with damage to P680 [10]. PSII is generally known as a photosystem with exceptionally high susceptibility to light damage [11,8]. The major fundamental problem of exposure to elevated light

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intensities arises from differences in the rates of energy absorption and transfer to the reaction centers of photosystems and subsequent electron transport. Being much slower than energy transfer, electron transport rates fulfils the fundamental thermodynamic requirement to minimize the uphill reactions and therefore stabilize energy, which is to be used in the chain of electron/proton transfer processes leading to NADPH and ATP synthesis. In fact, photoinhibition occurs when the demand from the carbon reduction cycle for ATP and mainly reductive power is decreased and thereby not enough $NADP^+$ is available to act as the terminal electron acceptor of the linear photosynthetic electron transport chain. Under increasing light intensity, the photosynthetic reaction centers become progressively saturated (closed), resulting in a reduction in the fraction of energy utilized in photosynthesis and the subsequent build-up of "unused", potentially harmful, excitation energy in the photosynthetic membrane. When electron donation to P680 is less efficient than oxidation, an increase in the $P680^+$ lifetime will take place. $P680^+$ will oxidize the nearest pigments and amino acids eventually leading to degradation of the PSII reaction center protein D1. Conversely, when electron donation from P680 to oxidized plastoquinone is inhibited by the build-up of reduced plastoquinone charge recombination can occur triggering P680 triplet formation [12,13]. In the triplet state P680 will interact with atmospheric triplet oxygen, causing formation of singlet oxygen, which in turn will bleach P680. Photoinhibition inevitably leads to a reduction in the number of active PSII units and because of the slow repair of damaged D1 proteins the decline in electron transfer normally persists for several hours, even in the dark or at low light intensity [14,7]. Since photoinhibition has a potential to lower productivity and plant growth, avoidance of photoinhibition is critical for the fitness and survival of plants in natural habitats [15]. However, it is not only the availability of light but also the metabolic state of the plants that sets the requirements for the photosynthetic machinery. Likewise, the photosynthetic machinery must have mechanisms to downregulate the efficiency of light harvesting when excess energy is available [16].

2. High temperature stress-induced changes occurring in photosynthetic apparatus of the plant

Some of the important photosynthetic processes affected by high temperature stress are summarized in Fig. 1 and described later in the text.

2.1. Effects on photosynthetic pigments

A number of reports indicated that plants exposed to high-temperature stress show reduced chlorophyll (Chl) biosynthesis [17]. High temperature stress leads to impairment of Chl biosynthesis in plastids [18]. Lesser accumulation of Chl in high temperature stressed plants may be attributed to impaired Chl synthesis or its accelerated degradation or a combination of both. The inhibition of Chl biosynthesis under high-temperature regimes results from a destruction of numerous enzymes involved in the mechanism of Chl biosynthesis [17]. The activity of 5-aminolevulinate dehydratase (ALAD), the first enzyme of pyrrole biosynthetic pathway, was found to decrease under high-temperature regimes [19,20]. Yordanov et al. [21] have reported that initial/pre high temperature treatment of etiolated barley seedlings treated for 4 and 8 h inhibited Chl biosynthesis, which could be an outcome of decreased protochlorophyllide number or its transformation. Protochlorophyllide (Pchlide) synthesis, porphobilinogen deaminase, and Pchlide oxidoreductase are analogously affected in hexaploid wheat (cv. HD2329) seedlings. This reveals that high temperature stress have alike effects on enzymes involved in Chl biosynthesis. In response to high temperature stress (38/28 °C), a decrease in total Chl content (18%), Chl *a* content (7%), Chl *a*/*b* ratio (3%), sucrose content (9%) and an increase in reducing sugar content (47%) and leaf soluble sugars content (36%) [22] was observed in soybean.

3. High temperature stress and Photosystem II

Photosystem II (PSII) is considered to be one of the most thermosensitive components of photosynthetic apparatus [4,23]. The two key factors that makes PSII electron transport most susceptible to heat stress are: (i) increase in fluidity of thylakoid membranes at high temperature which causes dislodging of PSII light harvesting complexes from thylakoid membrane and (ii) dependence of PSII integrity on electron dynamics. The sensitivity of PSII to stress depends on the organization level of the system in question (e.g. cyanobacteria, monocot, dicot, etc.) and there is also variation in the extent of the acclimation of PSII to heat stress [24,25].

