Review article
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Plant Responses to Heat Stress

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Abstract:
Climate change is happening at a breakneck speed around the world. Due to continuous alterations, the climate has created various abiotic stress situations in which heat stress is among the most crucial that negatively impact the growth, development, and metabolism of crops. When stress is too intense, the signaling process leading to cell apoptosis is also triggered. The plant reaction to high temperature (HT) depends on the degree, time duration for exposure, and plant type. Heat stress affects plant development by causing a variety of biochemical, morphological, physiological, and molecular modifications. The ability of plants to sense the HT signals, create and forward them, to begin necessary biochemical or physiological modifications to tolerate the stress is critical for their capability during HT stress. Furthermore, the responses of plants to heat stress (HS) are complicated, with unknown physiological features as well as molecular or gene pathways. A noteworthy feature of plants is that they show numeric transgenic, epigenetic approaches against heat stress. This review describes and discusses the numerous ways to improve plant thermotolerance and different molecular and physiological responses, modifications, and tolerance to HT at the cellular level.

***Keywords*:** stressors, cell apoptosis, morphological, physiological, metabolism.

Highlights:

- The physiological responses of plants to high temperature
- The molecular responses of plants to high temperature
- The Epigenetic Modifications of the plant to high temperature
- Strategies to develop heat stress tolerance in plant

1. Introduction:

Plants are unable to shift to more conducive surroundings when exposed to abiotic or biotic challenges, resulting in significant reductions in growing plants, maturation, and efficiency (Lippmann *et al*, 2019). Heat is implicated in widespread agriculture failures worldwide, frequently in conjunction with droughts or other stresses (Mittler *et al*., 2012). High temperatures (HT) have been recorded more often in recent years around the world, which is attributed to changing climate. Due to the high climate, global temperature poses a severe danger to food harvests (Wang *et al*., 2018). Heat stress(HS) is commonly characterized as a heating rate that exceeds a threshold value for a long time to inflict irreparable loss to plant growth (Wahid *et al*.,2007). Cellular damage and death can occur due to thermal stress and exposure bracketing to relatively high temperatures.

HS may significantly constrain agricultural crop yield in tropical and subtropical regions. HT reactions vary by crop life span or are generally physiological, molecular, and morphological responses or plant developmental processes. Germination percentage, photosynthesis efficiency, water usage efficiency (WUE), cell development and multiplication, flowering synthesis, pollen persistence, spikelet viability, seed yield, and plant quality are all adversely affected by the HS (Kaur *et al*., 2018; Sarwar *et al*., 2018; Hatfielf *et al*., 2015). Other damaging impacts of HS on plants are shown in Figure 1.

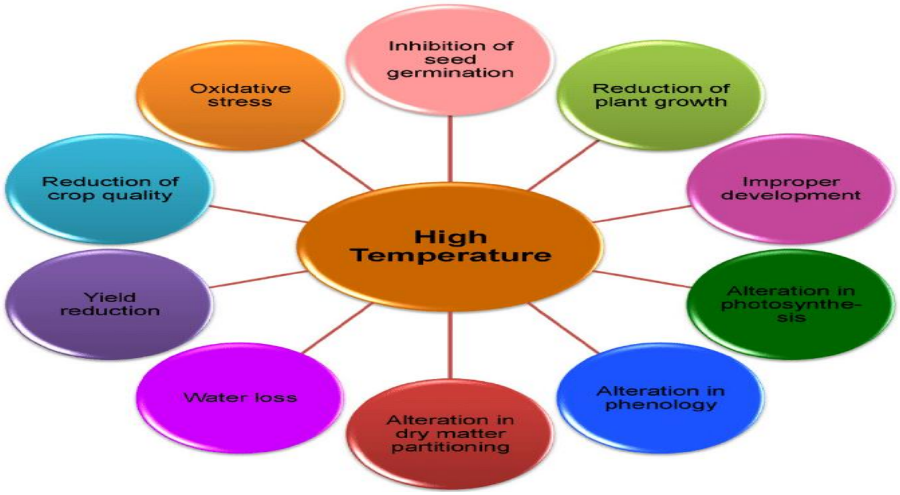

Figure 1: Shows the HT effect on plants.

Excessive formation of reactive oxygen species (ROS), which mainly contributes to oxidative stress, is one of the principal effects of HT stress (Hasanuzzaman *et al*., 2013). Plants constantly face extinction in various stressful environments, including high temperatures. A plant may survive extreme heat to a certain level by undergoing physiological modifications within its body and usually by sending out impulses to adjust its metabolic activity. Plants change their metabolic relation to HT in a variety of ways, including generating suitable soluble compounds that can arrange proteins and cellular structures, sustain cell turgidity through osmoregulation and alter the antioxidant system to reinstate cellular redox status and homeostatic conditions (Janská *et al*, 2010; Munns and Tester, 2008; Valliyodan and Nguyen, 2006). HS alters the genes' appearance intricated in protective action from HT at the molecular level (Chinnusamy *et al*., 2007; Shinozaki *et al*., 2007). In situations like HT, modifications of gene coding slowly alter physiological and biochemical pathways, leading to the formation of thermal dissipation in the manner of acclimatization and, in the best case, adaptations (Moreno and Orellana, 2011; Hasanuzzaman *et al*., 2013). (Mirza *et al*., 2010; Hasanuzzaman *et al*., 2012; Waraich *et al*., 2012; Barnabas *et al*., 2008). Exogenous treatments of protectants have recently been proven to be beneficial in reducing HT-induced destruction in crops are in the form of

- Plant hormones (gibberellin, salicylic acid, abscisic acid, brassinosteroids etc.)
- Polyamines (spermine, putrescine, spermidine)
- Trace elements (silicon, selenium etc)
- Osmoprotectants (trehalose, proline, etc)
- Nutrients (calcium, phosphorous, nitrogen, potassium etc)
- Messenger molecules (nitric oxide)

The temperature sensing system in plants is depicted in a broad sense. HS may alter the plasma membrane's fluidity, which might trigger Ca^{2+} networks, causing an influx of Ca^{2+} ions (Saidi *et al*., 2009). Such Ca^{2+} ions trigger crop signaling pathways, causing them to respond appropriately to HS via engaging HS-responsive genes (Ohama *et al*., 2017). HS additionally induces metabolic changes that impair protein expression, as well as an increase in Ros formation, which leads to metabolic instability and protein misfolding, which is identified as endoplasmic reticulum (ER) or cytosolic stress, which triggers the unfolded protein response (UPR) (Mittler *et al*., 2012). Micro RNAs, chromatin, and epigenetic modifications have recently been revealed to have a role in HSR modulation and HS memory retention (Stief *et al*., 2014b; Ahuja *et al*., 2010).

2. Plants Respond to Heat Stress:

The intensity of heat, time, and type of crop all influence plant sensitivity to HT. The most prevalent impact of heat stress is reported in Table (1). Intense HT can cause cell injuries or death in minutes, leading to a catastrophic failure of cells (Ahuja *et al*., 2010). Plants exposed to HS have shown three different sorts of reactions. Basal-acquired thermotolerance (AT) and programmed cell death (PCD) are among them (Mittler *et al*., 2012; Locato and De Gara, 2018; Guihur *et al*., 2021).

- The capability of an organism to deal with very increases in temperature is referred to as basal thermotolerance.
- AT relates to a plant's capacity to deal with fatal HT after acclimation to sublethal HT.

- The death of a cell as a result of internal activities such as apoptosis or autophagy is known as PCD.

Table 1 summarizes many of the most prevalent impacts of heat stress.

