Plant Life Science: A Handbook of Plant Physiology

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CHAPTER-9

ABIOTIC STRESS PHYSIOLOGY: TEMPERATURE EXTREMES AND FLOODING

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Abstract

Abiotic stresses such as extreme temperatures and flooding are among the most significant environmental constraints limiting plant growth, development and productivity. Plants frequently encounter high or low temperature episodes and waterlogging events due to rapid climatic fluctuations. Their ability to perceive, respond, and adapt to these stresses determines their survival and yield potential. This chapter synthesizes current knowledge on the physiological, biochemical, molecular and genetic mechanisms underlying plant responses to temperature extremes and flooding. High temperature stress disrupts membrane integrity, protein stability, photosynthetic efficiency, reproductive processes and carbon assimilation. Plants counteract these effects through adaptive traits, such as protective leaf structures, osmotic adjustments, the accumulation of compatible solutes, antioxidant defence, and the synthesis of heat shock proteins (HSPs) and chaperones. Molecular studies highlight the central roles of HSFs, HSPs, antioxidants and ubiquitin-mediated protein turnover

in thermotolerance, with significant contributions from ABA, SA, ethylene, Ca^{2+}, ROS and lipid-based signalling pathways. Overall, this compilation provides a comprehensive overview of the physiological complexities and adaptive strategies plants use under temperature extremes and flooding.

Keywords: Temperature Stress, Flooding, Tolerance, Stress Signalling Pathways and Plant Resilience Mechanism.

Introduction

Stress is a mechanical concept and is defined as a force per unit area applied to an object. The object develops a strain in response to a specific stress. However, unlike mechanical systems, defining stress in biological contexts is more complex. Although the mechanical interpretation can be applied, its biological meaning differs significantly

Classification of Stress

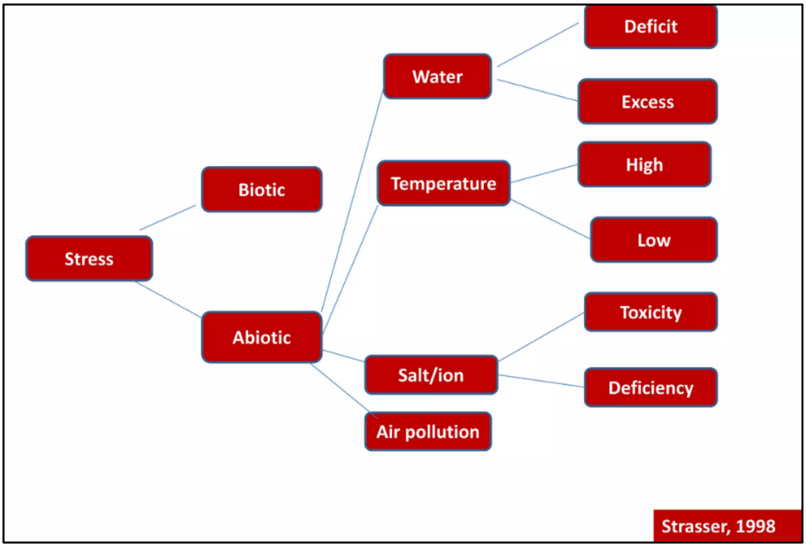


Fig. 1. Stress classification chart in plants

Any alteration in environmental factors that negatively impacts plant growth and development is considered a stress condition. In natural as well as agricultural ecosystems, plants are routinely subjected to various environmental stressors. In plant science, stress is typically described as any external factor that negatively affects plant growth or functioning. The idea of stress is closely linked with stress tolerance, which represents a plant's ability to withstand or adapt to adverse conditions (Kranner *et al.*, 2010).

Effects of stress

Plants respond to stress in different ways, and their reaction may be either elastic or plastic. An elastic response is temporary, allowing the plant to return to its original condition once the stress is removed. In contrast, a plastic response results in permanent changes, meaning the plant cannot fully recover. Stress effects may be immediately visible or plants may gradually develop tolerance when repeatedly exposed this process is known as hardening. In some cases, stress-induced changes can even persist across generations. For example, pea or bean plants exposed to low temperatures may become dwarf and this trait can continue for several generations. Recent research increasingly focuses on the genetic mechanisms underlying stress resistance, and breeders are working to develop varieties that are better adapted to varying climatic stresses. Overall, plant responses to stress are highly complex and involve a wide range of physiological adjustments (Kaviyazhagan and Prakash, 2023).

Temperature Stress

Suboptimal temperature stress refers to any decline in plant growth or damage at the metabolic, cellular, or tissue level that restricts the crop's inherent yield potential. It occurs when plants are exposed to temperatures either above or below the range required for optimal biochemical, physiological, or morphological processes (Greaves,1996).

- ✓ High Temperature Stress
- ✓ Low Temperature Stress

Levitt (1980) classified plants into

- ✓ Psychrophiles are those plants whose high temperature threshold is 15 to 20°C,
- ✓ Mesophiles are those plants whose high temperature threshold is 35 to 45°C, and
- ✓ Thermophiles are those plants whose high temperature threshold ranges from 45 to 100°C.

Effects of Heat Stress on Plants

- ✓ Seedling establishment is hampered
- ✓ Drying of leaf margins and scorching effect on leaves
- ✓ Reduction in plant growth
- ✓ Pollen development is affected
- ✓ Alteration in photosynthesis
- ✓ Total biomass is reduced
- ✓ Spikelet sterility
- ✓ Grain and fruit development and quality is affected

Mechanism of Tolerance (Adaptation)

1. Thick bark covering - Insulate covering
2. Reflective leaf hairs, Leaf rolling and orientation changes
3. Waxy leaf and Dimorphic leaf (White brittlebush)
4. High levels of Bound water & high protoplasmic viscosity
5. Accumulation of Polyamines
6. Synthesis of HSPs and LEA proteins

Mitigation of high temperature stress

- ✓ Plants need to be cultivated under shade conditions.
- ✓ Overhead irrigation to avoid sunburn.
- ✓ Application of Gibberellic Acid - Stimulate the α-Amylase production for seed germination.

