

Physiological Adaptations and Responses Of Maize (*Zea Maize*) To Heat Stress

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1.ABSTRACT

The maize crop is crucial to food production and agriculture. It provides millions of people with a basic food source, promoting food security and reducing hunger. Maize is also an important feed grain for animals, aiding the cattle sector. Its cultivation boosts rural development, provides farmers with cash, and opens up job possibilities. A major global concern for the cultivation of maize is heat stress. During crucial growth stages, maize is extremely susceptible to high temperatures, which results in lower crop yields & crop losses. Heat stress hinders plant growth and development in general, hinders pollination, lowers grain filling, and has an impact on the photosynthetic process. The effect of heat stress upon maize productivity is intensifying as a result of climate change's rising temperatures. Plant breeders must fully comprehend the physiological reactions and adaptive mechanisms in order to create heat-resistant cultivars. Increased heat stress tolerance in maize plants is mostly a result of physiological changes. Maize plants have developed special anatomical and biochemical systems that allow them to efficiently absorb and use carbon dioxide (CO₂) even in high temperature.

Keywords: physiological adaptations, Heat stress response, photosynthesis, heat stress, unfolded protein responses, maize

2.PHYSIOLOGICAL ADAPTATIONS

C₄ plants must develop physiological adaptations in order to tolerate heat stress and flourish in hot climates. These adaptations encompass a number of systems that allow C₄ plants to continue to function normally even in high temperatures, including photosynthesis and water control. One crucial adaptation that aids C₄ plants in optimizing water consumption and reducing water loss is stomatal control (Flexas et al., 2004). Another significant adaptation shown in C₄ plants when exposed to heat stress is heat shock protein accumulation, which protects proteins and aids in their stability (Kotak & Scharf, 2007). Furthermore, C₄ plants have a powerful antioxidant defense mechanism that helps them scavenge reactive oxygen species and lessen oxidative damage brought on by heat stress. (Mittler et al., 2012).

2.1 Biochemical Adaptations

Compared to Rubisco in C₃ plants, phosphoenolpyruvate carboxylase (PEPc) present in C₄ plants tends to have a greater affinity for CO₂ and is less susceptible to heat denaturation. (Sage, 2002). Effective CO₂ fixation is made possible by this biochemical trait, which also lessens the damaging effects of heat stress upon photosynthesis. PPDK (Pyruvate orthophosphate dikinase), an enzyme found in C₄ plants, is more thermally stable than pyruvate kinase found in C₃ plants. (Gowik & Westhoff, 2011). Under heat stress, it has been discovered that C₄ plants accumulate more Heat Shock Proteins (HSPs) (Kotak et al., 2007). HSPs are essential for preventing protein denaturation and helping in their refolding, which helps C₄ plants tolerate heat.

2.2 Structural Adaptations

Kranz anatomy, which has characteristics defined by concentric layers of mesophylls and (BSC) bundle sheath cells, is seen in C4 plants. This anatomical configuration improves carbon absorption in hot environments by lowering photorespiration and allowing efficient CO₂ concentration. (Majeran & van Wijk, 2009). C4 plants have a low carbon dioxide permeability, which reduces CO₂ leakage and maximizes CO₂ availability in the Calvin cycle. (von Caemmerer & Furbank, 2003). According to von

protein responses (UPR), that is located in the endoplasmic reticulum(ER) and activates transcriptional factors which stimulate a distinct family of the stress response genes(Li et.al.,2021).

3.1 Photosynthetic Equipment

One of the plant processes most vulnerable to heat stress is photosynthesis (Sinsawat et al. 2004).

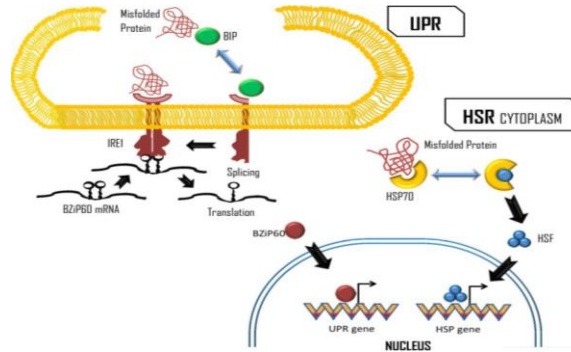


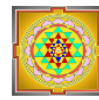
Figure 1 Plant responses to heat stress are mediated by two mechanisms. Heat stress responses (HSRs), a group of defensive mechanisms, are triggered by heat stress. Plants are protected from further stress by unfolded protein responses (UPR) within the ER and heat stress responses (HSR) in the cytoplasm.

Caemmerer and Furbank (2003).