Plant	Developmental stage	HT	Significant impact	References
Rice	Vegetative stage	25–42.5 °C	The level of CO_2 uptake has slowed.	(Yin et al., 2010)
Rice	Reproductive stage	32 °C (night)	Reduced grain size, thickness, and volume and enhanced spikelet viability.	(Mohammad and Tarpley, 2010)
Okra	During the entire growth	32°C, 34°C	Lower production and impairment to pods performance indicators like fiber content or Ca-pectate disintegration.	(Gunawardhana, and de Silva, 2011)
Rice	Heading stage	>33°C	fertility of pollens and spikelet were suppressed.	(Cao et al., 2009)
Maize	From Pre-anthesis to silking onwards	33-40°C	Plant and ear growth rates are greatly affected.	(Edreira and Otegui, 2012)
Chili pepper	Reproduction, maturation and harvesting stage	38°C (day), 30 °C (night)	Fruit size and volume were lowered, while the percentage of aberrant seeds per fruit improved.	(Pagamas and Nawata, 2008)

3. Plants' Physiological Reaction to HS:

HS harms several physiological activities, including respiration, photosynthesis, membrane thermostability, transpiration, and osmotic control. These are discussed as follows

3.1 Photosynthesis:

Photosynthesis is particularly sensitive to HT, and heat damage causes cell energy imbalance. In general, HS lowers photosynthetic activity, resulting in a shorter life cycle of plants and lower output (Xalxo *et al*., 2020). The photochemical responses in the thylakoid lamellae and metabolic pathway in chloroplast's stroma are mainly influenced by high temperatures (Wang *et al*., 2018; Wise *et al*., 2004). Photosystem II (PSII) is the most susceptible of the chloroplast thylakoid membrane protein molecules to HS. If PSII experiences substantial overheating, it significantly impacts photosynthetic electron transfer and ATP production (Wang *et al*., 2018). PSII light-harvesting units slip off the thylakoid membrane owing to enhanced volatility of thylakoids at HT, resulting in compromised PSII stability, thus affecting photosynthetic electron transport (Baker and Rosenqvist, 2004; Janka et al., 2013; Mathur et al., 2014). PSII breaks water with light energy and transports electrons to another reaction. PSII, on the other hand, is vulnerable to abiotic stress, particularly heat and oxidative stress. Plants experience oxidative stress in response to HT. According to a new analysis, during photosynthesis, reactive singlet oxygen produces $^1O_2$, which could destroy PSII reaction center proteins, triggering the PSII repair cycle (Dogra and Kim, 2019). Heat stress and oxidative stress may prevent the healing of damaged PSII by inhibiting PSII protein production (Takahashi and Murata, 2008), resulting in a reduction in plant photosynthesis efficacy.

Chlorophyll (Chl), the principal photosynthetic component found in chloroplasts' thylakoids, can absorb light energy and facilitate electron transport in the early and most critical phases of photosynthesis (Wang *et al*., 2018). Whenever plants are exposed to adverse environmental conditions, such as heat, the amount of chlorophyll in their leaves declines, resulting in leaf senescence or chlorosis (Allakhverdiev *et al*., 2008; Lim *et al*., 2007; Rossi *et al*., 2007). Thermal treatment dramatically raises the efficiency of chlorophyllase and Chl-degrading peroxidase, leading to a significant drop in Chl levels, as shown in Figure 2 (Wang *et al*., 2018). Balancing chlorophyll formation and degradation is critical for maintaining the photosynthetic machinery, which influences crop development and production.

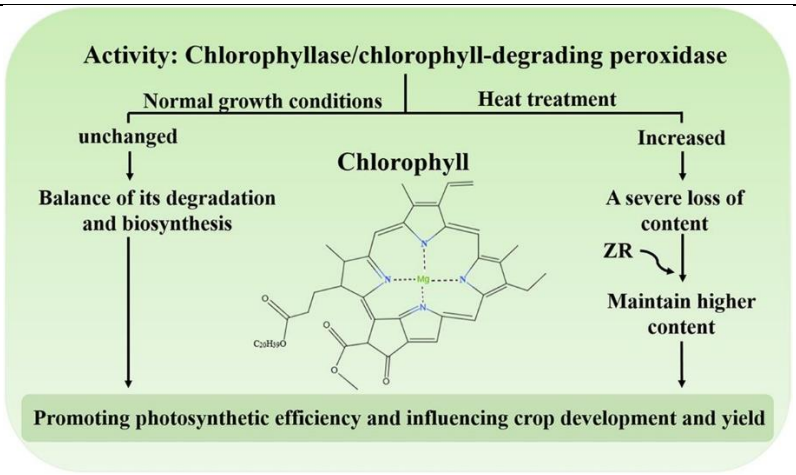

Figure 2: Under heat stress, chlorophyllase and Chl-degrading peroxidase efficiency.

3.2 Oxidative Damage:

Enzymes susceptible to varying levels of HTs are required for diverse metabolic processes. Plant responses to HS accumulate ROS, including singlet oxygen ($^1O2$), superoxide radical (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radical (OH), which causes oxidative damage (Nosaka and Nosaka, 2017). The activation sites of PSI and PSII in chloroplasts are primary locations of ROS formation, although ROS is produced in other organelles such as peroxisomes and mitochondria (Halliwell, 2006). The maximum efficacy of PSII and the acquired ROS have a linear relation. It is conceived that under high-temperature stress, less photon absorption happens due to thermal injury of photosystems I and II (Møller et al., 2007). If PSI and PSII acquire the photon energy under such stress conditions, the surplus necessary for CO2 fixation is termed surplus electrons, which act as a source of ROS (Møller et al., 2007). PSI and PSII are the primary sources of ROS. Under HS, ROS could also cause programmed cell death (PCD). Plants, on the other hand, have evolved ways to detoxify ROS and improve their heat resistance. ROS causes oxidative stress in plants by changing membrane characteristics, destroying proteins, and deactivating enzymes, all of which reduce plant cell survival. Though ROS have detrimental consequences on plant metabolism, they have also been speculated to have signal tendencies that activate heat shock response and lead to the development of heat tolerance mechanisms. These signaling behaviors are unknown and should be explored further (Asada, 1987).

3.3 Cell Membrane Disruption:

Plants that thrive at high temperatures would first retain permeability and stability, necessitating membrane fluidity variability. As discussed before, thylakoids and cell membranes are usually one of the first to be altered by high thermal effects (Źróbek-Sokolnik, 2012; Ruelland and Zachowski, 2010; Mittler *et al*., 2012). HT causes the decomposition of the structural proteins of the cellular membrane, which speeds up the flow of lipids. Such consequences enhance membrane permeability, enabling ions to escape or rendering the frameworks more susceptible to breaking, which inhibits a variety of cellular (ion and metabolite transfer) and physiological (e.g., photosynthesis) functions (Źróbek-Sokolnik, 2012; Ruelland and Zachowski, 2010; Taiz and Zeiger, 2015; Prasad *et al*., 2008). During stressful events, plants of *Arabidopsis thaliana*, subjected to 35°C for 22 days, showed a drop-in membrane concentration and an increase in lipid breakdown (Tang *et al*., 2016). Nijabat *et al*., (2020) looked at stress markers in sensitivity to HT, first at early. Then a late seedling developmental period of wild and cultivated carrot germplasm showed that cultivated carrots were found to be the most HT tolerant with the most stable cell membranes.

3.4 Chloroplast and Mitochondrion Responses:

Carbon metabolism and energy conversion occur in the chloroplast and, mitochondrion, membrane organelles. Plants' chloroplasts and mitochondria play a role in their sensitivity to heat stress, as shown in Figure 3. PSII in chloroplasts and complexity I/III in mitochondria is the other vital sources of ROS formation beside the plasma membrane (PM). The fluidity of the thylakoid membrane is affected by heat, and unsaturated lipids at the benzoquinones (BQ_s) site are more susceptible to peroxidation. The electron transport chain is constrained, and $^1O_2$ forms on the electron-accepting end while •OH forms on the electron-donating end. As a result, the PSII electron transport system is inactivated whenever plants are subjected to HT (Yadav and Pospíšil, 2012). HT activates the chloroplast unfolded protein response (UPR) and backward signals. Extreme heat promotes cardiolipin lipid peroxidation in the mitochondrial membrane, which reduces cytochrome c oxidase (CCO) function (Paradies *et al*., 1998); however, membrane depolarization through protonophore CCCP can prevent ROS

synthesis (Fedyaeva *et al*., 2014). The suppression of electron transfer in the respiratory chain causes the ROS formation, which activates both the mitochondrial UPR and the reverse signaling paths.