It is reported that the water oxidizing complex (WOC), PSII reaction center and the light harvesting complexes are initially disrupted by high temperature [26]. Many workers have reported that heat inactivation of PSII is mainly due to the dissociation of

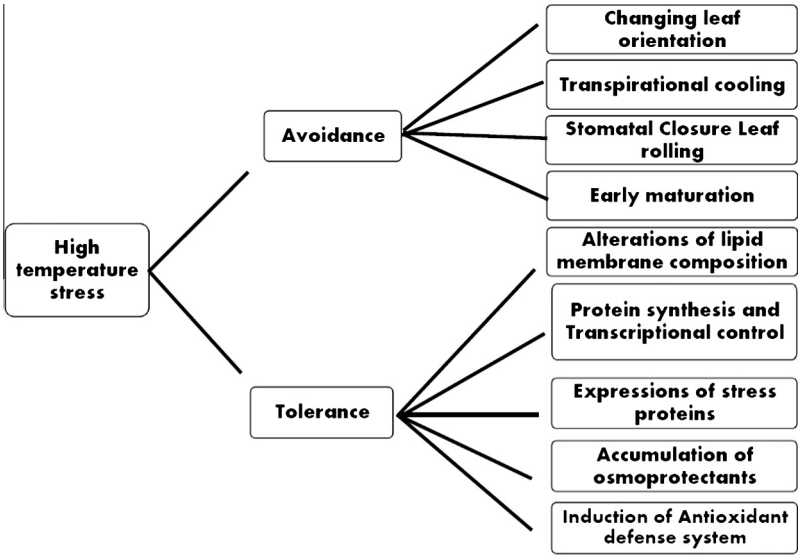

Fig. 1. High temperature induced biochemical and molecular responses in the photosynthetic machinery.

divalent Ca^{2+} and Mn^{2+} cations and Cl^- anion from the PSII pigment-protein complex and the release of extrinsic 18, 24 and 33 kDa polypeptides from the thylakoid membranes [4,27,28]. As many as five extrinsic proteins associate in different orientations depending on the type of organisms [29]. The extrinsic proteins are labelled as O (PsbO, 33 kDa), P (PsbP, 23 kDa), Q (PsbQ, 17 kDa), R (PsbR), U (PsbU), and V (PsbV). Only the PsbO protein (the 33-kDa or manganese stabilizing protein) is ubiquitous to all oxygenic photosynthetic organisms and is known to stabilize the Mn cluster. 23-kDa protein enhances the binding of calcium and chloride in addition to Mn cluster [29–31]. For advancement of cellular thermotolerance in *Synechocystis* sp. *PCC 6803* protection of the oxygen-evolving machinery by extrinsic proteins of PSII was found to be essential [32]. Dissociation of 33, 23, and 17 kDa proteins and loss of cofactors were results of high temperature stress to chloroplasts. Inhibition or inactivation of PSII, thermal damage of D1 protein, and production of reactive oxygen species (ROS) both in light and in dark are few consequences of loss of cofactors (especially PsbU) due to high temperature stress. It was suggested that PsbU provides protection from ROS, as PsbU mutants have enhanced mechanisms to detoxify exogenously applied H_2O_2 [33].

Amongst the intrinsic proteins of PSII, moderate heat exposure (40 °C for 30 min) of spinach thylakoids could cleave only the D1 protein producing a 9-kDa C-terminal and 23-kDa N-terminal fragments while D2, CP43, or CP47 remained uncleaved [34]. Due to high temperature, FtsH protease which is usually located in stroma segment of thylakoid migrated to granal segment and thus affected the degradation of phosphorylated D1protein [35,36]. It is thus assumed that degradation of thermally damaged (heat inhibited) and high light damaged (photoinhibited) D1 follows the same route [34,37]. Heat stress effects are also associated to thylakoid membrane integrity, ion conductivity and phosphorylation activity. Gounaris et al. [38] demonstrated that heat treatment may induce grana destacking and the formation of cylindrical inverted micelles. Heat-inactivation of PSII may be followed by dissociation of light harvesting complex II [LHC II] [39]. A further heat-induced effect that has been described is a shift of the redox equilibrium between Q_A and Q_B [40,41]. The inhibition of Q_A–Q_B electron transfer might result from a structural change in D1 and D2 proteins upon a high temperature-induced reorganization of the thylakoid membranes [42]. High temperature stress leads to downregulation of the quantum efficiency of PSII through a decrease in the rate of primary charge separation, a reduction in the stabilization of charge separation and the disconnection of some minor antenna from PSII [25].

Mn ion plays a critical role in photolysis of water. High temperature also causes dissociation of Mn cluster of PSII. Tiwari et al. [43] have reported that not much Mn^{2+} release was observed up to 45 °C, yet it indicates some perturbation in the polypeptides of the oxygen evolving complex (OEC) that could possibly result in an increase in the accessibility of Y_D to the lumen and thereby causing a complete reduction of the remaining Y_D^{ox}. At high temperature (47 °C) release of 18 kDa protein is the main cause behind the loss of essential Ca ion from the Mn_4Ca complex [44,45]. A slight change in the conformation of the bound 18 kDa may also facilitate the access of Y_D from the bulk and may favor a rapid reduction of it in dark at comparatively lower temperatures (35–45 °C) [43].