- ✓ BAP (Benzyl Amino Purine) reduces the leaf senescence & Lipid peroxidation.
- ✓ Salicylic acid enhances the Thermo tolerance capacity.
- ✓ Glycine betaine reduced the leakage of ions.
- ✓ Application of Ethylene enhances the seed germination

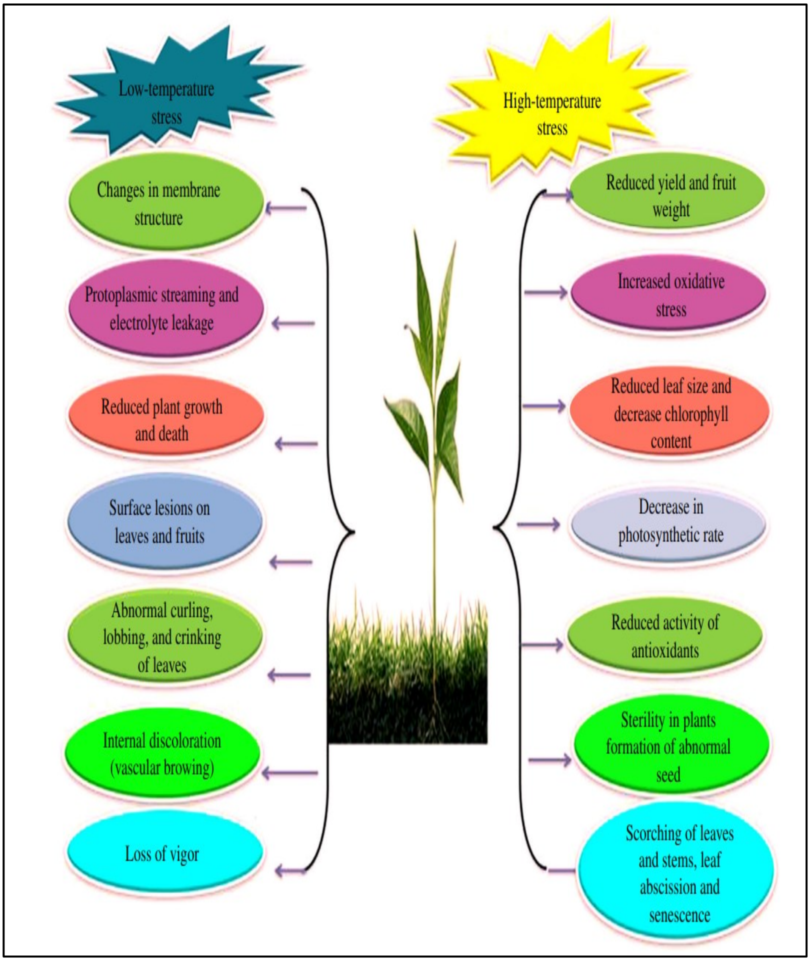


Fig. 2. Effects of temperature stress in plants (Adopted from: Tiwary *et al.*, 2020)

Plant response to temperature stress

Plants encounter high temperatures in multiple ways and their ability to adapt or acclimate to heat varies across time scales and organizational levels. Heat exposure may be chronic, as happens in consistently hot environments, or short-term and intense due to daily or seasonal temperature spikes. Different tissues, organs and developmental stages react differently depending on which cellular processes are most sensitive at the time of stress. Therefore, “heat stress” and “heat tolerance” are not single, uniform traits but encompass a complex set of disturbances affecting overall plant homeostasis. At the cellular level, elevated temperatures disrupt many structural and functional components. Heat increases membrane fluidity by altering lipid characteristics, which impairs membrane integrity and function. Since proteins operate within specific temperature ranges, high temperatures interfere with enzyme activity, disrupt metabolic pathways, and ultimately cause protein denaturation. These changes trigger the formation of active oxygen species (AOS) and when antioxidant defenses are insufficient, oxidative damage accumulates alongside direct thermal injury. Physiologically, heat-induced cellular damage manifests as reduced photosynthetic efficiency, poor assimilate transport and lowered carbon gain. Collectively, these effects lead to disrupted phenological patterns, reproductive failure and premature ageing. Because such responses involve many interconnected pathways, numerous genes contribute to heat tolerance.

In natural environments, heat rarely occurs in isolation. It frequently coincides with other stresses, particularly drought and high light intensity, resulting in overlapping injury patterns. Consequently, stress-response pathways for heat often intersect with those activated by other environmental stresses, sharing common signalling molecules and protective mechanisms (Malik, 2014).

This chapter outlines how elevated temperatures restrict plant growth and reproduction, followed by a detailed examination of molecular and genetic research on cellular responses to heat. Recent molecular studies have revealed key mechanisms involved in sensing, responding to, and acclimating to heat over short time periods. One of the best-known reactions to acute heat stress is the induction of heat shock proteins (HSPs), which act as molecular chaperones to maintain protein stability and proper folding (Boston *et al.*, 1996). However, HSPs represent only one facet of the plant's defence system, as multiple pathways collectively contribute to thermotolerance. Rapid advances in understanding heat-related signalling molecules have provided new insights into the sequence of events that occur as temperatures begin to rise.

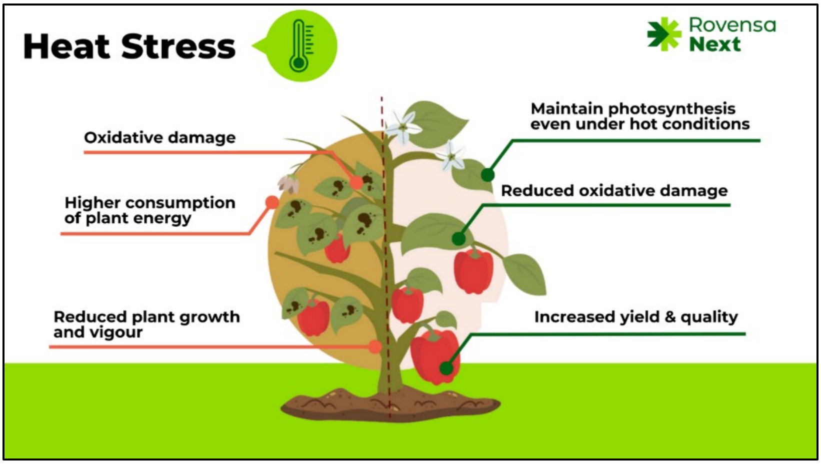


Fig. 3. How plants respond to the heat stress (Source: www.rovensanext.com)

Genomic, genetic, and transgenic approaches have also been instrumental in identifying the functions of individual genes and gene networks involved in heat responses. As these interacting pathways become more clearly defined, it will be increasingly feasible to decipher how heat stress intersects with other environmental stresses and to develop effective strategies for enhancing thermotolerance in crops. It is crucial to recognize that these cellular mechanisms operate within a highly complex organism composed of diverse cell and tissue types and progressing through multiple developmental stages. While deciphering cellular responses is challenging, the greater task lies in translating this knowledge into an understanding of how stress impacts agricultural productivity and the evolutionary fitness of plant species (Wang *et al.*, 2004).