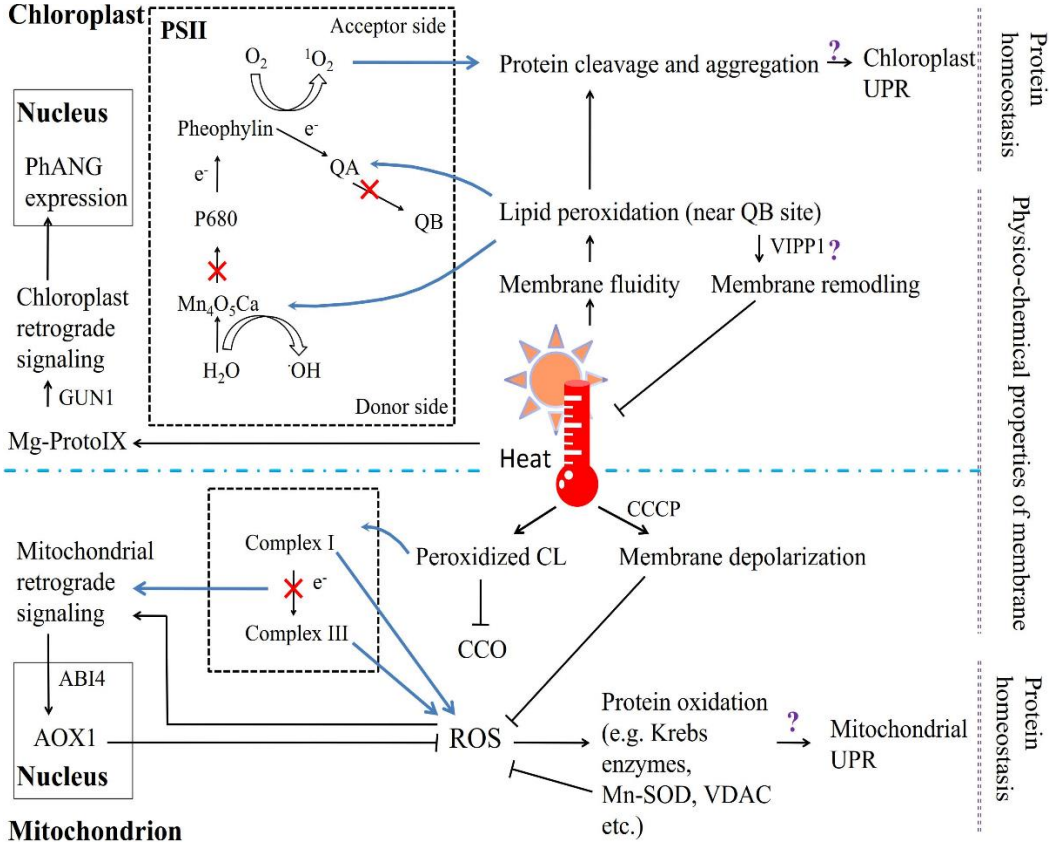

Figure 3: shows the role of chloroplast and mitochondria in response to heat stress.

4. Plants' Molecular Reaction to HS:

4.1 Heat Stress Responses and Transcriptional Regulation:

Numerous plants have looked into the molecular reactions to HS to discover the complexities and pathways involved in high-temperature adaptation and protection (Qu *et al*., 2013; Driedonks *et al*., 2016). When plants are exposed to HS, a set of heat shock transcription factor (HSF) and heat shock protein (HSP) genes are activated, and proteins coded by these genes, like chaperones and ROS scavengers, are essential for plant stress responses. HSFs quickly promote the transcription of HSPs, and both HSFs and HSPs are essential in the plant's HS responses and withstand extreme heat (Ohama *et al*., 2017; Ren *et al*., 2019).

HSF family members HsfA1s act as "master regulators" in *Arabidopsis*, stimulating HSR genes and increasing heat tolerance, as shown in Figure 4 (Ohama *et al*., 2017). HS promotes HsfA1 expression. Also, HsfA1 activity is strictly controlled by HT (Ohama *et al*., 2017). The inhibiting impact of HSP70 and HSP90 (Heat Shock Protein 70 and 90, respectively) on HsfA1 activity is prevented by HS, resulting in HsfA1 activation (Hahn *et al*., 2011). HS also causes a rise in cytoplasmic Ca^{2+} content, which may be critical for HsfA1 activity regulated by the Ca^{2+} channels CNGCs. CBK3, a CaM-binding protein kinase, phosphorylates HsfA1s, improving its interaction with intended genes downstream (Liu *et al*., 2008). After activation. HsfA1s influence the production of several HSR genes and microRNAs, including HsfA2, DREB2A, and miR398. These genes induce the responses against heat stress. m iR398 suppresses the activity of the ROS scavenger genes CSD1, CSD2, and CCS1, resulting in an increase in ROS generation and activation of HsfA1s. Furthermore, PAP (30-PHOSPHOADENOSINE 50-PHOSPHATE)-XRN module regulates miR398 transcription. Additionally, HsfA1s, JUB1 (JUNGBRUNNEN 1), and MBF1c (MULTIPROTEIN BRIDGING FACTOR 1C) all stimulate DREB2A in response to HS (Suzuki *et al*., 2011; Wu *et al*., 2012). HsfA2 is a crucial modulator of H3K4 methylation in HSR genes, which is required for their expression to persist. Under non-stress situations, upregulation of DREB2C, a similar DREB2A gene, promotes the HsfA3 gene; hence, DREB2C should regulate the HsfA3 gene during HS (Chen *et al*., 2010). HsfA3 is a key HS-responsive TF because deletion or suppression mutants of HsfA3 led to a decreased regulation of potential target HSP genes under HS (Yoshida *et al*., 2008; Schramm *et al*., 2007). HsfA2 is a HsfA1 primary

target gene. According to a study, a spliced version of HsfA2 produced during HS is converted into a tiny condensed HsfA2 isoforms (S-HsfA2), which then attaches to the HsfA2 locus and triggers the HsfA2 gene (Liu *et al*., 2013). In *Arabidopsis*, HsfBs (HsfB1 and HsfB2b) are also downstream target genes of HsfA1s. In a negative feedback system, HsfBs are considered to precisely modulate the HSR by inhibiting the function of HsfA1s (Ikeda *et al*., 2011). In contrast, in tomatoes, HsfB1 was shown to operate as a transcriptional activator of tomato HsfA1 (Bharti *et al*., 2004).

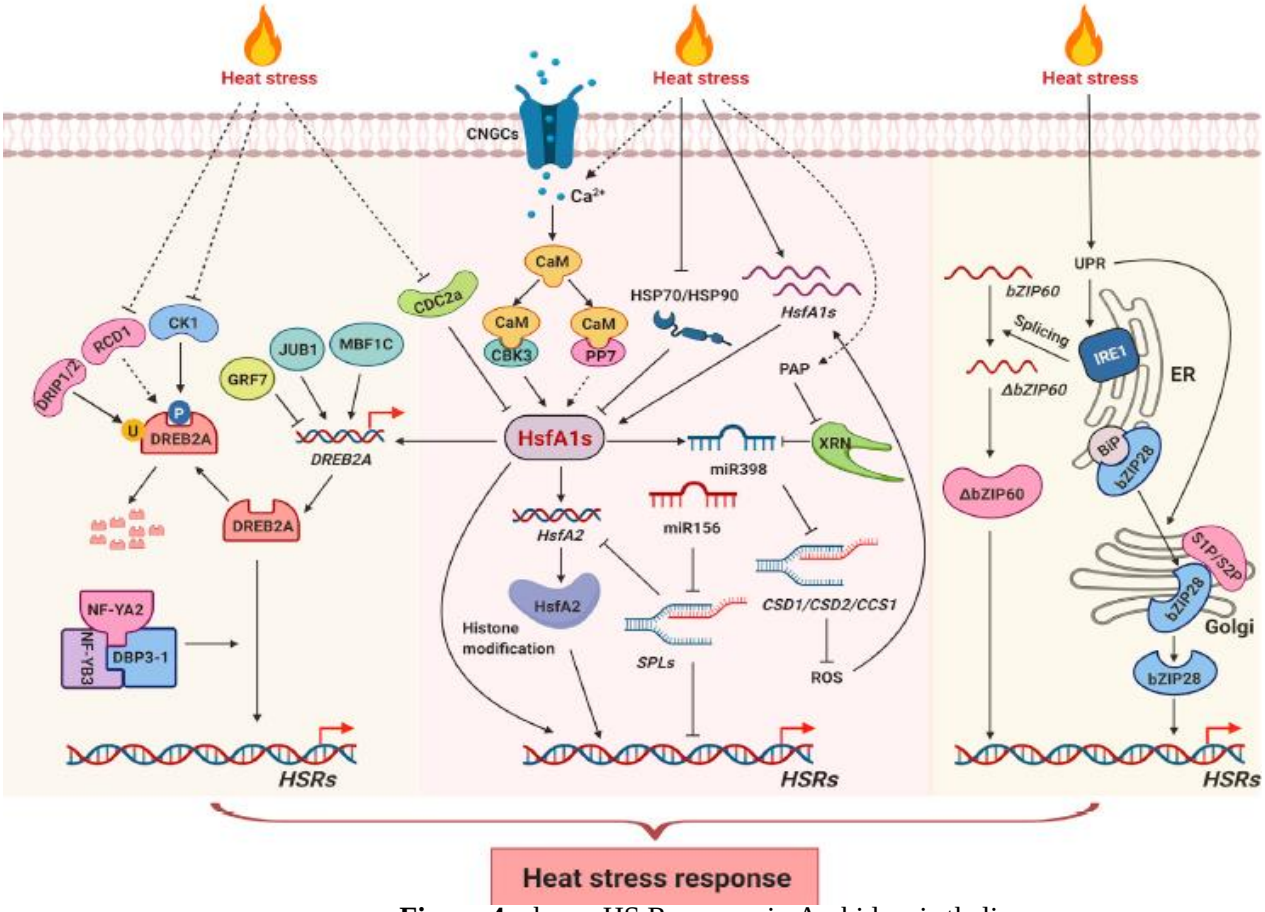

Figure 4: shows HS Response in Arabidopsis thaliana.