Chl *a* fluorescence measurements have intricate connection with the numerous processes taking place in PSII during the energy conversion of light into a stable chemical form [46,47]. Various fluorescence parameters have proven to be an excellent high temperature stress indicators. In response to high temperature stress (45 °C), an additional K band was observed at 300 μs [48] thus altering an OJIP curve to an OKJIP curve. The appearance of a K band is correlated with the inhibition of OEC [49,50,23], inhibition of electron transport from pheophytin to Q_A [49], changes in the structure of the LHC of PSII and partial uncoupling of the OEC [23]. The K band arises when electron flow to the acceptor side exceeds electron flow from the donor side, leading to oxidation of the RC [51,52]. It is reported that the antenna size, maximal fluorescence (Fm), Fv/Fm decreased while Fo and dissipation in the form of heat increased. For most plants at leaf temperatures above 35 °C, the initial decrease in photochemical efficiency is accompanied by a pronounced stimulation of nonphotochemical Chl *a* fluorescence quenching due to increased energy dissipation as heat [53,54]. An increase in energy dissipation at high temperature reduces the energy available for photochemistry under stress conditions [48]. Heat treatment of the plants leads to an inhibition of electron transfer at various sites which include the plastoquinol (PQH_2) oxidation site at Cytochrome (Cyt) b6/*f* complex.

3.1. Effect of high temperature on plastoquinone

Redox state of plastoquinone (PQ) pool is a major component in the regulation of photosynthetic reactions. There are many reports that suggests that PQ actively participates in a number of important photosynthetic processes viz. regulation of state-transition, chlorophyll biosynthesis, light-harvesting complex polypeptide accumulation, rate of photosystem protein synthesis and the balance of photosystem stoichiometry [55–58]. PQ-pool also acts as a sensor of any imbalance in electron transport. In the case of increased plastoquinone reduction, the limitation of Calvin cycle enzymes and the slowdown in ATP consumption, an increase in thylakoid ΔpH level and suppression of intersystem electron flow have been observed. An increased level of plastoquinone pool reduction causes the double reduction of Q_A (Q_A^{2-}) in the reaction center of PSII, a recombination of the ion-radical pair [$P680^+$ $Pheo^-$] and triplet $^3P680$ formation [11]. Oxidized plastoquinone molecules are known non-photochemical quenchers of chlorophyll excited states of both PSII and PSI antennas due to thermal dissipation [59].

A reduction in the PQ pool is observed as a consequence of high temperature stress. In green plants, the electron is recycled from the stroma-exposed side of PSI to the PQ-pool by the NAD(P)H dehydrogenase complex or the ferredoxin-plastoquinone oxidoreductase (FNR) [60]. Reduced PQ induces the increase in the photosynthetic capacity of PSI which is required to activate cyclic electron transport [59]. Moreover, reduced PQ molecules are known to be efficient scavengers of the singlet oxygen [61] that is generated in PSII [62–64]. Plastoquinol, as a phenolic compound, is oxidized during the scavenging of singlet oxygen formed in PSII [61]. Oxidation of plastoquinol by the superoxide anion radical generated in PSI is also known to be effective scavenging mechanism [65]. Thus, the decrease in level of reduced plastoquinone molecules could be caused by the scavenging function of plastoquinol. Thus, the stress could induce a decomposition of plastoquinols following by the scavenging of singlet oxygen in thylakoid membranes which could be compensated by fast biosynthesis of PQ molecules in the envelope [66].

3.2. Role of Cytb559 and Y_D under high temperature stress

Photosystem II is a multisubunit protein complex and besides several other components, contains a small heme–iron containing protein known as Cytochrome b559 (Cytb559).The C-terminal of Cytb559 is shielded by a 33 kDa Mn stabilizing protein. On the basis of midpoint potential Cytb559 is reported in two forms: high potential (HP) form. The dominant form (~80%) having midpoint potential of 330–400 mV, while the other is the low potential (LP) form (~20%) with a midpoint potential of 20–80 mV [67,68,43]. Vass et al. [69] have suggested the interconversion of HP form into LP form under stress conditions like low temperature, mild temperature stress, etc. Canaani and Havaux [70] have proposed that mild temperature stress causes a rapid conversion

of HP to LP form of Cytb559 and also causes an increase in oxidation–reduction changes. In addition Horton et al. [71] have shown a complete absence of HP form of Cytb559 under high temperature treated and trypsin digested chloroplasts. Schreiber and Neubauer [72] have reported that heat stress may cause cyclic electron flow around PSII involving Cytb559. Cyclic electron flow around PSII plays a crucial part under high temperature stress conditions. In such conditions thermally damaged OEC is replaced by a donation of electron from (cyclic electron flow around PSII) PSII acceptor side to $P680^+$ or Yz_{ox} thus restoring the photochemical activity of PSII [73]. Two redox active tyrosines, Y_D and Y_Z, with different functional roles are present in PSII. Studies based on site-directed mutagenesis have revealed that Y_Z is tyrosine 161 of the D1 polypeptide while Y_D is tyrosine 160 of the D2 polypeptide. Redox active tyrosine, Y_D, is also oxidized via P^+_{680} and is in slow redox equilibrium with the OEC. Y_D may be involved in assembly of the OEC, but is not required for oxygen evolution [74]. The probability of electron donation from Y_D to P^+_{680} increases under high temperature stress. Tiwari et al. [43] have suggested that the redox state of Y_D can be considered as PSII damage indicator due to high temperature stress.

