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Plant Response to Heat Stress

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Abstract—*The present rate of emission of greenhouse gases from different sources is responsible for a gradual increase in the world's ambient temperature and is resulting in global warming. Heat stress is a complex function of intensity (temperature in degrees), duration and the rate of increase in temperature. Heat stress due to high ambient temperature is a serious threat to crop production worldwide. At moderately high temperatures direct injuries include protein degradation, aggregation and increased fluidity of membrane lipids. Indirect injuries include inactivation of enzymes, inhibition of protein synthesis, protein degradation and loss of membrane integrity. Under high temperature conditions, plants accumulate different metabolites such as antioxidants, osmoprotectants, heat shock proteins and different metabolic pathways and processes are activated. A variety of osmolytes such as sugars, sugar alcohols and proline are accumulated. Heat stress alters the tertiary and quaternary structures of membrane proteins, enhance the permeability of membranes and increased loss of electrolytes. The generation and reactions of activated oxygen species cause the autocatalytic peroxidation of membrane lipids and pigments leading to the loss of membrane semi permeability and modifying its function. Heat-shock proteins/chaperones are responsible for protein folding, assembly, translocation and degradation in many normal cellular processes and stabilize proteins and membranes.*

Keywords*: Heatstress, Temperature, Heat shock protein, Antioxidants, Osmoprotectants, Chaperones.*

1. INTRODUCTION

Global warming is predicted to have a general negative effect on plant growth due to the damaging effect of high temperatures on plant development. The increasing threat of climate extremes including very high temperatures might lead to catastrophic loss of crop productivity and result in widespread famine. Heat stress is a complex function of intensity (temperature in degrees), duration and rate of increase in temperature. Heat stress due to high ambient temperature is a serious threat to crop production worldwide (Hall, 2001). Heat stress threshold is a value of daily mean temperature at which a detectable reduction in growth begins / the temperature at which growth and development of plant cease. Upper threshold is the temperature above which growth and development cease. Lower threshold (base temperature) is the temperature below which plant growth and development stop. At very high temperatures, severe cellular injury and even cell death may occur within minutes. At moderately high temperatures the direct injuries include protein degradation, aggregation and increased fluidity of membrane lipids while indirect injuries include inactivation of enzymes, inhibition of protein synthesis, protein degradation and loss of membrane integrity.

2. MORPHO-ANATOMICAL AND PHENOLOGICAL RESPONSES OF PLANTS TO HEAT STRESS:

Morphological symptoms includesunburns on leaves branches and stems, leaf senescence and abscission, shoot and root growth inhibition, fruit discoloration and damage and reduced yield etc. The reproductive phase isconsidered as the most sensitive to high temperature.The anatomical changes includereduced cell size, closure of stomata and curtailed water loss, increased stomatal density, damaged to mesophyll cells, and increased permeability of plasma membrane. High temperatures reduced photosynthesis by changing the structural organization of thylakoids (Karim *et al.,* 1997). Photochemical reactions in thylakoids lamellae and carbon metabolism in the stroma of chloroplast have been suggested as the primary sites of injury at high temperatures (Wise *et al*., 2004).Increasing leaf temperatures and photosynthetic c photon flux density influence thermo tolerance adjustments of PSII. High temperature alters the energy distribution and changes the activities of carbon metabolism enzymes, particularly the rubisco, thereby altering the rate of RUBP regeneration by the disruption of electron transport and inactivation of the oxygen evolving enzymes of PSII (Salvucci and Crafs-Brandner, 2004).High-temperature stress induces production of phenolic compounds such as flavonoids and phenylpropanoids Rivero*et. al.*2001). (Phenylalanine ammonia-lyase (PAL) is the principal enzyme of the phenylpropanoid pathway. Increased activity of PAL in response to thermal stress is considered as the main acclimatory response of cells to heat stress. The integrity and functions of biological membranes are sensitive to high temperature. Heat stress alters the tertiary and quaternary

structures of membrane proteins and enhances the permeability of membranes, increased loss of electrolytes.

3. MOLECULAR RESPONSES OF PLANTS TO HEAT STRESS

Heat stress may induce oxidative stress. There is generation of activated oxygen species (AOS) like, singlet oxygen ($^1O_2$), super oxide radical (O_2^-), hydrogen peroxide (H_2O_2), hydroxyl radical (OH^-). AOS cause autocatalytic per oxidation of membrane lipids and pigments leading to the loss of membrane semi permeability and modifying its function. The scavenging of O_2 by superoxide dismutase (SOD) results in the production of (H_2O_2), which is removed by APX (ascorbate peroxidase) or CAT (catalase). The Protection against oxidative stress is an important component in determining the survival of a plant under heat stress.