In various plants, the role of HSPs in the resistance mechanisms after HS has been well documented (Park and Seo, 2015; Comastri *et al*., 2018). Massive analysis of cDNA ends (MACE) was used to analyze the expression of genes in tomato leaves, and it revealed that 2203 genes (9.6% of the total) had increased transcription amount in exposure to HT (Fragkostefanakis *et al*., 2015). All those small coding HSP (sHSP) family members showing the most significant induction of nine out of 100 Hsp40 genes were slightly higher than heat.

Notably, activation of transcription of a small proportion of genes mediated by other transcriptional regulators, including WRKY, bZIP, and MYB, can result in a modest but considerable level of evolved HS tolerance in plants. WRKYs play a role in stress reactions and developmental and physiological events. WRKY18, WRKY25, WRKY26, WRKY33, WRKY39, WRKY40, WRKY46, and WRKY68 all work together to promote plant thermostability in HS by favorably modulating HSP-related signaling mechanisms (e.g., HSFs, HSPs, and MBF1c) (Li *et al*., 2010; Li *et al*., 2011).

4.2 Heat Stress Responses and Misfold Proteins Regulation:

Proteins sequencing during HS settings aids in the identification of proteins that respond to stress, and further study of such proteins elucidates their role in stress resistance pathways (Priya *et al*., 2019). Heat shock proteins (HSPs) are vital in securing cells from cytotoxicity by helping misfolded proteins fold properly or guiding them to destruction. As a result, prospective protein aggregates are prevented from injuring the cell by creating insertion. HSPs aid in the appropriate folding of freshly generated proteins, protein translocation through membranes, protein aggregation inhibition, and the destruction of misfolded proteins. HS causes misfolded proteins and the buildup of ROS, both of which are detrimental to plants (Qi *et al*., 2018). To maintain survival, plants should renature or destroy misfolded proteins and eliminate ROS (Figure 3). Most HSPs are molecular chaperones that help stabilize proteins and transmit signals during HS (Arce *et al*., 2018). In rice,

OsHSP101 is a good thermotolerance and heat storage regulator (Lin *et al*., 2014). Heat-induced cytotoxic deformed proteins are removed by the 26S proteasome a2 subunit protein OsTT1 (THERMOTOLERANCE 1), which improves rice thermotolerance (McLoughlin *et al*., 2019).

Stress disrupts protein folding, resulting in an overabundance of misfolded or fragmented proteins. Proteotoxic stress is caused by impaired proteostasis, which hampers cell functioning, and plants must refold or eliminate misfolded proteins to maintain healthy growth. HEAT SHOCK PROTEIN 101 (HSP101), a molecular chaperone protein, is further defined in the issue of Plant Physiology when McLoughlin *et al*. (2019) investigated the intracellular location of GFP-labeled HSP101 during Arabidopsis plant heat stress adaptation.

The endoplasmic reticulum (ER) is an essential organelle in plants that performs critical processes such as protein synthesis and folding. The deposition of misfolded proteins in the ER causes "ER stress." Molecular actors in the ER membrane, such as chaperones and co-chaperones, lessen this stress by upregulating lipid formation, assisting protein folding, suppressing translation, and expanding ER space (Braakman and Hebert, 2013; Pastor-Cantizano *et al*., 2020). In plants, a pair of unfolded protein response (UPR) genes are active in ER stress sensing and signaling. There exists an ER imbalance, which activates the UPR pathways (IRE1-bZip60 and bZip28/bZip17). The first arm, Inositol-requiring enzyme 1(IRE1), regulates unconventional splicing of bZip60 in the cytosol, while the second arm, bZip17/bZip28, mediates intramembrane sequential proteolysis (Deng *et al*., 2011; Liu and Howell, 2016). Each bZip TF has its processing and activating strategy that includes either proteolytic cleavage or alternative splicing (Lu *et al*., 2012; Sita *et al*., 2017). Heat stress, among so many other abiotic stresses, has been proposed as a primary source of ER stress (Afrin *et al*., 2020).

4.3 Heat Stress Responses and ROS Homeostasis:

ROS are produced by enzymatic and nonenzymatic processes in plants under normal and stressful situations and are linked to several stages of plant development as well as growth and HS triggers the release of ROS scavengers, which eliminate excessive ROS. Heat stress is more severe in ROS-scavenging genes Ascorbate peroxidase (APX) and Catalase (CAT) (Baxter *et al*., 2014; Vanderauwera *et al*., 2011). Additional major ROS scavengers in Arabidopsis are CSD1 (COPPER/ZINC SUPEROXIDE DISMUTASE 1), CSD2, and CCS1 (COPPER CHAPERONE FOR SOD 1) (Guan *et al*., 2013). During HS, hair grass, a species of grass that can withstand high temperatures, induced the release of cell wall binding protein Expansin 1 (AsEXP1), showing that EXP1 is involved in cell wall remodeling (Xu *et al*., 2007). In maize, it was discovered that sHSP26 protects chloroplasts from heat stress (Hu *et al*., 2015).

5. Heat Stress Responses and Epigenetic Modifications:

A comprehensive assessment of plant heat stress response signaling pathways was published (Ohama *et al*., 2017). At the DNA level, epigenetic modifications occur. In response to heat stress, epigenetic modifications, including DNA methylation, histone modifications, histone variations, short RNAs, long noncoding RNAs, and many unknown epigenetic processes, may upregulate to defend plants from destruction done by elevated temperature (Liu *et al*., 2015). These modifications are related to HSR gene expression.

The activation of methyltransferases has been examined extensively in reaction to HS. RNA-directed DNA methylation (RdDM) mechanism is necessary for basal thermotolerance, according to a study of heat resistance in DNA methylation-deficient mutants (Popova *et al*., 2013). Small RNAs can direct DNA methylation in plants (RNA-directed DNA methylation (RdDM)) by using two plant-specific RNA polymerases, PolIV and PolV. The rate of HS on transcription of major DNA methylation genes, DNA methyltransferase (MET1, CMT3, and DRM2), the biggest subunits of PoIIV (NRPD1), and PolV (NRPE1) was studied by Naydenov et al. (2015). They found that enhanced genome methylation in Arabidopsis under HS may be due to the activation of these epigenetic stimulators (Hu *et al*., 2015). DNA methylation to govern transposition on silencing, uneven gene expression, and sustained gene silencing in *Arabidopsis* is accomplished through three genetic mechanisms and accumulated at CG, CHG, and CHH sequences (Bucher *et al*., 2018; Gallusci *et al*., 2016).

Heat shock proteins are also thought to function in plant HS conditions. After a heat stress encounter, HSPs genes like HSP18, HSP22.0, APX2, and HSP70 will acquire histone H3 lysine 9 acetylation (H3K9Ac) and histone H3 lysine 4 trimethylation (H3K4me3). Histone modifications and DNA methylation through the RdDM process also contribute to plant heat tolerance (Lämke *et al*., 2016; Yang *et al*., 2018). In *A. thaliana*, HS has been demonstrated to be epigenetically mute genes. Popova *et al*. (2013) investigated a group of epigenetic mutants' heat tolerance. They discovered that the RNA-dependent DNA methylation pathway and the Rpd3-type histone deacetylase HDA6 are required for the transcriptional responses to HS. A histone variation H2A.Z has also been found to influence the transcription of stress-responsive genes following thermal treatment (Kim *et al*., 2015).

Stress memory is intimately linked to epigenetic alterations (Friedrich *et al*., 2019). Numerous studies have demonstrated stress treatment to cause alterations in the chromatin state of stress-responsive genes, which can last till restoration or in

progeny (Yang *et al*., 2017; Virlouvet *et al*., 2014). However, studies have revealed that, in contrast to primed memory throughout a plant's lifecycle, DNA sequence-independent epigenetic change can be passed down to the next generation, a phenomenon is known as "transgenerational memory" (Cong *et al*., 2019). This indicates that some epigenetic alterations are preserved and passed down as stress transgenerational memory to the following generation. This ensures plant flexibility and adaptability and a proper system between survival and reproduction (Molinier *et al*., 2006).

