Heat stress responses and thermotolerance

Abstract

The rising ambient temperature by plant cells is crucial for the timely activation of various molecular defences before the appearance of heat damage. The heat–threshold level varies considerably at different developmental stages. With a view to survive under heat stress, mechanisms of regulation at the molecular level enable plants to prosper. Traditional breeding contributed for improving heat tolerance meagrely. The genetic transformation approach needs to be accelerated that can mitigate harmful effects by developing improved thermotolerance of crop plants. In this background, a thorough understanding of physiological responses of plants to high temperature, mechanisms of heat tolerance and possible strategies is vital. Temperature changes are sensed through cellular responses due to signal transduction into the cell. A key ingredient for plants temperature sensing ability is a specialized histone protein, dubbed H2A.Z that wraps DNA into a more tightly packed structure. Further, metabolic reactions and internal conductance to CO₂ decrease Rubisco activation, inhibition of Rubisco activase and subsequently slowing down net photosynthesis (PN). Importance of internal conductance to CO₂, heat inducible transcription factor for heat shock response (HSR) involve different HSR genes. The mechanisms, which are regulated at the molecular level, facilitate plants to thrive under heat stress. Exogenous applications of protectants such as osmoprotectants, phytohormones, signaling molecules, trace elements etc. have shown beneficial effects on plants growing under high temperature, due to the growth promoting and antioxidant activities of these compounds. There is acute necessity of crop biotechnology research for evolving genetically modified (GM) crops for a high CO₂ world at this juncture. So, attaining thermotolerance is a lively process to escape damages caused by heat stress.

Keywords: heat-stress threshold, thermostability, heat sensing by plants, thermotolerance

Abbreviations: Pn, photosynthesis; HSR, heat shock response; GM, genetically modified; ROS, reactive oxygen species; MTS, membrane thermal stability; HSFs, heat stress transcription factors; RuBP, ribulose-1,5-bisphosphate; Chl, chlorophyll; Pchlide, protochlorophyllide; LEA, late embryogenesis abundant; CAT, catalase; APX, ascorbate peroxidase; SOD, superoxide dismutase; POX, peroxidase; GST, glutathione reductase; GPX, glutathione s-transferase; CDPKs, Ca-dependent protein kinases; MAPK/MPKs, mitogen-activated protein kinase; ABA, abscisic acid; FLU, fluridone; JA, jasmonic acid; EBL, 24–epibrassinolide; HSCis, heat shock genes; APX1, ascorbate peroxidase gene; HSF, heat shock factor; GUS, β-glucuronidase; miRNAs, microRNAs, HSPs, heat shock proteins

Introduction

Future global climate change, with predicted 1.5–5.8°C increases in temperatures by 2100 has to cause heat stress to create threats to agricultural production. An increase in global temperature ranging from 1.1 to 6.4°C depending on global emissions scenarios, will accompany the rises in atmospheric CO₂. Though high temperature and other abiotic stresses are clearly limiting factors for crops cultivated on marginal lands, crop productivity far and wide is often at the mercy of random environmental fluctuations. Existential assumption cultivated on marginal lands, crop productivity far and wide is often at the mercy of random environmental fluctuations. Existing assumption is acute necessity of crop biotechnology research for evolving genetically modified (GM) crops for a high CO₂ world at this juncture. So, attaining thermotolerance is a lively process to escape damages caused by heat stress. 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The unfavourable effects of heat stress can be mitigated by developing crop plants with improved thermotolerance using an assortment of genetic approaches. For this reason, a thorough understanding of physiological responses of plants to high temperature, mechanisms of heat tolerance and possible strategies for improving crop thermotolerance is crucial. Acquiring thermotolerance is a lively progression by which considerable amounts of plant resources are diverted to structural and functional maintenance to escape damages caused by heat stress. Although biochemical and molecular aspects of thermotolerance in plants are relatively well understood, additional studies focused on phenotypic flexibility and assimilate partitioning under heat stress and factors modulating crop heat tolerance are imperative. High temperature during seed germination may slow down or totally inhibit germination, depending on plant species and the intensity of the stress. At later stages, high temperature may adversely affect vital physiological processes like photosynthesis, respiration, water relations and membrane stability and also modulate levels of hormones and primary and secondary metabolites. Furthermore, for the duration of plant ontogeny, enhanced expression of a variety of heat shock proteins, other stress–related proteins and production of reactive oxygen species (ROS) constitute major plant responses to heat stress. Such studies combined with genetic approaches to identify...
and map genes (or QTLs) conferring thermotolerance will facilitate marker-assisted breeding for heat tolerance and paving the way for cloning and characterization of underlying genetic factors which could be useful for engineering plants with improved heat tolerance.