4. THE HEAT STRESS TOLERANCE MECHANISM IN PLANTS

The first response to heat stress such as plasma membrane fluidity disruption and osmotic changes triggers downstream signaling and transcriptional cascade activating a stress responsive pathway leading to reestablishment of cellular homeostasis and separation of damaged proteins and membranes (Fig.1). Initial effects are on the plasma membrane which becomes more fluid under stress triggering calcium influx and cytoskeleton reorganization, resulting in the up regulation of some mitogen activated and calcium dependent kinases. Signaling of these cascades at nuclear level leads to antioxidant production and compatible solute accumulation. Membrane fluidity changes also lead to ROS generation in organelles and signaling in cytoplasm. Thermo tolerance acquirement is correlated with the activities of CAT and SOD, higher ascorbic acid control and less oxidative damage (Tyagi, 2004). Inadequate or delayed response in any step may lead to cell death (Vinocur and Altman, 2005).

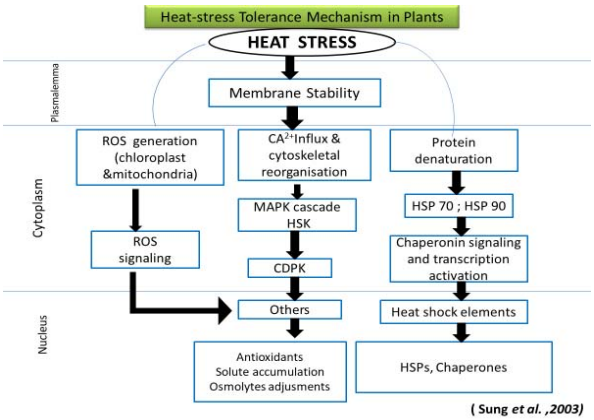

Fig. 1: Heat stress tolerance mechanism in plants. {MAPK, mitogen activated protein kinases; ROS, reactive oxygen species; HAMK, heat shock activated MAPK; HSE, heat shock element; HSPs, heat shock proteins; CDPK, calcium dependent protein kinase; HSK, histidine kinase}

5. ROLE OF HEAT-SHOCK PROTEINS AND A CHAPERONE IN PROTEIN FOLDING:

Induction of HSP and other proteins such as LEA and dehydrins interact with other stress response mechanism such as production of osmolytes and antioxidants. HSPs are involved in stress signal transduction and gene activation as well as in the regulation of cellular redox state. Heat shock proteins belong to a larger group of molecules called chaperons. Heatshock proteins (Hsps)/chaperones are responsible for protein folding, assembly, translocation and degradation in many normal cellular processes, stabilize proteins and membranes, and can assist in protein refolding under stress conditions. They can play a crucial role in protecting plants against stress by reestablishing normal protein conformation and thus cellular homeostasis. Most of the stress proteins are soluble in water and therefore contribute to stress tolerance presumably via hydration of cellular structures. Hsp synthesis is tightly regulated at the transcriptional level by heat shock factors (HSFs). HSF-1 is main regulator of the short-term induction of Hsp (Kim and Schoffl 2002).Five major families of Hsps/chaperonesrecognized are given below-

Protein class--	Size (kDa)	Locaton	Functions
HSP100	100-114	Cytoplasm	Disaggregation, unfolding.
HSP90	80-94	cytoplasm, ER	Facilitating maturation of signaling molecule, genetic buffering
HSP70	69-71	Cytoplasm, ER, mitochondria	Prevent aggregation, assisting refolding, protein import and translocation, signal transduction and transcriptional activation
HSP60	10-60	Chloroplast, mitochondria	Folding and assisting refolding.
smHSP	15-30	Cytoplasm, ER, mitochondria, Chloroplast	Preventaggregation, stabilize non- native proteins.

Besides HSPs, there are several other plant proteins, including ubiquitin, cytosolic SOD and Mn-Peroxidase. Heat stability is a notable feature of LEA proteins, i.e. they do not coagulate upon boiling (Thomashow, 1999). Group 1 LEA proteins from wheat prevent aggregation and protect the citrate synthase from desiccating conditions like heat and droughtstress (Goyal *et al*., 2005). Group 1 LEA proteins exhibit protective effect in presence of trehalose and acts synergistically to prevent heat-induced protein aggregation. Three low-molecular-weight dehydrins have been identified in sugarcane leaves with increased expression in response to heat stress (Wahid and Close, 2007, Wahid 2006).Abscisic acid (ABA) and ethylene (C_2H_4), as stress hormones, are involved in the regulation of many physiological properties by acting as signal molecules (Leung and Giraudat, 1998)

6. CONCLUSION

High temperatures affect plant growth at all developmental stages. Pollen viability, patterns of assimilates partitioning, and growth and development of seed/grain adversely affected. Other notable heat stress effects include structural changes in tissues and cell organelles, disorganization of cell membranes, disturbance of leaf water relations, and impedance of photosynthesis via effects on photochemical and biochemical reactions and photosynthetic membranes. In response to heat stress, plants manifest numerous adaptive changes. The induction of signaling cascades leading to profound changes in specific gene expression is considered an important heat-stress adaptation. A fundamental heat-stress response ubiquitous to plants is the expression of HSPs, which range from low (10 kDa) to high (100 kDa) molecular mass in different species. Evidence on synthesis and accumulation of some other stress-related proteins is also available.

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