In *A. thaliana*, histone modification and HSFA2 are critical for HS memory. After a primed heat shock, the amount of H3K4 methylation (H3K4me2/3) was more significant for at least 2 days, which is linked to transcriptional memory (Lämke *et al*., 2016). H3K4 methylation deposition is required for HSR expression and transcriptional HS memory, and HSFA2 is required for this change. In *A. thaliana*, the HSFA2 and H3K27me3 demethylases RELATIVE OF EARLY FLOWERING 6 (REF6) form a positive cycle that transmits long-term epigenetic memory (Liu *et al*., 2019). Furthermore, it reported that the ONSEN retrotransposon is transcriptionally stimulated in plant response to HS. ONSEN transposition appears to be more common in the descendants of RdDM mutants exposed to HS, demonstrating the RdDM-mediated epigenetic alteration prevents retrotransposons from propagating transgenerational in plants (Ito *et al*., 2011; Ito *et al*., 2013).

6. Biotechnological and Molecular Tactics for Developing HS Tolerance in Plants:

6.1 Heat Shock Proteins (HSPs):

Heat stress causes the upregulation of many heat-inducible genes, generally alluded to as "heat shock genes" (HSGs), which frame heat shock proteins, and these bioactive proteins are critical for plant survival in catastrophic high temperatures (Chang *et al*., 2007). Some of these proteins, expressed persistently at high temperatures, preserve intracellular proteins from denatured proteins and maintain overall integrity and functionality through protein folding, acting as chaperones (Baniwal *et al*., 2004). Hsps, stress-induced proteins, are a family of proteins produced in response to stress. Based on their molecular weights, these are also categorized into five groups in plants: Hsp100, Hsp90, Hsp70, Hsp60, and tiny heat-shock proteins (sHsps). Plants have about 20 sHsps in general, and one plant species can have up to 40 different sHsps. The diversity of such proteins is a result of adaptability to thermotolerance. Hsp gene expression is regulated mainly by regulatory proteins termed Hsfs, which are inert in the cytoplasm. Plants have at least 21 Hsfs, each of which functions in regulation, but they also work together during all phases of heat stress reactions. The role is illustrated in the tomato plant, whereby HsfA1a is the central regulator essential for induced-stress gene expression, including synthesizing both HsfB1 and HsfA2, which are discovered after heat treatment induction. Some mutants are thermo-tolerance deficient but have regular HSP production (Hong *et al*., 2003).

Heat treatment in the context of retained heat shock elements (HSEs) in the coding region of HSGs, which stimulate transcription in reaction to heat, can induce HSP production. HSP70 detaches from cytoplasmic monomeric HSFs and penetrates the nucleus to build a trimer that can interact with HSEs if the plant detects heat stress (Lee *et al*., 1995). When the heat shock factor links toward other transcription elements, genetic variation occurs within minutes of an increase in temperature. Even though all HSGs have HSE structures, upregulation of the HSF gene simultaneously activated nearly all HSGs, providing protection from HS. Although this basic system is found in all eukaryotes, it is incredibly complex in plants.

Most HSFs are temperature inducible, implying that the precise HSF implicated in gene transcription may differ based on the period and amount of stress. Increased levels of plant HSFs may promote thermotolerance overall, but gene eliminations of particular HSFs already have a negligible impact on HT survivability. Heat upregulates the expression of several non-HSP genes in plants (Morrow and Tanguay, 2012). In Arabidopsis, HSFA1a and HSFA1b tend to govern the initial response of several genes to HT. At the same time, additional HSFs, possibly including heat-inducible HSFs, appear to be important in the formation of genes revealed subsequently. One HSF (HSFA1) has been proposed as the 'master regulator of the heat shock response in tomatoes. Usually, HSP synthesis is disrupted when this gene is inhibited, and the plant becomes very vulnerable to HTs (Mishra *et al*., 2002). Liming *et al*. (2008) discovered that persistently expressing HSP24 from *Trichederma harzianum* in *Saccharomyces cerevisiae* conferred much greater HS tolerance to plants. Today's genetic studies aim to enhance HT tolerance in important agricultural crops. Although HS resistance is a polygenic characteristic (regulated by multiple genes), distinct resistance components are essential at various growth stages or in various plant tissues; thus, it exhibits a spacio-temporal process and modulation (Bohnert *et al*., 2006).

6.2 Transgenic and Genetic Engineering Strategies:

HS can be minimized by applying diverse genetic engineering and transgenic techniques to generate crop plants with improved thermal resistance. Heat endurance was already improved by transcriptional activation of particular proteins. Other transgenic plants with varying degrees of thermal dissipation have been generated in light of research addressing the synthesis of sHSPs/chaperones and modulation of HSF gene regulation. Lee et al. (1995) managed to change the overexpression of HSPs) in *Arabidopsis thaliana* by modifying the transcription factor (AtHSF1) relevant for HSPs,

resulting in transgenic HT stress tolerance Arabidopsis. The signaling pathway was not active for thermal dissipation when the AtHSF1 gene was highly expressed. However, merging this gene with the N or C terminus of the gusA gene product (for β-glucuronidase production) resulted in a fusion protein that could trimerize itself and/or other HSFs in the lack of heating. Transforming this protein complex into *A. thaliana* resulted in transgenic plants with continuous HSP activity and improved thermo-tolerance without needing previous thermal treatment.

High quantities of the suitable component GB were artificially introduced into *Arabidopsis* by converting a bacterial choline oxidase gene designed to localize the protein to a chloroplast. It greatly improved germination percentage and seedling growth at increased temperatures (Hayashi *et al*., 1998). Thermo-tolerance is achieved by altering tobacco with the Rubisco activate gene, which allows for the reversible decarboxylation of Rubisco; this adaptive strategy aids in the protection of plants' photosynthetic equipment (Sharkey *et al*., 2001). Shi *et al*. (2001) discovered that *Arabidopsis* plants consistently encode the barley APX1 gene and had a moderate enhancement in heat tolerance. Plants were preserved from HS under intense light levels by doubling the stock of xanthophyll cycle precursors by overexpressing β-carotene hydroxylase (Davison *et al*., 2002). Grover *et al*. (2013) suggested that transgenic plants could be used to develop HT stress resistance by enhanced expression of HSP genes or modifying levels of HSFs that govern the expression of heat shock and non-heat shock genes, as well as upregulation of other trans-acting factors such as DREB2A, bZIP28, and WRKY proteins.

7. Conclusion:

Plants are subjected to various abiotic conditions, one of which is high temperature. HT has now become a real problem for crop productivity in the world since it has a significant influence not only on development but also on yield parameters of plants. Detailed research into the adaptive changes of plants to elevated heat has improved our knowledge of thermal response to stress. Several photosynthetic functions in chloroplasts, such as chlorophyll synthesis, photochemical reactions, electron transfer, as well as CO_2 absorption, are harmed by HT. The degree to whom this happens in particular climates, though, is dependent on the frequency and duration of HT and the daily scheduling of HT. Response of plants to HT varied amongst species and at different phases of development. Plants acquire various metabolic functions, and activities are triggered in HT circumstances. These alterations highlight the relevance of physiological and molecular research in elucidating the mechanisms that underpin stress reactions. Furthermore, producing stress-tolerant crops will require an insight into the nature of signal transduction and the individual genes resulting from exposure to HT. Therefore, most of the existing HT studies in various parts of the country are also confined to the laboratory and short-term investigations. Field trials examining various biochemical and molecular techniques and agronomic control measures are required to evaluate real HS reactions, including their impact on ultimate agricultural output.

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References:

Afrin, T., Diwan, D., Sahawneh, K., & Pajerowska-Mukhtar, K. (2020). Multilevel regulation of endoplasmic reticulum stress responses in plants: where old roads and new paths meet. Journal of Experimental Botany, 71(5), 1659-1667.

Ahuja, I., de Vos, R. C., Bones, A. M., & Hall, R. D. (2010). Plant molecular stress responses face climate change. *Trends in plant science*, 15(12), 664-674.

Allakhverdiev, S. I., Kreslavski, V. D., Klimov, V. V., Los, D. A., Carpentier, R., & Mohanty, P. (2008). Heat stress: an overview of molecular responses in photosynthesis. *Photosynthesis research*, 98(1), 541-550.

Arce, D., Spetale, F., Krsticevic, F., Cacchiarelli, P., Rivas, L., De, J., Ponce, S., Pratta, G. and Tapia, E. (2018). Regulatory motifs found in the small heat shock protein (sHSP) gene family in tomato. *BMC genomics*, 19(8): 1-7.

Asada, K. (1987). Production and scavenging of active oxygen in photosynthesis. *Photoinhibition*, 227-287.

Baker, N.R. & Rosenqvist, E. (2004). Applications of chlorophyll fluorescence can improve crop production strategies: an examination of future possibilities. *Journal of experimental botany*, 55(403): 1607-1621.

Baniwal, S.K., Bharti, K., Chan, K.Y., Fauth, M., Ganguli, A., Kotak, S., Mishra, S.K., Nover, L., Port, M., Scharf, K.D. & Tripp, J. (2004). Heat stress response in plants: a complex game with chaperones and more than twenty heat stress transcription factors. *Journal of biosciences*, 29(4): 471-487.