3.3. Other processes in PSII influenced by high temperature

Besides direct effect of high temperature on redox reactions of PSII, there are several mechanisms which are modulated at high temperature. They include state transitions, membrane fluidity, ROS production, etc. "State transitions" is a process that regulates and balances the distribution of absorbed light energy between the two photosystems. Light absorbed by PSII gets funnelled to PSI in State 2 but not in State 1. The influence of high temperature on photosynthetic components alters the energy distribution in PSII. The phosphorylated LHCII get associated to PSI and get dissociated from PSII due to high temperature stress and thus enhances the cross section area of PSI causing a shift from state 1 to state 2 [75].

It is well established that high temperature causes an increase in the fluidity of membrane lipids followed by the formation of non-bilayer lipid structures [76] and also quantity of phospholipids decreased strongly [21]. Presumably, one of the main consequences of those lipid changes is a destabilisation of lipid–protein interactions, perturbing the organisation and function of PSII. It has been suggested that the strength of the hydrophilic interactions which presumably link the light-harvesting antennae with the PSII 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 [4]. Permeability of the thylakoid membranes which is regarded as very heat sensitive [77], could be counteracted by de-epoxidized zeaxanthin. Heat stress led to a decline in protein activity and the extent of damage to proteins increased with increased temperature [78].

Due to high temperature stress and photoinhibition production of reactive oxygen species (ROS) occurs which inhibits protein synthesis [79]. The generation of ROS such as $O_2^{\cdot-}$ and H_2O_2 can increase resulting in intracellular accumulation and oxidative stress. It has been reported that O_2 and $HO^{\cdot}$ are formed during dark incubation of spinach thylakoids at higher temperatures [80]. In these circumstances, the photosynthetic electron transport chain becomes over-reduced and ROS are formed, causing oxidative damage to the components of the photochemical apparatus. It is well established that the main target of oxidative damage is the D1 protein and that photoinhibition occurs when the accumulation of photooxidized D1 surpasses its *de novo* synthesis. These ROS can oxidize molecules in chloroplasts including D1 protein in the PSII reaction center and thiol enzymes in the Calvin–Benson cycle to inhibit partial reactions of photosynthesis eventually leading to photoinhibition [79]. Enhanced ROS levels leads to oxidation of saturated fatty acids in the thylakoid membranes. Increase in unsaturation of fatty acids further cause damage of membrane fluidity thereby affecting the stability of PSII proteins [78]. Additionally, at higher temperatures, plants can produce nonenzyme antioxidant species such as vitamin E [81] and ascorbic acid [82,83] that rapidly scavenge ROS.

3.4. Changes in heterogeneity of PSII under high temperature stress

PSII has been found to be more heterogeneous than other components such as PSI and Cyt b_6f in various aspects and differs in its structure and function both [84]. This diverse nature of PSII is known as heterogeneity. Two main aspects of PSII heterogeneity have been studied widely i.e., PSII antenna heterogeneity and PSII reducing side heterogeneity. On the basis of the differences in the antenna size PSII alpha (α), PSII beta (β) and PSII gamma (γ) centers have been defined while on the basis of acceptor/reducing side function, Q_B-reducing and Q_B-non-reducing centers have been proposed. A third type of PSII heterogeneity is known as structural heterogeneity which is related to the stacking and unstacking of grana and is based on distribution of PSII in appressed and non-appressed regions of thylakoid membranes.

Mathur et al. [85] have reported that subsequent increase in temperature from 25 °C to 45 °C in wheat leaves led to a decrease in the proportion of α centers while β and γ centers showed an increase. The β and γ centers seemed to increase at the cost of α centers and thus these components probably are interconvertable depending on the environmental conditions. Antenna heterogeneity is also studied in terms of connectivity. It was reported that as compared to 25 °C (control) a gradual loss of connectivity was observed in 40 °C but 45 °C showed no grouping at all in wheat plants. As the temperature was increased the sigmoidal component of the curve decreased suggesting a decrease in the connectivity between antenna molecules and an increase in the number of inactive centers. The treatment at 45 °C shows an additional positive '**L**' step (~150 μs). The presence of this L step at 45 °C indicates that the PSII units in high temperature treated samples are less grouped or less energy was being exchanged between independent PSII units [86]. Exposure of leaves to temperature up to 35 °C does not affect much the relative fractions of Q_B-non-reducing PSII centers but as the temperature was raised to 40 °C a gradual change in the fractions of Q_B-non-reducing PSII centers was observed while at temperature greater than 40 °C i.e. at 45 °C the fraction of Q_B-non-reducing increased drastically up to 50%. At high temperatures (45 °C), the fractions of Q_B-non-reducing centers increased which imply that these centers were unable to reduce PQ pool and also that the active Q_B-reducing centers were converted into inactive Q_B-non-reducing centers. High temperature caused structural alterations also (stacking/destacking) in the thylakoid membranes. With increasing temperature (from 35 °C to 45 °C), the membranes became more unstacked in wheat and spinach thylakoid membranes. Heating to 35–45 °C is known to cause destacking of thylakoid membranes and this destacking is expected to abolish differences between PSII centers located in grana and stroma thylakoids [87,85].