The changes in temperature might induce cellular responses indicating that temperature is perceived and the temperature signal is transduced into the cell. While the signaling pathways triggered temperature changes, the way plants sense temperature is often considered as hard to pin down. Plants have no inner thermometer as such; however, adjustments in cellular equilibriums triggered by temperature changes act as networked thermostats to sense heat and cold. Amongst these temperature-sensitive devices, membrane fluidity, protein conformation, cytoskeleton depolymerization and metabolic reactions were identified.7

Temperature variations play a role in the reset of internal clocks and diurnal synchronization. For certain species, exposure to a low temperature is necessary to trigger developmental processes such as flowering8 or germination.9 Temperature changes are perceived and then transduced to the nucleus where the transcriptome is altered.10 Nevertheless, advancement has been made on temperature sensing, especially on heat sensing, as heat is a physical parameter that influences molecular (protein, DNA) or supramolecular (membranes, chromosomes) structures through simple thermodynamic effects.

Breeding for selecting genotypes with increased heat tolerance is therefore, one of the most vital objectives in crop improvement programme. This review accentuates briefly on plant responses to heat stress at the whole plant elucidating, in general, the sensing of high temperature by plants providing physiological and molecular grounds, viz., heat stress threshold, cell membrane thermostability, effects on metabolic reactions and internal conductance to CO2; impairment of chlorophyll biosynthetic reactions; thermotolerance mechanisms; finally highlighting crop biotechnology in global climate change.

Heat sensing by plants

Science daily in 2013 September brought into light the 2010 report of Kumar et al.,11 that plants are incredibly temperature sensitive and can perceive changes of as little as one degree Celsius. Plants ‘feel’ the temperature rise and coordinate an appropriate response by activating hundreds of genes and deactivating others as per the DNA is packaged. Specialized histone protein is a key ingredient for plants temperature sensing ability that was evolved by means of the model plant Arabidopsis thaliana. The dubbed H2A.Z wraps DNA into a strongly packed structure known as a nuclosome where with rise of temperature H2A.Z histones allows DNA to progressively unwrap directing nucleosomes to loosen up. As argued by researchers, the basic discovery could ultimately establish to have vital implications for world food security.11 However, Saidi et al.,12 were of the opinion that when temperature elevates, the heat signal is probably transduced by several pathways for coming together into the final activation of HSFs, the expression of HSPs and the onset of cellular therмотolerance.

Heat–stress threshold

Heat stress is often defined as a period in which temperatures are hot enough for a sufficient period of time to cause irreversible damage to plant function or development. Plants can be damaged by either high day or high night temperatures and by either high air or soil temperatures. A plant’s temperature usually runs just above the air temperature. A threshold temperature refers to a value of daily mean temperature at which a detectable reduction in growth begins. Knowledge of lower threshold temperatures is important in physiological research as well as for crop production. Base threshold temperatures vary with plant species. For other plant species, the higher threshold temperature may be lower or higher than 35°C. High temperature sensitivity is particularly important in tropical and subtropical climates as heat stress may become a major limiting factor for field crop production. About 90% of the water that enters a plant’s roots is used for cooling under warm dry conditions. The amount of water lost by a plant depends on its size, the intensity of the surrounding light, temperature, humidity and wind speed. All of which influence evaporative demand. Brief exposure of plants to high temperatures during seed filling can accelerate senescence, diminish seed set and seed weight and reduce yield.13 In addition, heat stress problems also make the plant susceptible to pests and other environmental problems.

The threshold maximum seed zone temperature for emergence of cowpea is around 37°C compared with 25 to 33°C for lettuce, for example. High day temperatures can cause harm to components of leaf photosynthesis by reducing carbon dioxide assimilation rates. A perception of photosynthesis to heat is largely due to damage to photosystem II constituents positioned in the thylakoid membranes of the chloroplast and membrane belongings. Membrane thermostability has been evaluated by measuring electrolyte leakage from leaf disks subjected to extreme temperatures.14 Additionally stable membranes exhibit slower electrolyte leakage.