3. PHYSIOLOGICAL RESPONSES OF MAIZE TO HEAT STRESS.

Plants will experience more frequent high-temperature episodes as the average global temperature is predicted to rise (ICPP report)(Hoegh-Guldberg et.al., 2018). The rate at which photosynthesis occurs was dramatically lowered within plants undergoing heat stress because of significantly lower rate of stomatal conductance. Additionally, excessive heat results in reduction of net photosynthesis, seed weight, biomass accumulation and leaf area (Meena H et.al.,2016). In order to reduce damage and defend themselves from more stress, plants tend to have developed a number of response to heat stresses. Heat stress triggers the cytoplasmic heat stress responses (HSR), within which group of genes producing heat shock proteins become activated by the heat shock transcription factors (HSFs). Additionally, heat stress causes the unfolded

Chloroplast damage can result in a permanent or temporary loss in chlorophyll accumulation and photosynthetic efficiency due to stress of high temperature on functional structure and structural behavior(Cui et al. 2006). According to several investigations, plants under high-temperature stress exhibit decreased chlorophyll (Chl) production(B. Efeoglu et.al.,2009). Under high-temperature conditions, “5-aminolevulinatase (ALAD)” very first discovered enzyme of the pyrol biosynthesis pathway, had shown being less active(S. Mohanty et.al.,2006). One of the parts of the photosynthetic machinery that is thought to be particularly sensitive to temperature is photosystem II (PSII) (A.Srivastava et.al.,1997). According to reports, high temperatures initially cause the PSII reaction centres, waters oxidizing complex(WOC) and light harvesting complex to malfunction (Salvucci et.al.,2001). One of the key factors in the control of photosynthetic processes is the plastoquinone (PQ) pool's redox state (A. Murakami et.al.,2996). As a result of the high temperature stress, a decrease in PQ pool is shown. In



green plants, the NADPH dehydrogenase complex as well as the (FNR) ferredoxin-palatoquinon oredoreducate recycles the electron that comes from exposed-stroma site of the PSI to PQ-pool(M. Havaux et.al.,2005).

3.2 Cytoplasmic Heat Stress Response

Heat stress responses (HSRs), a group of defensive mechanisms, are triggered by heat stress. HSRs are homeostatic systems that help plants recover from heat stress and guard against further stress. Transcriptional factors include heat shock factors(HSFs) that serve as essential for HSRs because they promote the production of clusters of heat shock proteins(HSPs) (Li et.al., 2021). The transcriptional, translational, & post-translational levels are only a few of the levels where HSF activity is controlled(Guo et.al., 2016). Class A1 HSFs are kept in the cytoplasm during non-stress conditions as a result of their connection with HSP90/70 and related cochaperones. Misfolded proteins attract chaperones far from HSFs in responses to the heat stress (Shi et.al.,1998) and trimerized, released HSFs then get imported into the cell nucleus. (Sistonen et.al.,1994). According to Guo et al. (2008), HSF trimerization makes it easier for HSFs to bind to and trigger the transcription of HSP gene promoters' heat shock response elements (HSEs).

3.3 Unfolded Protein Responses (UPRs)

Despite the HSRs, heat stress additionally triggers another reaction in the ER (endoplasmic reticulum) known to be as unfolded protein response(UPR), which works in mizimizing effects of heat stress and defend plants from additional stresses (Li et al., 2021). The UPR serves as a stress defense mechanism that has been preserved throughout evolution. Misfolded proteins are what cause both the HSRs and UPR to be activated that build within ER and cytoplasm, respectively, although they take place in separate cellular compartments (Figure 1). Plants' UPR genes are activated when stress-transducing transcription factors are present (Howell and S.H., 2013) across two UPR signaling pathway branches. One of the branches includes the enzyme IRE1, riboneuclase and dual proteins kinase, that, when activated, splicing basic leucine zipper (bZIP) in 60 mRNAs. Two of ER membrane-anchored transcriptional factors, bZIP28 and bZIP17, mediate the other branch(Howell and S.H,2013). Both branches react and assist in providing protection against the heat stress. For this instance,

bZIP60s increases levels of the expressions of type of HSFs gene called "Hsftf13" that in turn increases expressions of a collection of HSPs genes (Li et al., 2020).

3.4. Other Transcriptional Regulation Network involved in Heat Stress Responses of Plants

Other signaling and response routes, such as pretreatment with the SA (salicylic acid), ABA(abcisic acid) ACC (1-aminocyclopropane-1-carboxylic acid, an ethylene precursor), and H₂O₂, might cause plants to develop thermotolerance. Mutants in these chemicals' biosynthesis or response pathways exhibit defects in basal thermotolerance (Larkindale et.al.,2005). It is thought that ABA contributes to acquired or heat tolerance generated by heat acclimatization. Heat sensitivity is seen in ABA-deficient and -insensitive mutants, but AREB/ABF-OE plants exhibit improved thermotolerance(Larkindale et.al.,2005). Throughout directly binding of FaAREB3 or FaDREB2c to ABR cis-elements in the FaHSFA2c promoter, the overexpression of HSPs and HSFA2c by ABA increased the heat tolerance in Arabidopsis and tall fescue (Wang et.al., 2017).

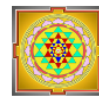
4. CONCLUSION

When a Plant standing in the field faces heat stress certain natural adaptations and responses occur as a result. PEP enzyme in Maize plant has a higher tolerance to getting denatured in high temperatures and is found working efficiently. Concentric layers of mesophylls cells and (BSC)bundle sheath cells prevent leakage of CO₂ and protect Rubisco from high temperature stress. HSFs also play avital role in preventing damage to plant by heat stress. UPR is another response which is activated by heat stress and protects the plant from further stress conditions. Absisic acid is also said to improve plants tolerance against acquired heat stress.

5.CONFLICT OF INTEREST

The Authors have no conflict of interest.

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