Baxter, A., Mittler, R. & Suzuki, N. (2014). ROS as key players in plant stress signaling. *Journal of experimental botany*, 65(5): 1229-1240.

Bharti, K., von Koskull-Döring, P., Bharti, S., Kumar, P., Tintschl-Körbitzer, A., Treuter, E. & Nover, L. (2004). Tomato heat stress transcription factor HsfB1 represents a novel type of general transcription coactivator with a histone-like motif interacting with the plant CREB binding protein ortholog HAC1. *The Plant Cell*, 16(6): 1521-1535.

Bohnert, H.J., Gong, Q., Li, P. & Ma, S. (2006). Unraveling abiotic stress tolerance mechanisms–getting genomics going. *Current opinion in plant biology*, 9(2): 180-188.

Braakman, I. &Hebert, D.N. (2013). Protein folding in the endoplasmic reticulum. *Cold Spring Harbor perspectives in biology*, 5(5): a013201.

Bucher, E., Kong, J., Teyssier, E., & Gallusci, P. (2018). Epigenetic regulations of fleshy fruit development and ripening and their potential applications to breeding strategies. *In Advances in botanical research* (Vol. 88, pp. 327-360). Academic Press.

Cao, Y., Duan, H., Yang, L., Wang, Z., Liu, L. &Yang, J. (2009). Effect of high temperature during heading and early grain filling on grain yield of indica rice cultivars differing in heat-tolerance and its physiological mechanism. *Acta Agronomica Sinica*, 35(3): 512-521.

Chang, H. C., Tang, Y. C., Hayer-Hartl, M., & Hartl, F. U. (2007). SnapShot: molecular chaperones, *Part I. Cell*, 128(1), 212-e1.

Chen, H., Hwang, J. E., Lim, C. J., Kim, D. Y., Lee, S. Y., &Lim, C. O. (2010). Arabidopsis DREB2C functions as a transcriptional activator of HsfA3 during the heat stress response. *Biochemical and Biophysical Research Communications*. 401(2): 238–244.

Chinnusamy, V., Zhu, J., Zhou, T., & Zhu, J. K. (2007). Small RNAs: big role in abiotic stress tolerance of plants. *In Advances in molecular breeding toward drought and salt tolerant crops* (pp. 223-260). Springer, Dordrecht.

Comastri, A., Janni, M., Simmonds, J., Uauy, C., Pignone, D., Nguyen, H.T. &Marmiroli, N. (2018). Heat in wheat: exploit reverse genetic techniques to discover new alleles within the Triticum durum sHsp26 family. *Frontiers in plant science*, 9: 1337.

Cong, W., Miao, Y., Xu, L., Zhang, Y., Yuan, C., Wang, J., Zhuang, T., Lin, X., Jiang, L., Wang, N. &Ma, J. (2019). Transgenerational memory of gene expression changes induced by heavy metal stress in rice (*Oryza sativa* L.). *BMC plant biology*, 19(1): 1-14.

Davison, P.A., Hunter, C.N. & Horton, P. (2002). Overexpression of β-carotene hydroxylase enhances stress tolerance in Arabidopsis. *Nature*, 418(6894): 203-206.

Deng, Y., Humbert, S., Liu, J.X., Srivastava, R., Rothstein, S.J. & Howell, S.H. (2011). Heat induces the splicing by IRE1 of a mRNA encoding a transcription factor involved in the unfolded protein response in Arabidopsis. *Proceedings of the National Academy of Sciences*, 108(17): 7247-7252.

Dogra, V. & Kim, C. (2019). Chloroplast protein homeostasis is coupled with retrograde signaling. *Plant signaling & behavior*, 14(11): 1656037.

Driedonks, N., Rieu, I. & Vriezen, W.H. (2016). Breeding for plant heat tolerance at vegetative and reproductive stages. *Plant reproduction*, 29(1): 67-79.

Edreira, J.I.R. & Otegui, M.E. (2012). Heat stress in temperate and tropical maize hybrids: Differences in crop growth, biomass partitioning and reserves use. *Field Crops Research*, 130: 87-98.

Fedyaeva, A.V., Stepanov, A.V., Lyubushkina, I.V., Pobezhimova, T.P. & Rikhvanov, E.G. (2014). Heat shock induces production of reactive oxygen species and increases inner mitochondrial membrane potential in winter wheat cells. *Biochemistry (Moscow)*, 79(11), pp.1202-1210.

Fragkostefanakis, S., Simm, S., Paul, P., Bublak, D., SCHARF, K.D. & Schleiff, E. (2015). Chaperone network composition in S olanum lycopersicum explored by transcriptome profiling and microarray meta-analysis. *Plant, cell & environment*, 38(4): 693-709.

Friedrich, T., Faivre, L., Bäurle, I. & Schubert, D. (2019). Chromatin-based mechanisms of temperature memory in plants. *Plant, Cell & Environment*, 42(3): 762-770.

Gallusci, P., Hodgman, C., Teyssier, E. & Seymour, G.B. (2016). DNA methylation and chromatin regulation during fleshy fruit development and ripening. *Frontiers in Plant Science*, 7: 807.

Grover, A., Mittal, D., Negi, M. & Lavania, D. (2013). Generating high temperature tolerant transgenic plants: achievements and challenges. *Plant Science*, 205: 38-47.

Guan, Q., Lu, X., Zeng, H., Zhang, Y. & Zhu, J. (2013). Heat stress induction of mi R 398 triggers a regulatory loop that is critical for thermotolerance in A rabidopsis. *The Plant Journal*, 74(5): 840-851.

Guihur, A., Fauvet, B., Finka, A., Quadroni, M. & Goloubinoff, P. (2021). Quantitative proteomic analysis to capture the role of heat-accumulated proteins in moss plant acquired thermotolerance. *Plant, Cell & Environment*, 44(7): 2117-2133.

Gunawardhana, M.D.M. & De Silva, C.S. (2011). Impact of temperature and water stress on growth yield and related biochemical parameters of okra. *Trop. Agric. Res*. 23: 77–83.

Hahn, A., Bublak, D., Schleiff, E. & Scharf, K.D. (2011). Crosstalk between Hsp90 and Hsp70 chaperones and heat stress transcription factors in tomato. *The Plant Cell*, 23(2), 741-755.

Halliwell, B. (2006). Oxidative stress and neurodegeneration: where are we now? *Journal of Neurochemistry*, 9(6):1634–1658.

Mirza, H., Hossain, M. A., & Fujita, M. (2010). Physiological and biochemical mechanisms of nitric oxide induced abiotic stress tolerance in plants. American Journal of Plant Physiology, 5(6), 295-324.

Hasanuzzaman, M., Hossain, M. A., & Fujita, M. (2012). Exogenous selenium pretreatment protects rapeseed seedlings from cadmium-induced oxidative stress by upregulating antioxidant defense and methylglyoxal detoxification systems. *Biological Trace Element Research*, 149(2), 248-261.

Hasanuzzaman, M., Nahar, K., & Fujita, M. (2013). Extreme temperature responses, oxidative stress and antioxidant defense in plants. *Abiotic stress-plant responses and applications in agriculture*, 13, 169-205.

Hayashi, H., Sakamoto, A. & Murata, N. (1998). Enhancement of the tolerance of Arabidopsis to high temperatures by genetic engineering of the synthesis of glycinebetaine. *The Plant Journal*, 16(2): 155-161.

Hong, S.W., Lee, U., Vierling, E. (2003). Arabidopsis hot mutants define multiple functions required for acclimation to high temperatures. *Plant Physiology*, 132: 757–767.

Hu, X., Wu, L., Zhao, F., Zhang, D., Li, N., Zhu, G., Li, C. & Wang, W. (2015). Phosphoproteomic analysis of the response of maize leaves to drought, heat and their combination stress. *Frontiers in plant science*, 6: 298.

Ikeda, M., Mitsuda, N. & Ohme-Takagi, M. (2011). Arabidopsis HsfB1 and HsfB2b act as repressors of the expression of heat-inducible Hsfs but positively regulate the acquired thermotolerance. *Plant physiology*, 157(3): 1243-1254.

Ito, H., Gaubert, H., Bucher, E., Mirouze, M., Vaillant, I. & Paszkowski, J. (2011). An siRNA pathway prevents transgenerational retro transposition in plants subjected to stress. *Nature*, 472(7341): 115-119.

Ito, H., Yoshida, T., Tsukahara, S. & Kawabe, A. (2013). Evolution of the ONSEN retrotransposon family activated upon heat stress in Brassicaceae. *Gene*, 518(2): 256-261.