Critical studies comparing responses of extreme temperatures reveal premature death of plants. Agriculturally important grain legume crop field pea is very sensitive to high day temperatures with death of the plant occurring when air temperatures exceed about 35°C for sufficient duration, whereas barley is very heat tolerant, especially during grain filling. Reproductive development of many crop species is damaged by heat such that they produce no flowers or if they produce flowers they may set no fruit or seeds. The damaging effects of heat stress on reproductive development for cowpea, common bean, tomato, cotton, rice, wheat, maize and sorghum have discussed comprehensively.15,16 In case of wheat, the threshold temperature is 26°C at the post–anthesis stage.17

Cell membrane thermostability

Sustained function of cellular membranes under stress is pivotal for processes such as photosynthesis and respiration.14 Physiological and biochemical screening techniques as a complement to empirical breeding methods could increase selection efficiency. The genes securing heat tolerance may be lost in the breeding programs which rely mainly on only empirical selection. Membrane thermal stability (MTS) can be a significant selection criterion for heat stress tolerance. MTS is determined by measuring of electrical conductivity of aqua use phase in which leaf tissue exposure to high temperature. Membrane stability parameters of genotypes decreased during the later developmental stages. Genetic variation among genotypes for membrane stability can be utilized in wheat breeding in heat–stressed environments.

The integrity and functions of biological membranes are sensitive to high temperature, as heat stress alters the tertiary and quaternary structures of membrane proteins enhancing the permeability of membranes evident from increased loss of electrolytes. Membranes

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are moving mosaics of proteins and lipids. Lipids stagger between monolayers, diffuse within the plane of a monolayer and rotate about their own axes, with their acyl chains also rotating around C–C bonds. Much of the heat sensing occurred through protein unfolding. Since protein conformation changes with temperature, both temperature downshift and temperature upshift can lead to protein unfolding. Yamada et al.,18 evinced that heat–induced protein denaturation could participate to the activation of some heat stress transcription factors (HSFs). Eukaryotic HSFs have a central role in heat induced transcriptome remodelling through their binding to the heat stress elements of promoters. Upon heat exposure, HSP90 is inactivated and this would probably lead to At HSF A1d being activated and inducing heat–responsive genes.9

Heat stress also leads to the oligomerization of a thioredoxin and a thioredoxin–like protein from Arabidopsis, and this oligomerization correlates with them switching from a disulfide reductase activity to molecular chaperone activity.21,22 FAD8, the Arabidopsis ω–3 fatty acid desaturase, has a C–terminal region determining its instability when shifted from 22°C to 27°C. A characteristic of the heat response is less lipid desaturation, so FAD8 desaturation can be considered as a perception event that directly occurs on response effectors.21 A temperature shift from 27°C to 42°C resulted in the disruption of the majority of microtubules in tobacco cells after 30min, as in Arabidopsis roots shifted from 20°C to 42°C.2,24

Metabolic reactions and internal conductance to CO₂

Photosynthesis

Cell metabolism is influenced by temperature effects on enzyme activities. Changes may generate signaling pathways triggering heat/ cold responses. This is illustrated here by temperature–sensitive photosynthesis related processes. Ribulose–1,5–bisphosphate (RuBP) carboxylase/oxygenase (Rubisco) catalyzes the first step in net photosynthetic CO₂ assimilation and photorespiratory carbon oxidation. The enzyme is notoriously inefficient as a catalyst for the carboxylation of RuBP and is subject to competitive inhibition by O₂, inactivation by loss of carbamylation and dead–end inhibition by RuBP. These insufficiencies make Rubisco rate limiting for photosynthesis and an obvious target for increasing agricultural productivity. The regeneration of Pi is necessary for optimal carbon fixation. At low temperatures, all the metabolic reactions, including those of sucrose synthesis, are slower,25 while phosphorylated intermediates accumulate, Pi is not released,26–28 limiting Ribulose Bisphosphate (RuBP) regeneration and photosynthesis.25,29,30 the limitation can be overcome by supplementing cells with phosphate.21,31,32 Phosphate depletion is a signal and some genes induced by low temperatures are also induced by depletion in phosphate.31