4. Effect of high temperature on Photosystem I

Many investigations have shown that PSI activity is much more heat stable than PSII [88,27]. Results of moderately high temperatures stimulate PSI activity *in vivo* and *in vitro* and caused increased thylakoid proton conductance and increased cyclic electron flow around PSI [89–93]. Under conditions when PSII activity is severely diminished, this stimulation of proton conductance of cyclic electron flow (CEF) around PSI could be an adaptive process, producing ATP. Heat stress also significantly increases the dark reduction of

PQs [91,94–97] and enhances the transthylakoid proton gradient which was interpreted through a stimulation of CEF around PSI [91,95]. There is more for ATP during mild temperature stress because of ribulose-1,5-bisphosphate carboxylase/oxygenase (Rubisco) activase and active photorespiration [98]. The higher ATP demand under mild heat stress conditions would result in a higher NADPH/ATP ratio favoring non-photochemical reduction of the PQ pool from stromal donors which in turn would activate the NADH-mediated cyclic electron pathway. This would help dissipating excess energy and provide additional ATP to maintain active CO_2 fixation. In the absence of NDH-mediated cyclic electron pathway, like in tobacco transformants deficient in the NDH complex, the incapacity to equilibrate the NADPH/ATP ratio at high temperature would favor ROS generation [99]. However, the increase in non-photochemical reduction of PQs observed in response to heat stress was not affected in tobacco transformants deficient in the NDH complex [96,100,101].

4.1. Initiation of cyclic electron flow around PSI

There are extensive effects of moderate heat on PSI and Cytochrome complex reactions [91,95,102]. The dark reduction of PSI was found to undergo 'spectacular acceleration' with the half life of $P700^+$ falling from over 500 ms to less than 50 ms between 34 and 40 °C [95]. Yamane et al. [100] reported a flow of electrons from the stroma to the plastoquinone pool in the dark at 36 °C. PSI was more reduced by heat stress [89–91,95] while PSII and the stroma become more oxidized at high temperature, which indicates that the redox balance of different components of photosynthetic electron transport can change in opposite directions and heat significantly alters the redox balance of the components of electron transport away from PSII and toward PSI. It is possible that heat disrupts the control normally found at the $Cytb_6f$ complex or the control is temperature-dependent, and is altered to be non-limiting under moderate heat stress [103].

5. Effect of high temperature on stomatal opening

Transpiration is a physical process in plants in which part of the net radiation energy is converted into latent heat, under physiological control by changes in stomatal aperture. Plant photosynthesis and stomatal conductance shares a strong relationship. A decrease in soil and root hydraulic conductance as a result of soil drying may be an important control mechanism of stomatal closure. Low leaf water potential which is caused by high transpirational rates results in midday reduction in stomatal conductance. The capability of plant to sustain leaf gas exchange and CO_2 assimilation rates under heat stress is directly concomitant with heat tolerance. High temperature noticeably affects the leaf water status, leaf stomatal conductance (gs) and intercellular CO_2 concentration [104]. Closure of stomata under high temperature is another reason for impaired photosynthesis that affects the intercellular CO_2 [22,105]. Stomatal conductance (gs) and net photosynthesis (Pn) are inhibited by moderate heat stress in many plant species due to decreases in the activation state of Rubisco [106–108]. Temperature changes not only directly affect vapour pressure density (VPD) but can also result in changes in plant hydraulic conductance and water supply to the leaf surface [109–113].