Physiological responses to high temperature

High temperature limits to optimal plant performance

Plants are exposed to substantial temperature variation on both daily and seasonal scales, and as a result, they have evolved various mechanisms to maintain cellular functioning across a range of temperatures, although many of these mechanisms are not yet fully understood. The ideal temperature range for most plant species is relatively narrow, approximately 10 °C. Temperatures outside this window, even if not lethal, impose stress. For temperate species, the upper survival limit generally lies between 40 °C and 55°C depending on the duration of exposure (Klueva *et al.*, 2001), and these limits play a major role in determining where plants can successfully grow and how much land is suitable for agriculture. Plant sensitivity to heat varies widely based on the intensity, duration and developmental stage at which stress occurs, making it challenging to accurately quantify yield losses due to high temperatures. Interactions between plant water status and temperature further complicate assessments of heat versus drought effects under field conditions. In maize, for example, the best predictor of yield loss is how often daily maximum temperatures exceed 32 °C during pollination and grain filling. Likewise, soybean yield is highest during cooler reproductive periods, and cereal crops generally achieve optimal yields at temperatures between 20 °C and 30 °C.

Even moderately elevated temperatures, such as warm nights, can be harmful, causing increased respiratory carbon loss, reduced yields, disrupted flowering, and even inhibition of floral bud formation. These milder stresses differ from the extreme temperatures faced in arid and semiarid regions, where soil surfaces may reach 55 °C. In dense maize canopies in the U.S. Midwest, leaf temperatures can surpass 40 °C, and developing pea seeds in Idaho have been recorded at temperatures as high as 68 °C. Overall, it is evident that many plants experience heat stress at some point during their life cycle (Jenks and Hasegawa, 2005)

Heat sensitivity of photosynthesis

It is well established that heat stress reduces photosynthetic efficiency, and this decline occurs at temperatures far lower than those that are fatal to the plant. However, the exact mechanism behind this reduction remains debated. Current evidence does not clearly indicate whether the decrease in photosynthesis under heat stress results from a single dominant process or from multiple contributing factors. Many components of the photosynthetic machinery are sensitive to high temperatures, but identifying which component becomes limiting under heat stress has been challenging. Photosystem II (PSII) has long been considered the most heat-sensitive part of the photosynthetic system. Other potential points of vulnerability include the mechanisms that facilitate CO_2 movement from the intercellular spaces into the chloroplasts, the electron transport chain, and the capacity to regenerate ribulose bisphosphate (RuBP) (Wise *et al.*, 2004).

According to Sokolnik (2020), the instability of these photosynthetic components under heat may actually stem from the inability of cells to keep Rubisco (RuBP carboxylase/oxygenase) in its fully active form. During heat stress, Rubisco activity decreases not because the enzyme is denatured, but because “misfire” byproducts accumulate and block the active site. The high temperature sensitivity of Rubisco activase prevents it from efficiently removing these inhibitory compounds, thereby reducing Rubisco function under heat stress.

Heat sensitivity of reproduction

Beyond the long-term impacts of high temperatures on overall crop yield, many studies show that reproductive processes, particularly fertilization and seed filling, are far more heat-sensitive than vegetative growth. However, the exact reasons for reduced fertility under high temperatures are not tied to a single physiological failure; rather, they differ depending on the severity of heat stress and the plant species involved. Heat-induced sterility may arise from disruptions in male meiosis, impaired pollen germination, reduced pollen tube growth, or abnormalities in the megagametophyte. Additionally, high temperatures can negatively affect flower formation, grain set, endosperm development, photosynthetic supply, and the movement and allocation of assimilates, all of which ultimately influence seed number and weight. Despite extensive research, many heat-sensitive reproductive processes remain poorly understood across plant species. The most substantial molecular insights to date relate to the factors that restrict starch synthesis in wheat grains exposed to elevated temperatures.

Heat shock proteins/molecular chaperones

One of the most widely studied responses of plants and other organisms to high-temperature stress is the rapid induction of HSPs (Rasul *et al.*, 2017). When temperatures rise about 5-10 °C above the optimal level, HSP gene transcription is triggered within seconds. Transcript abundance typically peaks within 1-2 hours and then declines. Using sensitive antibodies, newly synthesized HSPs can be detected in less than an hour after the onset of stress. The amount of HSP mRNA and protein produced closely reflects the intensity of the heat stress and increases proportionally until temperatures approach lethal levels.

Five major families of HSPs have been identified across both plants and other organisms: Hsp100/ClpB, Hsp90, Hsp70/DnaK, Hsp60/GroE, and the small heat shock proteins (sHSPs). Among these, Hsp70 and Hsp60 are some of the most evolutionarily conserved proteins, highlighting their essential roles in cellular stress protection. All HSP classes function as molecular chaperones, a diverse group of proteins that interact with partially unfolded or unstable proteins. Chaperones assist with protein folding, membrane transport, regulation of protein activity, targeted degradation, and prevention of irreversible protein aggregation. This last role is especially important under heat stress and explains why HSPs are strongly induced at high temperatures (Singh *et al.*, 2019)

Table 1. Phenotypes of Plants with Mutations in Chaperones and Related Proteins

Mutant (species)	**Gene**	**Affected phenotype**	**Conditions tested (measurement)**	**Comments**
hot1 (Arabidopsis)	Hsp101	Reduced thermotolerance	2–4-day-old seedlings: 38°C for 90 min; 45°C (preconditioning-based): 20 min; 38°C 90 min + 45°C (post-stress)	No phenotype at normal temperatures
hot1 (Arabidopsis)	Hsp101	Reduced thermotolerance	5-day-old seedlings: 22°C → 38°C → 45°C	No phenotype at normal temperatures
Hsp90.1 mutant (Arabidopsis)	Hsp90.1	Reduced thermotolerance	Details not given	No change in phenotype; Hsp90.1 heat-induced