Ribulose–1,5–bisphosphate carboxylase/oxygenase (Rubisco; EC 4.1.1.39) activase mRNA and protein synthesis were measured in the leaves of cotton (Gossypium hirsutum L.) plants under control (28°C) or heat–stress (41°C) conditions. In response to high temperature, de novo protein synthesis quickly shifted from mostly expression of Rubisco large and small subunits to the major heat–shock proteins, while de novo synthesis of the constitutively expressed 47– and 43– kDa activase polypeptides was not appreciably altered. However, a 46–kDa cross–reacting polypeptide was also present in heat–stressed plants and constituted about 5% of the total activase after 48h at high temperature. The identity of the heat–induced 46– kDa polypeptide as activase was confirmed by protein sequencing, which showed that its N–terminal sequence was identical to that of the constitutive 47– kDa activase polypeptide. The presence of multiple isoforms for both the 47– and 43– kDa activase polypeptides on immunoblots of two–dimensional gels and the complex banding pattern on Southern blots together suggest the resolution of more than one activase gene and the possibility that the synthesis of the heat–induced activase polypeptide may be regulated transcriptionally. Induction of a new form of activase may constitute a mechanism of photosynthetic acclimation to heat stress in cotton.24

Heat stress inhibits photosynthesis.25,29,30,35,36 Inhibition of net photosynthesis (Pn) by moderate heat stress has been attributed to an inability of Rubisco activase to maintain Rubisco in an active form. The temperature optimum of Rubisco activation was 10°C higher in the creosote bush (Larrea tridentata) compared with the Antarctic hairgrass (Deschampsia antarctica), resembling the temperature response of Pn, which increased markedly with increasing internal CO₂ concentration subjected to moderate heat stress even under nonphotorespiratory conditions. Nonphotochemical quenching of chlorophyll fluorescence, the effective quantum yield of photochemical energy conversion (ΔF/F(m)’) and the maximum yield of PSI (F(v)/F(m)) were more sensitive to temperature in Antarctic hairgrass and two other species endemic to cold regions [i.e. Lysimachia pumila and spinach (Spinaceaoleracea)] compared with creosote bush and three species [i.e. jojoba (Simmondsia chinensis), tobacco (Nicotiana tabacum) and cotton (Gossypium hirsutum)] from warm regions.

Internal conductance to CO₂

Internal conductance to CO₂ transfer from intercellular spaces to chloroplasts poses a major limitation to photosynthesis, but only three studies have investigated the temperature dependence. Based on chloroplast CO₂ concentration, neither maximum rates of carboxylation nor RuBP–limited rate of electron transport peaked between 10°C and 35°C. Both were described well by an Arrhenius function and had similar activation energies (57–70kJ mol (–1)).37

Heat stress–in duced impairment of chlorophyll biosynthetic reactions

When plants are uncovered to low– or high–temperature stress, Chl biosynthesis is affected.38,39 Biosynthesis of porphyrins and particularly that of Chl during early greening stages of seedlings has been elucidated in detail.40–42 Chlorophyll (Chl) biosynthesis in chill (7°C)– and heat (42°C)–stressed cucumber (Cucumis sativus L. cv poinsette) seedlings was affected by 90 and 60%, respectively. Inhibition of Chl biosynthesis was partly due to impairment of 5–aminolevulinic acid biosynthesis both in chill– (78%) and heat–stress (70%) conditions. Protochlorophyllide (Pchlide) synthesis in chill– and heat–stressed seedlings was inhibited by 90 and 70%, respectively.

In heat–stressed seedlings, although δ–aminolevulinic acid dehydratase and porphobilinogen deaminase were partially inhibited, one of the porphyrinogen–oxidizing enzymes, uroporphyrinogen decarboxylase, was stimulated and coproporphyrinogen oxidase and protoporphyrinogen oxidase were not substantially affected, which demonstrated that protoporphyrin IX synthesis was relatively more resistant to heat stress. Protochlorophyllide (Pchlide) oxidoreductase, which is responsible for phototransformation of Pchlide to chlorophyllide, increased in heat–stress conditions by
Heat stress responses and thermotolerance

Plant scientists involved in research on high temperature stress are endeavouring to discover the plant responses that lead to high temperature and they are also trying to investigate how plants can be managed in high temperature environments. Recent widely studied molecular approaches have included omics techniques and the development of transgenic plants through manipulation of target genes.6–8 Such underlying molecular processes may provide ways to develop stress tolerant varieties and to grow agriculturally important crop plants under heat stress. Heat stress differentially affects the stability of various proteins, membranes, RNA species and cytoskeleton structures and alters the efficiency of enzymatic reactions in the cell for which the major physiological processes obstacle and creates metabolic imbalance.9–11 Different metabolic pathways are depended upon enzymes which are sensitive to various degrees of high temperature. It has been suggested that, like other abiotic stress, heat stress might uncouple enzymes and metabolic pathways which cause the accumulation of unwanted and harmful ROS most commonly singlet oxygen (O2), superoxide radical (O2−), hydrogen peroxide (H2O2) and hydroxyl radical (OH) which are responsible for oxidative stress.12 The reaction centres of PSI and PSII in chloroplasts are the major sites of ROS generation though ROS are also generated in other organelles viz. peroxisomes and mitochondria.13 A linear relationship exists between maximal efficiency of PSI and the accumulated ROS.