6. Effects of high temperature on Rubisco activity

The difference between the rate of photosynthetic CO_2 assimilation and respiration largely decides the crop productivity. Within the photosynthetic process, Rubisco is the enzyme in charge for CO_2 fixation, the importance of which on the primary productivity has been estimated to be above 1,011 tons of atmospheric CO_2 being annually fixed [114,115]. Mechanistically, it has been proposed that the activity of Rubisco activase is insufficient to keep pace with the faster rates of Rubisco inactivation at these high temperatures [5]. The peculiar response of net photosynthesis to temperature is caused by the complex kinetics of Rubisco and some very unusual properties of this enzyme. Rubisco catalyses the first step in two competing pathways, photosynthesis and photorespiration, whose rates are determined by the rates of the carboxylase and oxygenase activities respectively [116]. The V_{Max} of the carboxylase activity increases with temperature, but the affinity of Rubisco for CO_2 and the solubility of CO_2 decrease [116,117]. Photorespiratory activity increases with temperature because of decreases in both the relative specificity of Rubisco for CO_2 compared with O_2 and the relative solubility of CO_2 compared to O_2 [118]. When CO_2 fixation is inhibited by moderate heat stress it affects three measures of thylakoid energization namely non-photochemical chlorophyll fluorescence quenching, the electrochromic absorption shift and light scattering, representing that the energy provided for photosynthesis is not being utilized by the Calvin cycle [119,120]. The rate of net photosynthesis in heat stressed leaves increases with increasing CO_2 concentration even under non-photorespiratory conditions. This indicates that under moderate heat stress the capacity for electron transport and RuBP regeneration is in excess [121]. According to Portis [122] and Salvucci and Crafts Brandner [123] the probable reason of reduced Rubisco activation at high temperature is activase's low temperature optimum and thermal lability. Although not strictly coupled to Rubisco activation, ATPase activity is required for Rubisco activation and both activities change in parallel in response to temperature and other conditions [122,124]. Lower expression of Rubisco per area under heat stress has been reported in various species, concomitantly to decreases of its protein content [125–127].

However, nature has provided plants with remarkable tolerance and adaptive mechanisms to cope up with high temperature stress. Some of the important adaptive mechanisms are described below.

7. Adaptation of photosynthetic apparatus to high temperature stress

Plant adaptation to heat stress mainly includes avoidance and tolerance mechanisms (Fig. 2) which employ a number of strategies. Under high temperature conditions, plants exhibit short-term avoidance or acclimation mechanisms such as changing leaf orientation, transpirational cooling, or alteration of membrane lipid compositions. Closure of stomata and reduced water loss, increased stomatal and trichomatous densities, and larger xylem vessels are common heat induced features in plant [128]. Plants with small leaves are also more likely to avoid heat stress: they evacuate heat to ambient more quickly due to smaller resistance of the air boundary layer in comparison with large leaves. Plants rely on the same anatomical and physiological adaptive mechanisms those are deployed in a water deficit to limit transpiration. In well-hydrated plants, intensive transpiration prevents leaves from heat stress, and leaf temperature may be 6 °C or even 10–15 °C lower than ambient temperature. Such morphological and phenological adaptations are commonly associated with biochemical adaptations favoring net photosynthesis at HT (in particular C4 and CAM photosynthetic pathways).

Some major tolerance mechanisms, including ion transporters, late embryogenesis abundant (LEA) proteins, accumulation of osmoprotectants, induction of antioxidant defence, and factors involved in signaling cascades and transcriptional control and protein synthesis are essentially significant to counteract the stress effects In case of sudden heat stress, short term response, i.e., leaf orientation, transpirational cooling and changes in membrane lipid composition are more important for survival [129].

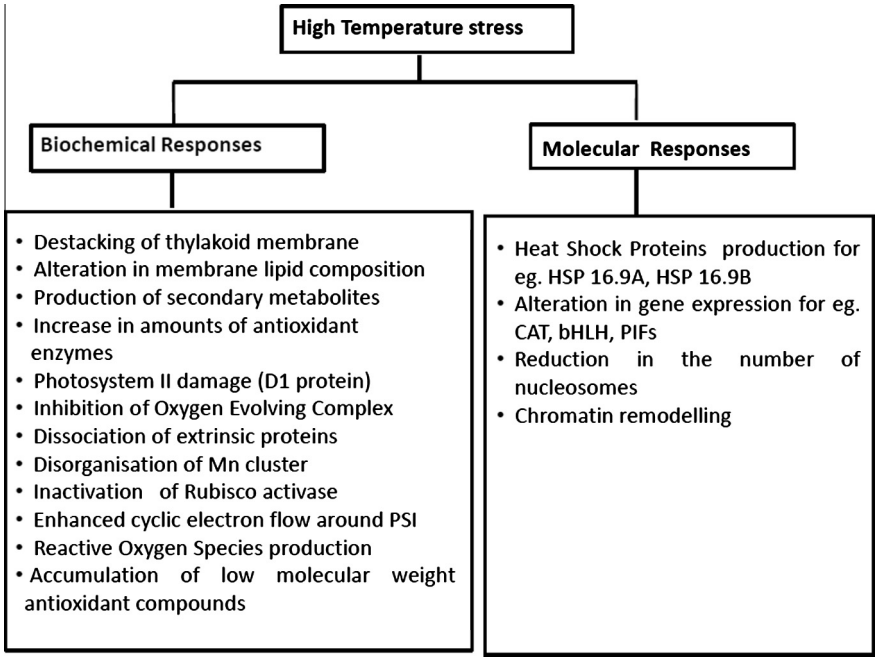

Fig. 2. A schematic diagram showing avoidance and tolerance mechanisms of plant in response to high temperature stress.