Hsp90.2 mutant (Arabidopsis)	Hsp90.2	Not tested	Not tested	Not tested
Hsp90.4 mutant (Arabidopsis)	Hsp90.4	Not tested	Not tested	Not tested
PasB1 mutant (Arabidopsis)	Hsp90 (cytosolic)	Accelerated cell damage by heat	5-week-old plants: 2 h at 45°C (photoperiodic conditions)	Details not given
BiP2 mutant (Arabidopsis)	BiP2	Not tested	Not tested	Not tested
hsp70 mutant (Arabidopsis)	Hsp70	Heat shock binding protein–interacting factor	2–3-day-old seedlings: 22°C → 31.5°C → 45°C	No effect (tested on cotyledons)
emp2 (Arabidopsis)	EMP2	Delayed seedling development; embryonic lethality possible	Not tested	Embryos die; overexpression of ERFS

Other components of the response to heat

Although the induction of HSPs plays a key role in heat tolerance, it is clear that many additional mechanisms contribute to surviving high-temperature stress. In several species, mutants lacking specific HSPs are still capable of developing thermotolerance. For example, in yeast, deletion of HSF1, the major regulator of HSP expression, does not prevent thermotolerance. Similarly, Drosophila mutants with significantly reduced HSP70 levels can still exhibit very high heat tolerance. Some mutants are sensitive to heat stress despite showing normal HSP induction, and in many organisms, chemical treatments that enhance thermotolerance do so without increasing HSP production. Together, these observations demonstrate that pathways other than HSP induction are also essential for acquiring thermotolerance (Aolymat *et al.*, 2023)

Antioxidant production

In plants, several genes unrelated to HSPs are also induced by heat stress. One example is the Arabidopsis cytosolic ascorbate peroxidase gene APX1, which is heat-responsive and contains a functional heat shock element (HSE) in its 5′ promoter region. This HSE is regulated by heat shock transcription factors (HSFs). Arabidopsis mutants lacking APX1 have been developed and shown to be sensitive to high-light stress, although their tolerance to high temperatures was not evaluated (Pnueli *et al.*, 2003). These knockout plants accumulate HSPs normally during heat stress, but not under light stress. Similarly, the barley APX1 gene, homologous to Arabidopsis APX3, which encodes a peroxisomal APX, is also induced by heat. Although the exact roles of the various APX isoforms under heat stress are not yet fully defined, it is well known that heat generates secondary oxidative stress in plants, and APX enzymes likely help mitigate this oxidative damage.

Heat stress also alters the activity of several other antioxidant-related enzymes. In *Agrostis palustris*, superoxide dismutase (SOD) activity increases during prolonged heat exposure, whereas APX, catalase, and glutathione reductase activities decrease (Du *et al.*, 2013). During the initial week of heat stress, however, APX and peroxidase activities rise while catalase activity declines, and SOD activation occurs more gradually. In maize, all of these antioxidant enzymes increase in activity under heat stress. Pretreating plants with chemicals that modify antioxidant capacity can also influence their ability to withstand subsequent heat stress. Notably, such treatments typically do not affect HSP production, indicating that enhanced thermotolerance through improved antioxidant defence operates independently of HSP induction. Recent microarray analyses of Arabidopsis APX-deficient plants show that certain HSPs can be expressed abnormally under non-heat stress conditions, even though HSP induction during heat stress remains normal. This suggests that, although the antioxidant and HSP pathways function separately in heat stress, some degree of crosstalk may occur between them (Timperio *et al.*, 2008).

Other heat-stress-regulated genes

Because high temperatures cause protein denaturation, it is reasonable to expect that the protein degradation system would also become more active during heat stress. Indeed, recent research in yeast shows that the buildup of misfolded or aggregated proteins is a major factor contributing to heat-induced toxicity. Enhanced cytoplasmic proteolysis can even compensate for the lack of HSP production. For example, Friant *et al.* (2003) found that overexpressing UB14 suppressed the thermosensitive phenotype of the *lcb1-100* mutant, which has impaired HSP synthesis. Increasing ubiquitin levels in these mutants restored thermotolerance, despite their low HSP content. This suggests that boosting protein degradation can reduce heat stress damage in yeast. In plants, several genes involved in the ubiquitin pathway are also upregulated during heat stress. Heat-tolerant soybean lines show faster accumulation of both free and conjugated ubiquitin compared to commercial varieties. In Arabidopsis, UBQ14 transcript levels rise in parallel with HSP70, whereas UBQ11 declines. Specific ubiquitin genes are increased in particular maize organs under heat stress, and a ubiquitin-conjugating enzyme is heat-induced in tomato. Heat-stressed tobacco plants also show increased expression of ubiquitin pathway genes. In Arabidopsis, the E3 ligase AtCHIP is induced by heat, but overexpression of this gene unexpectedly results in greater heat sensitivity. Small ubiquitin-like modifiers SUMO1 and SUMO2 accumulate to high levels in heat-stressed Arabidopsis, though to a lesser extent in HSP70-overexpressing plants, while SUMO3 does not respond similarly. These observations indicate that activation of the ubiquitin proteasome pathway likely supports the removal of denatured proteins during heat stress. Overall, the data suggest that while the ubiquitin system plays a role in plant heat responses, it may be less central than in yeast, possibly because plants rely more heavily on additional protective mechanisms. Nonetheless, there is likely coordination and crosstalk between ubiquitin-mediated degradation pathways and HSP regulatory networks (Abosheasha *et al.*, 2020).