Janka, E., Körner, O., Rosenqvist, E., & Ottosen, C.O. (2013). High temperature stress monitoring and detection using chlorophyll a fluorescence and infrared thermography in chrysanthemum (dendranthema grandiflora). *Plant Physiology and Biochemistry*, 67: 87–94.

Janská, A., Maršík, P., Zelenková, S. & Ovesná, J. (2010). Cold stress and acclimation–what is important for metabolic adjustment?. *Plant Biology*, 12(3): 395-405.

Kaur, H., Sirhindi, G., Bhardwaj, R., Alyemeni, M.N., Siddique, K.H. &Ahmad, P. (2018). 28-homobrassinolide regulates antioxidant enzyme activities and gene expression in response to salt-and temperature-induced oxidative stress in Brassica juncea. *Scientific Reports*, 8(1): 1-13.

Kim, J.M., Sasaki, T., Ueda, M., Sako, K. & Seki, M. (2015). Chromatin changes in response to drought, salinity, heat, and cold stresses in plants. *Frontiers in plant science*, 6, 114.

Lämke, J., Brzezinka, K., Altmann, S. & Bäurle, I. (2016). A hit-and-run heat shock factor governs sustained histone methylation and transcriptional stress memory. *The EMBO journal*, 35(2): 162-175.

Lee, J.H., Hübel, A. & Schöffl, F. (1995). Derepression of the activity of genetically engineered heat shock factor causes constitutive synthesis of heat shock proteins and increased thermotolerance in transgenic Arabidopsis. *The Plant Journal*, 8(4): 603-612.

Li, S., Fu, Q., Chen, L., Huang, W., & Yu, D. (2011). Arabidopsis thaliana WRKY25, WRKY26, and WRKY33 coordinate induction of plant thermotolerance. *Planta*, 233(6), 1237-1252.

Li, S., Zhou, X., Chen, L., Huang, W., & Yu, D. (2010). Functional characterization of Arabidopsis thaliana WRKY39 in heat stress. *Molecules and cells*, 29(5), 475-483.

Lim, P.O., Kim, H.J. and Gil Nam, H. (2007). Leaf senescence. *Annu. Rev. Plant Biol*., 58, pp.115-136.

Liming, Y., Qian, Y., Pigang, L., & Sen, L. (2008). Expression of the HSP24 gene from Trichoderma harzianum in Saccharomyces cerevisiae. *Journal of Thermal Biology*, 33(1), 1-6.

Lin, M. Y., Chai, K. H., Ko, S. S., Kuang, L. Y., Lur, H. S., & Charng, Y. Y. (2014). A positive feedback loop between HEAT SHOCK PROTEIN101 and HEAT STRESS-ASSOCIATED 32-KD PROTEIN modulates long-term

acquired thermotolerance illustrating diverse heat stress responses in rice varieties. *Plant physiology*, 164(4), 2045-2053..

Lippmann, R., Babben, S., Menger, A., Delker, C. & Quint, M. (2019). Development of wild and cultivated plants under global warming conditions. *Current Biology*, 29(24): R1326-R1338.

Liu, H.T., Gao, F., Li, G.L., Han, J.L., Liu, D.L., Sun, D.Y. & Zhou, R.G. (2008). The calmodulin-binding protein kinase 3 is part of heat-shock signal transduction in Arabidopsis thaliana. *The Plant Journal*, 55(5): 760-773.

Liu, J., Feng, L., Gu, X., Deng, X., Qiu, Q., Li, Q., & He, Z. (2019). An H3K27me3 demethylase-HSFA2 regulatory loop orchestrates transgenerational thermomemory in Arabidopsis. *Cell research*, 29(5), 379-390.

Liu, J., Feng, L., Li, J., & He, Z. (2015). Genetic and epigenetic control of plant heat responses. *Frontiers in plant science*, 6, 267.

Liu, J., Sun, N., Liu, M., Liu, J., Du, B., Wang, X., & Qi, X. (2013). An autoregulatory loop controlling Arabidopsis HsfA2 expression: role of heat shock-induced alternative splicing. *Plant physiology*, 162(1), 512-521.

Liu, J. X., & Howell, S. H. (2016). Managing the protein folding demands in the endoplasmic reticulum of plants. *New Phytologist*, 211(2), 418-428.

Locato, V., & De Gara, L. (2018). Programmed cell death in plants: an overview. *Plant Programmed Cell Death*, 1-8.

Lu, S. J., Yang, Z. T., Sun, L., Sun, L., Song, Z. T., & Liu, J. X. (2012). Conservation of IRE1-regulated bZIP74 mRNA unconventional splicing in rice (Oryza sativa L.) involved in ER stress responses. *Molecular Plant*, 5(2), 504-514.

Mathur, S., Agrawal, D., & Jajoo, A. (2014). Photosynthesis: response to high temperature stress. *Journal of Photochemistry and Photobiology B: Biology*, 137, 116-126.

McLoughlin, F., Kim, M., Marshall, R. S., Vierstra, R. D., & Vierling, E. (2019). HSP101 interacts with the proteasome and promotes the clearance of ubiquitylated protein aggregates. *Plant Physiology*, 180(4), 1829-1847.

Mishra, S. K., Tripp, J., Winkelhaus, S., Tschiersch, B., Theres, K., Nover, L., & Scharf, K. D. (2002). In the complex family of heat stress transcription factors, HsfA1 has a unique role as master regulator of thermotolerance in tomato. *Genes & development*, 16(12), 1555-1567.

Mittler, R., Finka, A., & Goloubinoff, P. (2012). How do plants feel the heat?. *Trends in biochemical sciences*, 37(3), 118-125.

Molinier, J., Ries, G., Zipfel, C. and Hohn, B., 2006. Transgeneration memory of stress in plants. *Nature*, 442(7106): 1046-1049.

Møller, I.M., Jensen, P.E. and Hansson, A., 2007. Oxidative modifications to cellular components in plants. *Annu. Rev. Plant Biol*., 58: 459-481.

Moreno, A. A., & Orellana, A. (2011). The physiological role of the unfolded protein response in plants. *Biological research*, 44(1), 75-80.

Morrow, G. & Tanguay, R.M. (2012). Small heat shock protein expression and functions during development. *The international journal of biochemistry & cell biology*, 44(10): 1613-1621.

Munns, R., & Tester, M. (2008). Mechanisms of salinity tolerance. *Ann. Rev. Plant Biol.* 59: 651–681.

Naydenov, M., Baev, V., Apostolova, E., Gospodinova, N., Sablok, G., Gozmanova, M., and Yahubyan G. (2015). High-temperature effect on genes engaged in DNA methylation and affected by DNA methylation in Arabidopsis. *Plant Physiology and Biochemistry*, 87: 102–108.

Nijabat, A., Bolton, A., Mahmood-ur-Rehman, M., Shah, A.I., Hussain, R., Naveed, N.H., Ali, A. &Simon, P. (2020). Cell membrane stability and relative cell injury in response to heat stress during early and late seedling stages of diverse carrot (*Daucus carota* L.) germplasm. *Hortscience*, 55(9): 1446-1452.

Nosaka, Y. & Nosaka, A.Y. (2017). Generation and detection of reactive oxygen species in photocatalysis. *Chemical reviews*, 117(17): 11302-11336.

Ohama, N., Sato, H., Shinozaki, K. &Yamaguchi-Shinozaki, K. (2017). Transcriptional regulatory network of plant heat stress response. *Trends in plant science*, 22(1): 53-65.

Pagamas, P. &Nawata, E. (2008). Sensitive stages of fruit and seed development of chili pepper (Capsicum annuum L. var. Shishito) exposed to high-temperature stress. *Scientia Horticulturae*, 117(1): 21-25.

Paradies, G., Ruggiero, F.M., Petrosillo, G. & Quagliariello, E. (1998). Peroxidative damage to cardiac mitochondria: cytochrome oxidase and cardiolipin alterations. *FEBS letters*, 424(3): 155-158.

Park, C.J. &Seo, Y.S. (2015). Heat shock proteins: a review of the molecular chaperones for plant immunity. *The plant pathology journal*, 31(4): 323.

Pastor-Cantizano, N., Ko, D.K., Angelos, E., Pu, Y. & Brandizzi, F. (2020). Functional diversification of ER stress responses in Arabidopsis. *Trends in biochemical sciences*, 45(2): 123-136.

Popova, O.V., Dinh, H.Q., Aufsatz, W. & Jonak, C. (2013). The RdDM pathway is required for basal heat tolerance in Arabidopsis. *Molecular Plant*, 6(2): 396-410.