7.1. Effect of high temperature stress on primary and secondary metabolites in photosynthesis

Homeostasis in general, including biosynthesis and compartmentalization of metabolites, is disturbed in high temperature challenged plant tissues [130]. High temperature modifies the activities of enzymes involved in carbon metabolism, starch accumulation, and sucrose synthesis by down-regulating specific genes in carbohydrate metabolism [131]. Among the primary metabolites accumulating in response to heat stress are proline, glycine betaine, or soluble sugars [132]. Many plant species accumulate other osmolytes as well, such as sugar alcohols (polyols), or tertiary and quaternary ammonium compounds. Osmolyte production under heat stress is thought to increase protein stability and stabilize the structure of the membrane bilayer [133,134]. Secondary metabolites such as phenolics including flavonoids, anthocyanins, and plant steroids are notably involved in plant responses under high temperature stress and play a vital role in abiotic stress responses associated with tolerance [132]. For example, stress increased phenylalanine ammonia lyase activity and decreased peroxidase and polyphenol oxidase activity, presumably as part of the acclimation to heat [135]. Several key phytohormones including abscisic acid (ABA), salicylic acid (SA), and ethylene (ET) also increase their levels under heat stress, while others decrease, such as cytokinin (CK), auxin (AUX), and gibberellic acid (GAs), fluctuations that ultimately cause premature plant senescence [136–138].

7.2. The heat-shock proteins

Heat shock proteins are produced as a result of stress. Heat-shock proteins (HSPs) are important for protecting cells against high temperature and other stresses [139]. Addition of purified chloroplast localized HSPs conferred heat tolerance to the photosynthetic electron transport chain in isolated chloroplasts. *In vivo* experiments have demonstrated that small HSPs could associate with thylakoids and protect O_2 evolution and OEC proteins of PSII against heat stress. Evidence for the significance of chloroplast-localized HSPs for thermotolerance was obtained in tomato species [140,141]. Neta-Sharir et al. [142] demonstrated that chloroplasts small heat-shock protein, HSP21, induced by heat treatment in tomato leaves protected PSII from temperature-dependent oxidative stress. It is considered that chloroplast HSPs do not participate in the repair of stress-related damage, but rather they function to prevent damage [143,141]. Six nuclear gene families have been observed to encode all plant sHSPs, and each gene family characterises the proteins found in distinct cellular compartments, i.e. cytosol (class I and class II), chloroplast, endoplasmic reticulum, mitochondria and membranes [144]. More than 20 sHSPs have been found in higher plants and 40 different sHSPs may be found in the same species [145]. HSP16.9A, HSP16.9B and HSP16.9C are few HSPs identified in wheat plant [144]. Changes in enzyme levels, photosynthesis activity, cellular membrane structure and protein metabolism take place as a response of heat shock in plants [146]. HSPs can function in protection as molecular chaperons to prevent but no reverse protein denaturation and aggregation, as membrane stabilizers and possibly, as site-specific antioxidants [139]. HSP101 has been shown to be essential for thermotolerance by genetic analysis [147]. By genetic manipulation, the role and sub-cellular localization of the small (16 kDa) HSP-HspA was investigated comparing the cyanobacterium Synechococcus strain *ECT16-1*, with constitutively expressed HspA, with the reference strain [148]. The protein possesses the unique property to associate with thylakoid membranes during heat stress and support stability of thylakoid membranes. It is also supposed that other compounds different from HSPs can contribute to heat tolerance [149,150] for example PsbU in cyanobacteria. However, the levels of HSPs, namely, the homologs of HSP70, HSP60, and HSP17 remained unaffected by mutation in psbU gene suggesting that HSPs are not involved in cell acclimation to high temperature [1,149].

8. Omic approaches and high temperature stress

In photosynthesis, physiological and molecular (or now days the so called "OMICS") techniques go hand in hand for studying the response of high temperature stress new advances in "omic" technologies have provided new opportunities and hopes for the

identification of transcriptional, translational and post-translational mechanisms and signaling pathways that regulate the plant response(s) to abiotic stress including high temperature stress [22,151]. Such omic approaches helps in systematic analysis and correlation between the changes in the genome, transcriptome, microme, proteome and metabolome to the variability in plant's response to temperature extremes and their application to increase the chances of developing stress tolerant plants. Understanding the functions of these stress-inducible genes helps to unravel the possible mechanisms of stress tolerance.