Heat tolerance is the ability of the plant to grow and produce economic yield under high temperature. Plants have evolved various mechanisms for flourishing under higher prevailing temperatures. They include short term avoidance/acclimation mechanism or long term evolutionary adaptations. Some major tolerance mechanisms, including ion transporters, late embryogenesis abundant (LEA) proteins, osmoprotectants, antioxidant defense and factors involved in signaling cascades and transcriptional control are essentially significant to counteract the stress effects.14,15 The stress responsive mechanism is established by an initial stress signal that may be in the form of ionic and osmotic effect or changes in the membrane fluidity. This helps to re-establish homeostasis and to protect and repair damaged proteins and membranes.16

Activities of different antioxidant enzymes are temperature sensitive and activation occurs at different temperature ranges but the activities of these enzymes increase with increasing temperature. Chakrabortty et al.17 observed that catalase (CAT), ascorbate peroxidase (APOX) and superoxide dismutase (SOD) showed an initial increase before declining at 35°C; while peroxidase (POX) and glutathione reductase (GR) activities declined at all temperatures ranging from 20 to 50°C. In addition, total antioxidant activity was at a maximum at 35–40°C in the tolerant varieties and at 30°C in the susceptible ones. Their activities also differ depending upon tolerance or susceptibility of different crop varieties, their growth stages and growing season.18,19

The activity of the enzymes glutathione S-transferase (GST), ascorbate peroxidase (APOX) and catalase (CAT) was more enhanced in the cultivar showed better tolerance to heat stress and projection against ROS production. They reported that the tolerance of the wheat varieties appeared to be correlated with the antioxidant level, though changes in activity were observed for different antioxidant enzymes. Antioxidant defense mechanism plays an important role in the heat stress tolerance of wheat genotypes and it was observed that the activities of SOD, APOX, CAT, GR and POX increased significantly at all stages of growth in heat tolerant cultivars (C 306) in response to heat stress treatment, while susceptible cultivar (PBW 343) showed a significant reduction in CAT, GR and POX activities in the heat tolerance treatment. Further, to create response in specific cellular compartments or tissues against a certain stimuli, interaction of cofactors and signaling molecules are required. Signaling molecules are involved in activation of stress responsive genes. Various signal transduction molecules related to stress responsive gene activation are depending upon plant type, types of stresses. Some broad group of those are the Ca–dependent protein kinases (CDPKs), Mitogen–activated protein kinase (MAPK/MPKs), NO, sugar (as signaling molecule), phytohormones.17 These molecules together with transcriptional factors activate stress responsive genes.

In recent decades, exogenous application of protectant such as osmoprotectants, phytohormones, signaling molecules, trace elements, etc., have shown beneficial effect on plants grown under heat tolerance as these protectants has growth promoting and antioxidant capacity.64–66 Accumulation of osmolytes such as proline, glycine betaine and trehalose is a well-known adaptive mechanism in plants against abiotic stress conditions including heat tolerance. Since heat sensitive plants apparently lack the ability to accumulate these substances, heat tolerance in such plants can be improved by exogenous application of osmoprotectants.24,25 Proline and glycine betaine application considerably reduced the H2O2 production, improved the accumulation of soluble sugars and protected the developing tissues from heat stress effects. However, Pro was more effective than glycine betaine in that study. Exogenous proline and glycine betaine application also improved the K+ and Ca2+ contents, and increased the concentrations of free proline, glycine betaine and soluble sugars which rendered the buds more tolerant to heat tolerance. Identically, exogenous applications of several phytohormones were found to be effective in mitigating heat stress in plants. Chhabra et al.19 studied the phytohormones induced amelioration of heat tolerance stress in Brassica juncea and found that soaking seeds in 100μM IAA, 100μM GA, 50 and 100μM Kinetin and 0.5 and 1μM ABA were effective for mitigating the effect of heat stress (47 ± 0.5 °C). The significant observation was that both growth promoting and growth retarding hormones were effective in mitigation of heat stress effects. The role of growth promoting hormone in the mitigation of heat stress was at a concentration which was otherwise lethal or toxic to its growth seedling stage. Salicylic acid is a plant hormone found to be an effective protectant under heat stress.