Other heat-protective responses

Beyond heat-induced gene expression, plants also rely on various protective small molecules that help maintain protein and membrane stability during thermal stress. These “Compatible Solutes” are best understood in yeast, where the non-reducing disaccharide trehalose accumulates under high temperatures and protects cells from heat-induced mortality (Gu *et al.*, 2023). Although plants have the enzymes needed to synthesize trehalose, they do not appear to accumulate it to meaningful levels during heat stress. Overall, research on heat-induced metabolic shifts in plants is still in its early stages. Rizhsky *et al.* (2004) examined polar metabolite changes in Arabidopsis exposed to 38°C for six hours, with and without simultaneous drought stress. Under heat alone, only minor metabolite changes were detected, such as increases in lactitol, fucose, and melibiose. However, combined heat and drought stress caused major metabolic alterations not observed in drought alone, including a substantial rise in sucrose, and elevated melibiose, maltose, galactose and fucose. The large sucrose increase likely reflects major changes in metabolic regulation, while the significance of the other shifts, whether metabolic consequences or adaptive solute accumulation, remains unclear.

Importantly, heat stress suppressed the drought-induced accumulation of proline, and high proline levels appeared harmful to heat-stressed plants, suggesting that plants may actively inhibit proline buildup under high temperatures. Membrane stability is another critical factor in heat tolerance. The volatile hydrocarbon isoprene protects photosynthesis during short-term heat episodes in species that naturally produce it (e.g., red oak), and similar protection occurs when non-producing species like *Phaseolus vulgaris* receive external isoprene. Mutants and transgenic lines with altered lipid unsaturation levels also show differences in heat tolerance. Higher membrane lipid saturation correlates with enhanced heat resistance of photosynthesis (Murakami *et al.*, 2000) and bentgrass exhibits increased lipid saturation during the acquisition of thermotolerance. Additionally, increasing membrane fluidity can trigger some heat-responsive transcripts even without temperature elevation, indicating that heat-induced membrane changes interact closely with thermotolerance pathways. This raises the possibility that physical membrane properties play a direct role in rapid heat sensing, as membrane-perturbing compounds can influence heat-stress responses at both protein and physiological levels, including effects involving sHSPs (Niu and Xiang, 2018).

Table 2. Other Mutant Plants with Defects in Thermotolerance

Mutant	**Gene**	**Affect**
*Nah*G (Arabidopsis)	NahG Transgene: salicylate hydroxylase	Decreased heat tolerance
npr1 (Arabidopsis)	Involved in SA signalling in pathogenesis	Decreased heat tolerance, normal sHSP synthesis
abi1 (Arabidopsis)	Phosphatase involved in ABA signalling	Decreased heat tolerance
abi2 (Arabidopsis)	Phosphatase involved in ABA signalling	Decreased heat tolerance

abo- (Catharanthus roseus)	Constitutively produces ABA	Sensitive to heat, salinity and drought
etr1 (Arabidopsis)	Mutant in ethylene signalling	Somewhat decreased heat tolerance, especially in non-acclimated plants
fad7 (Arabidopsis)	Omega-3 desaturase, decreased trienoic fatty acids in chloroplasts	Increased heat tolerance of photosynthesis
fad7/8 (Arabidopsis)	Omega-3 desaturase, decreased trienoic fatty acids in chloroplasts	Increased heat tolerance of photosynthesis
fad3 (Arabidopsis)	Omega-3 desaturase, decreased trienoic fatty acids in ER	No change

Genetic variation in heat tolerance

Agricultural/horticultural plants

Although many studies have reported heritable variation in heat tolerance across crop and horticultural species, comparisons among them are difficult due to differences in species examined and the wide range of traits measured such as yield, leaf firing, pollen viability, or electrolyte leakage. Physiological assays have also shown cultivar-specific differences in acquired thermotolerance in barley, potato, rice, pearl millet and sorghum. However, these findings have not been followed by detailed quantitative genetic analyses, leaving uncertainty about the number and types of genes contributing to heat tolerance. Breeding for heat tolerance is challenging because temperature cannot be easily controlled in field conditions, leaving breeders dependent on natural weather patterns. Water availability further complicates the evaluation of heat tolerance unless trials are irrigated. Researchers have attempted to relate genotypic variation in heat tolerance to differences in HSP levels, isoforms, or timing of their induction. However, linking variation in a small set of genes to whole-plant heat tolerance is difficult and often unreliable. Most studies have found poor or inconsistent correlations between HSP expression profiles and thermotolerance, with only occasional weak associations observed (Klueva *et al.*, 2001).

For instance, a wheat line lacking a segment of chromosome 1 in one genome copy exhibited unusually high heat tolerance and could acquire thermotolerance at 48 °C following a milder preconditioning treatment than its parent line. This enhanced tolerance corresponded with HSP accumulation at temperatures 4 °C lower than those required for induction in the parent genotype. Another study showed that tissue-culture-derived variants of creeping bentgrass (*Agrostis stolonifera*) with differing thermotolerance levels displayed unique expression patterns of chloroplast-localised sHSPs. A well-studied example linking a heat-induced protein to stress tolerance comes from the work of Ristic and colleagues. They identified a chloroplast elongation factor isoform not classified as a traditional HSP but possessing chaperone-like activity that is associated with improved heat and drought tolerance. This finding highlights the possibility that maintaining chloroplast protein synthesis during heat stress is a key component of thermotolerance.

Natural variation in heat tolerance

Studies on naturally occurring variation in plant heat stress responses provides valuable support to forward and reverse genetic studies seeking to identify cellular mechanisms associated with improved thermotolerance. Although ecotypic differences in heat tolerance have been recognized for many years, the specific genes and pathways responsible remain largely unknown. Some recent studies have attempted to connect natural variation in HSP levels with differences in heat tolerance. For example, Barua *et al.* (2003) reported that the abundance of chloroplast-localised sHSPs correlated with variation in the thermotolerance of photosynthetic electron transport. Likewise, desert and coastal populations of *Encelia californica* showed distinct rates of sHSP accumulation and exhibited different sensitivities in photosynthetic electron transport under heat stress. As gene mapping in natural populations expands, new opportunities will emerge to better understand stress responses and the extent of their overlap. Integrating natural variation with quantitative trait locus (QTL) mapping is particularly well suited for dissecting complex, multigenic traits like heat tolerance, yet it remains underutilized. Applying this approach to crops such as rice or model species like Arabidopsis would help identify major-effect loci, determine how they vary under specific heat stress conditions, and enable the cloning of key genes.