Prasad, P.V.V., Staggenborg, S.A. & Ristic, Z. (2008). Impacts of drought and/or heat stress on physiological, developmental, growth, and yield processes of crop plants. Response of crops to limited water: *Understanding and modeling water stress effects on plant growth processes*, 1: 301-355.

Priya, M., Dhanker, O.P., Siddique, K.H., HanumanthaRao, B., Nair, R.M., Pandey, S., Singh, S., Varshney, R.K., Prasad, P.V. & Nayyar, H. (2019). Drought and heat stress-related proteins: an update about their functional relevance in imparting stress tolerance in agricultural crops. *Theoretical and Applied Genetics*, 132(6): 1607-1638.

Qi, J., Song, C.P., Wang, B., Zhou, J., Kangasjärvi, J., Zhu, J.K. & Gong, Z. (2018). Reactive oxygen species signaling and stomatal movement in plant responses to drought stress and pathogen attack. *Journal of integrative plant biology*, 60(9): 805-826.

Qu, A.L., Ding, Y.F., Jiang, Q. & Zhu, C. (2013). Molecular mechanisms of the plant heat stress response. *Biochemical and biophysical research communications*, 432(2): 203-207.

Ren, S., Ma, K., Lu, Z., Chen, G., Cui, J., Tong, P., Wang, L., Teng, N. & Jin, B. (2019). Transcriptomic and metabolomic analysis of the heat-stress response of Populus tomentosa Carr. *Forests*, 10(5): 383.

Rossi, S., Burgess, P., Jespersen, D. & Huang, B. (2017). Heat-induced leaf senescence associated with chlorophyll metabolism in bentgrass lines differing in heat tolerance. *Crop Science*, 57(S1): S-169.

Ruelland, E. & Zachowski, A. (2010). How plants sense temperature. *Environmental and experimental botany*, 69(3): 225-232.

Saidi, Y., Finka, A., Muriset, M., Bromberg, Z., Weiss, Y.G., Maathuis, F.J. & Goloubinoff, P. (2009). The heat shock response in moss plants is regulated by specific calcium-permeable channels in the plasma membrane. *The Plant Cell*, 21(9): 2829-2843.

Schramm, F., Larkindale, J., Kiehlmann, E., Ganguli, A., Englich, G., Vierling, E. &Von Koskull-Döring, P. (2008). A cascade of transcription factor DREB2A and heat stress transcription factor HsfA3 regulates the heat stress response of Arabidopsis. *The Plant Journal*, 53(2): 264-274.

Sharkey, T.D., Badger, M.R., von Caemmerer, S. & Andrews, T.J. (2001). Increased heat sensitivity of photosynthesis in tobacco plants with reduced Rubisco activase. *Photosynthesis research*, 67(1): 147-156.

Shi, W.M., Muramoto, Y., Ueda, A. & Takabe, T. (2001). Cloning of peroxisomal ascorbate peroxidase gene from barley and enhanced thermotolerance by overexpressing in Arabidopsis thaliana. *Gene*, 273(1): 23-27.

Shinozaki, K. &Yamaguchi-Shinozaki, K. (2007). Gene networks involved in drought stress response and tolerance. *Journal of experimental botany*, 58(2): 221-227.

Sita, K., Sehgal, A., HanumanthaRao, B., Nair, R.M., Vara Prasad, P.V., Kumar, S., Gaur, P.M., Farooq, M., Siddique, K.H., Varshney, R.K. & Nayyar, H. (2017). Food legumes and rising temperatures: effects, adaptive functional mechanisms specific to reproductive growth stage and strategies to improve heat tolerance. *Frontiers in Plant Science*, 8: 1658.

Stief, A., Brzezinka, K., Lämke, J. & Bäurle, I. (2014). Epigenetic responses to heat stress at different time scales and the involvement of small RNAs. *Plant signaling & behavior*, 9(10): e970430.

Suzuki, N., Sejima, H., Tam, R., Schlauch, K. & Mittler, R. (2011). Identification of the MBF1 heat-response regulon of Arabidopsis thaliana. *The Plant Journal*, 66(5): 844-851.

Taiz, L., Zeiger, E., Møller, I.M. & Murphy, A. (2015). Plant physiology and development (No. Ed. 6). *Sinauer Associates Incorporated*.

Takahashi, S., & Murata, N. (2008). How do environmental stresses accelerate photoinhibition? *Trends Plant Sci*. 13: 178–182.

Tang, T., Liu, P., Zheng, G. & Li, W. (2016). Two phases of response to long-term moderate heat: Variation in thermotolerance between Arabidopsis thaliana and its relative Arabis paniculata. *Phytochemistry*, 122: 81-90.

Valliyodan, B., & Nguyen, H.T. (2006). Understanding regulatory networks and engineering for enhanced drought tolerance in plants. *Curr. Opin. Plant Biol.* 9: 189–195.

Vanderauwera, S., Suzuki, N., Miller, G., Van De Cotte, B., Morsa, S., Ravanat, J.L., Hegie, A., Triantaphylidès, C., Shulaev, V., Van Montagu, M.C. &Van Breusegem, F. (2011). Extranuclear protection of chromosomal DNA from oxidative stress. *Proceedings of the National Academy of Sciences*, 108(4): 1711-1716.

Virlouvet, L., Ding, Y., Fujii, H., Avramova, Z. & Fromm, M. (2014). ABA signaling is necessary but not sufficient for RD 29 B transcriptional memory during successive dehydration stresses in A rabidopsis thaliana. *The Plant Journal*, 79(1): 150-161.

Wahid, A., Gelani, S., Ashraf, M. & Foolad, M.R. (2007). Heat tolerance in plants: an overview. *Environmental and experimental botany*, 61(3): 199-223.

Wang, Q.L., Chen, J.H., He, N.Y. & Guo, F.Q. (2018). Metabolic reprogramming in chloroplasts under heat stress in plants. *International journal of molecular sciences*, 19(3): 849.

Waraich, E.A., Ahmad, R., Halim, A. & Aziz, T. (2012). Alleviation of temperature stress by nutrient management in crop plants: a review. *Journal of soil science and plant nutrition*, 12(2): 221-244.

Wise, R.R., Olson, A.J., Schrader, S.M. &Sharkey, T.D. (2004). Electron transport is the functional limitation of photosynthesis in field-grown Pima cotton plants at high temperature. *Plant, Cell & Environment*, 27(6), 717-724.

Wu, A., Allu, A.D., Garapati, P., Siddiqui, H., Dortay, H., Zanor, M.I., Asensi-Fabado, M.A., Munne-Bosch, S., Antonio, C., Tohge, T. & Fernie, A.R. (2012). JUNGBRUNNEN1, a reactive oxygen species–responsive NAC transcription factor, regulates longevity in Arabidopsis. *The Plant Cell*, 24(2): 482-506.

Xalxo, R., Yadu, B., Chandra, J., Chandrakar, V. & Keshavkant, S. (2020). Alteration in carbohydrate metabolism modulates thermotolerance of plant under heat stress. *Heat Stress Tolerance in Plants: Physiological, Molecular and Genetic Perspectives*, 77-115.

Xu, J., Tian, J., Belanger, F.C. & Huang, B. (2007). Identification and characterization of an expansin gene AsEXP1 associated with heat tolerance in C3 Agrostis grass species. *Journal of experimental botany*, 58(13): 3789-3796.

Yadav, D.K. & Pospíšil, P. (2012). Role of chloride ion in hydroxyl radical production in photosystem II under heat stress: electron paramagnetic resonance spin-trapping study. *Journal of bioenergetics and biomembranes*, 44(3): 365-372.

Yang, C., Shen, W., Chen, H., Chu, L., Xu, Y., Zhou, X., Liu, C., Chen, C., Zeng, J., Liu, J. & Li, Q. (2018). Characterization and subcellular localization of histone deacetylases and their roles in response to abiotic stresses in soybean. *BMC plant biology*, 18(1): 1-13.

Yang, H., Berry, S., Olsson, T.S., Hartley, M., Howard, M. & Dean, C. (2017). Distinct phases of Polycomb silencing to hold epigenetic memory of cold in Arabidopsis. *Science*, 357(6356): 1142-1145.

Yoshida, T., Sakuma, Y., Todaka, D., Maruyama, K., Qin, F., Mizoi, J., Kidokoro, S., Fujita, Y., Shinozaki, K. &Yamaguchi-Shinozaki, K. (2008). Functional analysis of an Arabidopsis heat-shock transcription factor HsfA3 in the transcriptional cascade downstream of the DREB2A stress-regulatory system. *Biochemical and biophysical research communications*, 368(3): 515-521.

Źróbek-Sokolnik, A. (2012). Temperature stress and responses of plants. In Environmental adaptations and stress tolerance of plants in the era of climate change (113-134). *Springer*, New York, NY.

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