The oxidative damages in abscisic acid (ABA) treated plants were also much lower than non–treated plants under heat stress condition which was indicated by reduced MDA and H2O2 contents. In the contrary, inhibitor of ABA biosynthesis, fluridine (FLU) reverted the actions induced by ABA which suggest a clear role of ABA in mitigating heat–induced damages. Chen et al.,21 treated grape seedlings with 50μM jasmonic acid (JA) solution and observed that JA could extenuate the change of stress under heat stress (42°C). This protection was accompanied by the upregulation of antioxidant enzyme’s (SOD, CAT and POD) activity compared with these
untreated under heat stress. Kumar et al., investigated the effect of different concentrations of 24–epibrassinolide (24–EBL) on growth, antioxidant enzyme of mustard (*B. juncea*) seedlings. Polyamine provides protection to plant from high temperature stress in different ways. They can affect photosynthesis in different ways. Structure and function of the photosynthetic apparatus can be regulated effectively by PAs. Polyamines are able to maintain thermostability of thylakoid membranes under heat thus increase photosynthetic efficiency. The up-regulation of several heat inducible genes, commonly referred as “heat shock genes” (HSGs) which encode HSPs and these active products are very much necessary for plant’s survival under fatal high temperature. High temperature induced constitutive expression of most of these proteins protect intracellular proteins from being denaturation and preserve their stability and function through protein folding; thus it acts as chaperones. The HSPs are extremely heterogeneous in nature and this dynamic protein family is expanding continuously as per the recent researches are going on. Over expression of plant HSFs can increase plant’s thermo-tolerance, but gene knockouts of individual HSFs tested so far have had little effect on survival at high temperature. In plants, there are a number of non-HSP transcripts that are upregulated by heat. In particular, the *Arabidopsis* cytosolic ascorbate peroxidase gene (*APX1*) has been shown not only to be heat upregulated, but also to contain a functional heat shock element (HSE) in its 5′-promoter region. As heat stress tolerance is a polygenetic trait (controlled by different sets of genes), various different components of tolerance are critical at different developmental stages or in different tissues of plant; hence, it shows spatio-temporal mechanism and regulation. Thus, the use of genetic stocks with different degrees of heat tolerance, correlation and co-segregation analyses, molecular biology techniques and molecular markers to identify tolerance QTLs are promising approaches to dissect the genetic basis of plant’s thermostolerance.

**Heat-inducible transcription factor**

The heat shock response (HSR), defined as a transient reprogramming of gene expression, is a conserved biological reaction of cells and organisms to elevated temperatures. HSR has been of great interest for studying molecular mechanisms of stress tolerance and regulation of gene expression in plants. The temperature for the induction of HSR coincides with optimum growth temperature for any given species, which is normally 5–10°C above normothermic conditions. The kinds of this response include induction of HSPs and subsequently acquisition of a higher level of thermostolerance. The transient synthesis of HSPs suggests that the signal triggering the response is lost, inactivated or no longer recognized under conditions of long-term heat treatment. The involvement of HSPs in heat-stress tolerance is a logical model, but direct support for function of HSPs in promoting thermostolerance has been difficult to obtain. Eukaryotic cells respond to elevated temperature or heat shock (HS) by inducing the transcription of genes encoding proteins such as molecular chaperones. Many of these proteins are involved in preventing or repairing the damage caused by heat stress and thus confer increased thermostolerance. This phenomenon, known as HSR response, is initiated by the activation of the HS transcription factor (Hsf), a conserved protein present in all eukaryotic organisms studied to date.