In Arabidopsis, natural accessions vary in their capacity to grow at high temperatures and exhibit differences in enzyme kinetics depending on their native thermal environments. Recently, QTL mapping has been used to analyze stress tolerance during seed germination, including responses to high temperatures during imbibition. Notably, one QTL associated with heat tolerance overlapped with loci for salt resistance, ABA resistance, seed longevity, and germination speed, indicating that general seed vigour is linked to tolerance of heat and other stresses. Importantly, none of the detected QTL overlapped with Hsp101, a gene whose deletion causes extreme heat sensitivity during imbibition. This highlights the value of QTL mapping in uncovering previously unidentified loci that might be missed through mutant screens. Continued work of this kind is strongly justified (Chen *et al.*, 2021).

Flooding stress

Flooding may be defined as any situation of excess water. Sudden inundation following high rainfall events also poses a severe physiological stress on crops.

Types of floods

- **Flash flood -** short duration over a few weeks and not very deep

❖ **Deepwater flood -** deep flooding that lasts for a long time

Effects of flooding stress on plants

- Leaf rolling & curling
- Decay and death of leaves
- Wilting
- Abscission
- Epinasty
- Lenticels formation
- Nutrient deficiency & Toxicity: Under anaerobic condition, Fe toxicity is high.
- This leads to an increase in the polyphenol oxidase activity, leading to the production of oxidized polyphenols.
- It also causes leaf bronzing and reduced root oxidation power.

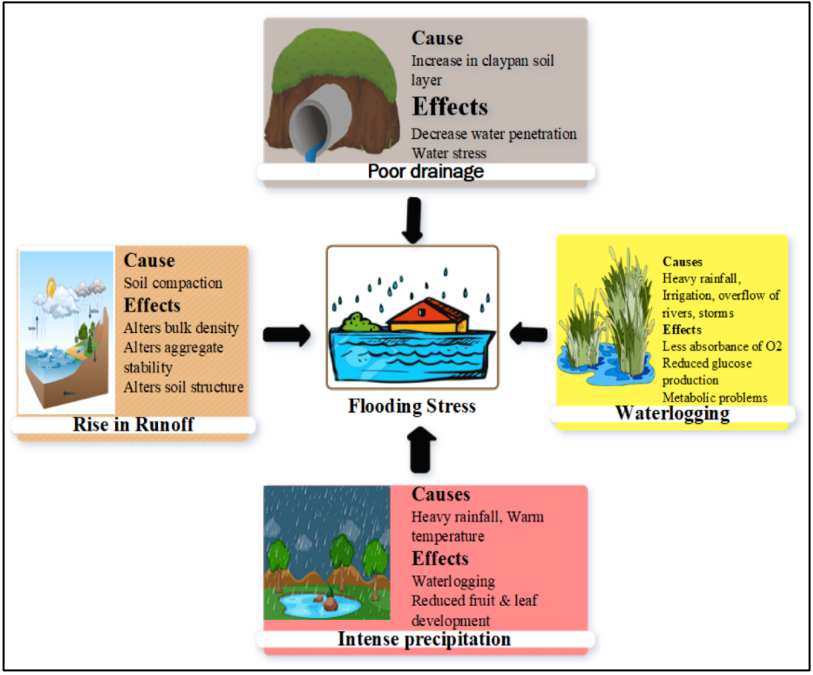


Fig. 4. Causes and effects of flooding (Adopted from Aslam *et al.*, 2023)

Adaptive mechanisms to floods

1. Thick root hypoderm (reduce oxygen depletion)
2. Aerenchyma (continuous intercellular spaces for O_2 move)
3. Lenticels (oxygen exchange)
4. Adventitious aerial roots from stem (Cucurbits)
5. Pneumatophores (superficial roots - negative geotropism)
6. Increased internode elongation by ethylene
7. Suberized & Lignified cells (Not allowing escape of O_2)
8. Roots get O_2 through enzymatic process
9. Production of Anaerobic Response Proteins (ANP's)
10. Increased glycolytic flow (Pasteur effect)

Flooding sensitive crops - Soybean, Tomato, Peas and Chillies

Flooding-tolerant crops - Rice, Wheat, Sorghum & Maize

Mitigation of flooding stress

- ✓ Providing adequate drainage to drain off excessive stagnant water around the root system.
- ✓ Spray of growth retardant of 500 ppm Cycocel for arresting apical dominance and thereby promoting the growth of laterals.
- ✓ Foliar spray of 2% DAP + 1% KCl (MOP).
- ✓ Spray of 0.5 ppm Brassinolide for increasing photosynthetic activity.
- ✓ Foliar spray of 100 ppm salicylic acid for increasing stem reserve utilization under high moisture stress.
- ✓ Foliar spray of 0.3 % Boric acid + 0.5 % ZnSO4 + 0.5 % FeSO4 + 1.0 % urea during critical stages of the stress.
- ✓ Balance the use of fertilizers (NPK or NPK + lime).
- ✓ Apply sufficient K fertilizer. Apply lime on acid soils, do not apply excessive amounts of organic matter (manure, straw) to soils containing large amounts of Fe and organic matter.

Flooding occurs naturally in many ecosystems, and numerous wild plant species have evolved excellent adaptations for surviving in waterlogged environments. In such conditions, maintaining effective gas exchange with the atmosphere is critical to avoid internal oxygen deficiency (hypoxia). To achieve this, many plants either constitutively develop or induce aerenchyma long, interconnected air-filled spaces that facilitate rapid internal gas movement across the plant body. This adaptation is often accompanied by modifications in root architecture to reduce the distance, and therefore the resistance, between aerial tissues and submerged root tips. The formation of adventitious roots, emerging from the stem or hypocotyl, further enhances this airway system by acting like a network of snorkels that extend into the anaerobic soil. Many species also produce a physical barrier that reduces oxygen leakage from the root into the surrounding low-oxygen soil, significantly improving tolerance to flooding (Abiko *et al.*, 2012).

A well-developed aerenchyma system is highly effective when shoots remain above water, as it enables air to be channelled down to the roots. However, during complete submergence, when shoots lose contact with the atmosphere, this system becomes far less effective. In such situations, some wetland plants attempt to re-establish aerial contact by rapidly elongating their leaves, internodes, or petioles an escape strategy used by certain rice varieties and other flood-adapted species. Alternatively, some plants adopt a quiescence strategy, suppressing metabolic activity to conserve energy until floodwaters recede. This approach is challenging because plants must minimize carbon and energy use while still maintaining the basic cellular functions required for survival (Voesene and Bailey-Serres, 2015).