At the molecular level, heat stress causes alterations in expression of genes involved in direct protection from high temperature stress [152,153]. These include genes responsible for the expression of osmoprotectants, detoxifying enzymes, transporters, and regulatory proteins [154,155]. In conditions such as high temperature, modification of physiological and biochemical processes by gene expression changes gradually leads to the development of heat tolerance in the form of acclimation, or in the ideal case, to adaptation [156]. Currently, transcriptomics and proteomics have been used to identify heat stress-responsive genes and proteins in plants [157–159], which can be divided into two groups. The first group includes signaling components, such as protein kinases and transcription factors. The other group includes functional genes, such as heat shock proteins (Hsps) and catalase (CAT) [160,161]. A class of bHLH (helix-loop-helix) transcription factors known as phytochrome interacting factors (PIFs) has also been connected in the heat related signaling mechanisms. PIFs have a wide range of regulatory roles in photomorphogenesis, skotomorphogenesis, and the down-stream regulation of hormone levels, in particular AUX and GA and implicitly on other phytohormones such as ABA. The precise role of these proteins in heat stress remains unclear; however, the transcriptional response of PIF4 in particular has clear knock on effects on early stages of plant development. Heat stress also leads to the transient activation of repetitive elements or silenced gene clusters close to the centromeric regions as well as the transient loss of epigenetic gene silencing [162,163]. Such gene silencing mechanisms are thought to be involved in transcriptional repression by hetero-chromatinization of repetitive DNA regions in plants [164]. Recent studies have indicated that regulation of stress responsive genes often depends on chromatin remodelling. High temperatures cause transcriptional repression of genes involved in cell growth, such as histones and DNA polymerases and deregulation of DNA methylation and transposon activation [163,165,166]. For example, AtCHR12, a SNF2/Brahma(BRM)-type chromatin remodelling factor in *Arabidopsis* was shown to play a role in mediating the temporary growth arrest in response to drought and heat stress [167]. In addition, whereas histone modifications show only minor variations upon heat stress, there is evidence for a dramatic reduction in the number of nucleosomes associated with DNA, leading to loss of chromo center organization, and this reduction in nucleosome density occurs throughout the genome. Efficient resilencing of some of these activated targets seems to require the chromatin assembly factor 1 (CAF-1) complex [138,168].

Generally, tolerance to heat is characterized by a lesser effect on essential processes such as photosynthesis and by consistent increases of transcripts involved in the biosynthesis of protective components. Multiple opportunities for plant improvement exist, as tolerance to high temperatures is a multigenic character. At the molecular level, abiotic stress tolerance can be achieved through gene transfer by altering the accumulation of osmoprotectants, production of chaperones, superoxide radical scavenging mechanisms, exclusion or compartmentation of ions by efficient transporter and symporter systems. Quantitative trait locus (QTL) analysis in tolerant and sensitive crops is now receiving much attention. The key benefit of QTL-based approaches is that they allow loci to be identified that are linked to heat tolerance. The identification of markers linked to QTLs enables breeding of stress-tolerant crops by combining or "pyramiding" QTLs for tolerance to various stresses. Tolerant genotypes may also be selected in controlled environments. Regardless the screening method, a key objective for plant breeders is to develop an effective set of thermo-tolerance markers which can be used to further implement heat tolerance into various crop species. The emerging phenomics methodologies are used to identify genes associated with traits of interest by establishing functional relationships between genetics and the associated phenotype. Such tools are also used to characterize plant performance under controlled environments or in the field [169]. Ideally, systematic phenotyping approach that includes arranges of heat stress conditions may increase the chances of identifying the functions of potential heat stress response genes.

9. Conclusion

Photosynthetic apparatus of plants respond to high temperature stress by inhibiting various redox and metabolic reactions taking place in PSII, PSI, $Cytb_6f$ complex and Rubisco. Plants adapt to high temperature, predominantly by producing compatible solutes that are able to organize proteins and cellular structures, maintain cell turgor by osmotic adjustment, and modify the antioxidant system to re-establish the cellular redox balance and homeostasis. In case of high temperature stress, adaptive mechanisms include change in the composition and degree of saturation of fatty acids, changes in the levels of protective antioxidants and treinoic fatty acids, changes in membrane permeability and increased cyclic electron flow. Interconversions in PSII heterogeneity is also considered as an adaptive mechanism to cope up with changing higher temperature. Down regulation of various components like OEC, Cytb559, PQ, Rubisco, etc. is the consequences of high temperature stress.

10. Abbreviations

Chl	chlorophyll
CEF	cyclic electron flow
Cyt	Cytochrome
FNR	ferredoxin-plastoquinone oxidoreductase
HS	heat stress
HSP	heat-shock protein
LHC II	I fluorescence band at 20 ms, J fluorescence band at 2 ms, K fluorescence band at 300–400 μs, light harvesting complex II
Fm	maximal fluorescence
Pn	net photosynthesis
Fo	original/initial fluorescence at 50 μs
OEC	oxygen evolving complex
Pheo	pheophytin
PIFs	phytochrome interacting factors
PSI	Photosystem I
PSII	Photosystem II
PQH_2	plastoquinol
PQ	plastoquinone
Rubisco	ribulose-1,5-bisphosphate carboxygenase/ oxygenase
ROS	reactive oxygen species
gs	stomatal conductance
Y_D and Y_Z	tyrosines
Fv	variable fluorescence
WOC	water oxidizing complex

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