In the last decade, two research groups independently reported the function of HsfA2 by characterizing the same *Arabidopsis* T-DNA knockout line. The disruption of HsfA2 caused a slight reduction in expression of some Hsp genes upon HS treatment and that the knockout mutant displayed reduced basal and AT as well as oxidative stress tolerance, while overexpression of HsfA2 enhances tolerance under these stress conditions. Schramm et al. identified a subset of genes whose expression was reduced upon HS treatment in the knockout mutant. They showed that HsfA2 bound to the promoter regions of selected target genes and activated a β-glucuronidase (GUS) reporter that was fused to them. According to the transcriptome studies, six Hsf genes, HsfA2, HsfA4a, HsfA7a, HsfB1, HsfB2a and HsfB2b, are significantly up-regulated in *Arabidopsis* leaves by HS treatment (37°C for 1h). This result is comparable to microarray data except that of *Arabidopsis* sample (ecotype Columbia [Col–0] 5–day-old seedlings), HsfA7b but not HsfA4a was significantly responsive to heat. From the HSR microarray data available to date, HsfA2 is the gene most induced by heat, which suggests a dominant role for the gene in sustaining the HSR, a role that cannot be replaced by HsfA7a and HsfA7b.

“**Ommics** technologies”

The “omics” technologies have provided novel opportunities and expectations 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. DNA is the starting point of all molecular evidences related to heat stress tolerance in plants and contains several heat stress responsive genes in their genome (genomics). A large number of genes with potential roles in heat stress responses have been identified using genetic screens and genome wide expression studies. Transcriptory products (mRNAs), from such genes in the genome, have made their transcriptome (transcriptomics) and then proteome (proteomics) when they translate into the functional proteins (responsible for stress tolerance). In response to developmental and environmental cues, plants employ a post-transcriptional regulation of gene expression by non-protein coding small RNAs or microRNAs (miRNAs). Overexpression of miRNA-resistant target genes will help to overcome post-transcriptional gene silencing, and thus may lead to better expression of engineered trait in transgenic plants. Understanding the roles of small RNAs in transcriptome homeostasis, cellular tolerance, phenological and developmental plasticity of plants under heat stress and recovery will help genetic engineering of stress tolerance in crop plants. In addition, microarray technology has recently become a powerful tool for the systematic analysis of expression (or transcriptome) profiles of large numbers of genes those are induced or repressed by heat treatment. Recent microarray studies in *Arabidopsis* deficient with *APX* gene, however, have found that certain HSPs are expressed typically under other stress conditions, although expression of HSPs under heat stress occurs normally.

**Crop biotechnology in global climate change**

Advances in the understanding of crop-environment interactions at the molecular, biochemical, physiological, and agronomic scales, as well as their relevance to biotechnological crop improvement, have been extensively reviewed. An entire range of research tools is encompassed by Biotechnology that scientists use to comprehend, apply and thereby influence the genetic make-up of organisms for use in agriculture, crops, forestry, livestock and fisheries. Genetically modified crops have the ability to grow faster with development time being reduced from years to months as compared to that of...
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The wheat yield could be limited by the grain number and the grain size, which are established to a large extent at the period around anthesis (flowering), a stage in development known to be sensitive to high temperature stress. A modelling study for the main wheat growing regions of Australia demonstrated that variations in average growing–season temperatures of 2°C can cause reductions in grain production of up to 50%, most of which can be attributed to increased leaf senescence as a result of high temperatures. Another study in the USA demonstrated that nonlinear temperature effects on grain yields, which show a steep yield decline after temperature exceeds crop–specific thresholds, could lead to severe damages to U.S. corn and soybeans yields under climate change. If the crop is unstressed, it establishes the grain number and its potential size at sufficiently large values to accommodate biomass produced during grain filling. In this case, the yield is effectively source–limited. The grain number and the grain size can be substantially reduced if a cultivar, sensitive to heat stress, is exposed to a short period of high temperature around flowering, limiting the capacity of grains to store newly produced biomass. In this case, grain growth becomes a sink–limited process. In an experiment on the combine effects of CO₂ and temperature on the grain yield, Mitchell et al. observed that a temperature of 27°C or higher applied mid–way through anthesis could result in a high number of sterile grains and considerable yield losses. Wheeler et al. used a temperature gradient tunnel system to demonstrate that at 30°C or elevated prior to anthesis significantly reduced the grain number and, subsequently, yield. Crop yields are predicted to decrease approximately 10% for every one–degree increase in temperature. Wheat cultivation spread to a wider range of climatic conditions, approximately 10% for every one–degree increase in temperature. Crop yields are predicted to decrease at 30°C or elevated prior to anthesis significantly reduced the grain yield, Mitchell et al., Wheeler et al., and high–temperature unfolding of yeast frataxin under physiological conditions. 

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Conflict of interest

The author declares no conflict of interest.

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