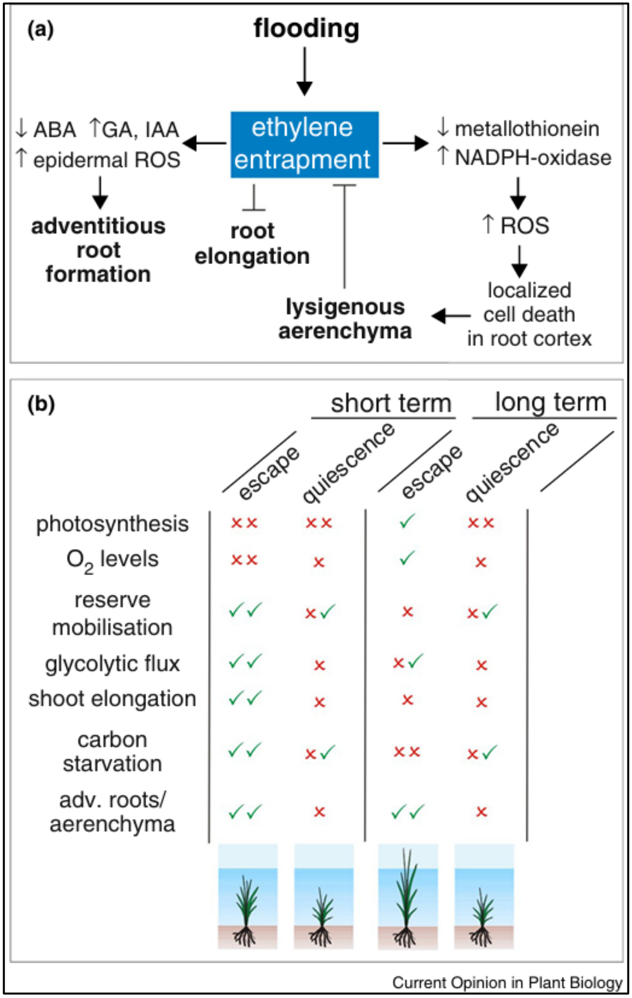
Plant response to flooding stress

The submerged plant: low oxygen and high ethylene

Ethylene is a gaseous hormone, it cannot easily diffuse out of plant tissues under flooded conditions, causing it to accumulate rapidly. This makes ethylene an immediate and reliable internal signal that plants use to recognize flooding. Oxygen availability also acts as a key signal. Internal concentrations of ethylene and oxygen reflect a balance between production, consumption, and diffusion. As a result, metabolically active tissues such as roots and meristems quickly experience oxygen depletion once flooding occurs. In photosynthetic tissues, oxygen levels depend on both light conditions and the balance between oxygen production and consumption (Voesenek and Sasidharan, 2013).

Ethylene serves as the primary regulator of most flood-adaptive responses. It triggers a hormonal cascade involving ABA, GA, and finally auxin to stimulate the formation of adventitious roots in species such as tomato, *Solanum dulcamara*, and rice. However, for these roots to emerge, ethylene must promote ROS production in epidermal cells, leading to programmed cell death that enables root penetration through the outer tissue layers. Similarly, ethylene-dependent reductions in antioxidant activity facilitate lysigenous aerenchyma formation, where ROS accumulation induces cell death in specific cortical cells. Interestingly, the critical suberin-based oxygen barrier in roots is not influenced by ethylene, though genes potentially involved in its formation have been identified (Shiono *et al.*, 2014).

Ethylene also regulates the escape response, in which plants elongate their shoots to reach the water surface. However, downstream signalling differs across plant groups: in rice, it operates through group VII ERF transcription factors, whereas in *Rumex palustris* it uses pathways typical of shade-induced elongation. Notably, ethylene pretreatment enhances anoxia tolerance in *Rumex palustris* by increasing hypoxia-responsive gene expression. This effect is absent in *Rumex acetosa*, a species that experiences fewer flood events and relies on a quiescence strategy rather than escape-driven shoot elongation. This highlights the importance of a link between ethylene and hypoxic signalling pathways (Van Veen *et al.*, 2014).



	short term escape	short term quiescence	long term escape	long term quiescence
photosynthesis	××	××	✓	××
O_2 levels	××	×	✓	×
reserve mobilisation	✓✓	×✓	×	×✓
glycolytic flux	✓✓	×	×✓	×
shoot elongation	✓✓	×	×	×
carbon starvation	✓✓	×✓	××	×✓
adv. roots/ aerenchyma	✓✓	×	✓✓	×

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Fig. 5. Role of ethylene in (a) root development and (b) during submergence and waterlogging

During flooding, the elevated ethylene levels typically inhibit root elongation. However, plants capable of forming aerenchyma can effectively vent excess ethylene, reducing its inhibitory effects. Species that are poor at producing aerenchyma therefore suffer strong reductions in root growth when submerged. The strong dose dependence of ethylene signalling may help explain why it plays such contrasting developmental roles during flooding. To avoid harmful effects caused by persistently high ethylene levels, some species that inhabit aquatic or flood-prone environments have partially or entirely lost their ability to produce, perceive, or respond to ethylene. Darkness common in turbid floodwaters accounts for a large share of the transcriptomic changes observed during complete submergence in Arabidopsis. This suggests that acclimation to flooding in dark conditions depends mainly on sugar and energy signalling, a pattern also seen in rice. Although hypoxia may contribute relatively little to gene regulation during dark submergence, variation in hypoxia-responsive gene expression correlates strongly either positively or negatively with flooding tolerance in natural populations of *Arabidopsis*, *Rumex* and *Rorippa*. This makes hypoxia-regulated signalling an important area for further study (Lee *et al.*, 2009).

Flooding Stress Signalling and Adaptation

Flooding is a complex, multifactorial stress that exposes plants to several challenges, with oxygen (O_2) deficiency being one of the most critical. Because the diffusion of O_2, CO_2, and other gases is far slower in water than in air, submerged plants receive only minimal oxygen, insufficient to sustain normal aerobic metabolism. Along with partial or complete oxygen deprivation (hypoxia or anoxia), rapid passive accumulation of ethylene occurs during submergence, serving as an early signal that triggers adaptive responses preparing the plant for impending low-oxygen conditions. Under hypoxia, photosynthetic activity declines due to reduced chlorophyll content and accelerated leaf senescence. This leads to decreased CO_2 assimilation, impaired nutrient uptake, and reduced stomatal conductance, ultimately causing lower biomass production and yield losses in most crop species. Through evolutionary adaptation, however, wetland plants possess far greater tolerance to submergence than terrestrial species, employing strategies such as low-oxygen quiescence syndrome (LOQS) and low-oxygen escape syndrome (LOES) (Nakamura and Noguchi, 2020). LOQS involves growth suppression to conserve energy, while LOES is characterized by rapid shoot elongation to reach oxygen-rich air above the water surface.

Flood-tolerant species like rice can also develop apoplastic barriers that prevent oxygen leakage from roots into the anaerobic soil. At the cellular level, fluctuating oxygen availability triggers extensive transcriptomic and metabolic reprogramming. The mitochondrial electron transport chain (mtETC) and oxidative phosphorylation become severely limited because oxygen is no longer available as the terminal electron acceptor. Consequently, energy production drops dramatically by approximately 65-97% forcing cells to depend primarily on anaerobic glycolysis. To maintain redox balance, NAD^+ regeneration shifts from the TCA cycle to fermentation pathways, with pyruvate being redirected accordingly (Hartman *et al.*, 2021).

Flooding signal proteins with roles in multiple stresses

A notable insight from abiotic stress research is that many signalling molecules, such as reactive oxygen species (ROS) and various regulatory proteins, function across multiple stress types. This overlap positions them as key mediators of plant responses to combined or sequential stresses. To better understand the regulatory mechanisms that operate during stress combinations involving flooding, it is useful to examine components of flooding-related signalling and summarize their additional roles in other abiotic stresses.

Because oxidative stress arises in many environmental stress conditions through elevated ROS production, ROS-associated proteins frequently play roles in multiple stress responses. For example, overexpressing UCP1 in Arabidopsis enhances tolerance not only to oxidative stress but also to salt, drought, osmotic stress and cold. These improvements, evidenced by increased biomass or larger seed size compared to the wild type, are attributed to UCP1's ability to stabilize N-degron pathway substrates and protect mitochondria from excessive ROS formation (Barreto *et al.*, 2022).

In tobacco and tomato, overexpressing Arabidopsis UCP1 enhances tolerance to heat, drought and salinity. Conversely, UCP1 and anac017 knockdown or knockout lines show heightened susceptibility to reductive stress (Fuchs *et al.*, 2022), and loss of ANAC017 also leads to drought sensitivity. Under methyl viologen-induced oxidative stress, ANAC013 knockdown plants exhibit greater reductions in fresh weight and primary root growth. Beyond hypoxia-responsive genes, ANAC013 regulates a set of genes that are differentially expressed during heat, salt, and osmotic stress, positioning ANAC013 as a potential early integrator of multiple stress signals. This role is further supported by the fact that ANAC013 itself is transcriptionally responsive to these stresses.

The NADPH oxidase RBOHD, another significant ROS generator under hypoxia alongside mitochondria, was initially linked to ROS bursts during plant defence. It is now recognized as a central component of rapid ROS-mediated systemic signaling triggered by diverse stresses, including waterlogging, salinity, high light, heat, cold and combinations such as heat plus light (Peláez-Vico *et al.*, 2023). Under saline conditions, RBOHD-derived ROS are essential for maintaining Na+/K+ balance and enabling NaCl-induced calcium influx processes that are disrupted in rbohD/rbohF double mutants and the rbohD single mutant under combined salinity and hypoxia.

The rbohD mutant also displays heat sensitivity and RBOHD expression is transcriptionally regulated by the ERF-VII factor RAP2.12 during flooding, drought and heat stress.

ERF-VII transcription factors are central regulators of hypoxia adaptation but also contribute to osmotic, salt and drought stress responses, the latter two sharing an osmotic component (Seok *et al.*, 2022). Although best known for oxygen sensing during flooding, the N-degron pathway also participates in sensing other abiotic stresses. Mutation of PRT6, a key N-degron component, improves salinity tolerance by maintaining constitutive stabilization of ERF-VIIs. Under salinity, reduced nitric oxide levels stabilize ERF-VIIs, potentially enhancing tolerance by mitigating osmotic stress through ABA-dependent and ABA-independent mechanisms (Lamichhane *et al.*, 2020). Additionally, the enhanced osmotic and salt stress tolerance observed in acbp1 mutants, ACBP1 being an interaction partner of RAP2.12 at the plasma membrane under normoxia, further links ERF-VII mediated signalling with broader abiotic stress adaptation.

Conclusion

Temperature extremes and flooding represent two of the most critical abiotic stresses limiting agricultural productivity under current and future climatic scenarios. Plants respond to these stresses through intricate physiological, biochemical and molecular mechanisms that ensure short-term acclimation and long-term adaptation. High temperatures damage proteins, membranes and photosynthetic machinery, yet plants counter these effects through HSP synthesis, antioxidant defence, osmotic regulation and alterations in membrane composition. Similarly, plant tolerance to low temperatures and subsequent chilling or freezing injury involves metabolic adjustments, kinase signalling, and stabilization of cellular structures.

Flooding stress disrupts oxygen availability, root respiration, nutrient uptake and hormonal balance. Ethylene-driven signalling, aerenchyma formation, adventitious rooting and fermentation pathways allow tolerant species to survive prolonged waterlogging. The contrasting quiescence and escape strategies among species reflect evolutionary adaptations to diverse hydrological environments. Furthermore, the interconnected roles of ROS, hormones, ERFVII and mitochondrial retrograde signalling highlight the complexity and overlap among stress-response networks.

Advancements in genomics, proteomics, and metabolomics have enabled better understanding of these stress pathways and the identification of key regulatory genes useful for breeding and biotechnological interventions. However, the multigenic nature of stress tolerance and strong environmental interactions continue to challenge crop improvement efforts. Future research must integrate molecular breeding, crop modelling, stress-forecasting tools, and climate-smart agronomic practices to enhance resilience. Overall, strengthening plant tolerance to temperature extremes and flooding is essential for sustaining global food security in an era of rapid climatic change.

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