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EFFECT OF HEAT STRESS ON GROWTH AND DEVELOPMENT OF CROPS

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Abstract

Extreme heat stress during the crop reproductive period can be critical for crop production. Crop species and cultivars differ in their sensitivity to high temperatures. Rates of photosynthesis and respiration increase with an increase in temperature until a threshold maximum photosynthesis level is achieved. Surpassing the high temperature peak, various enzymes get inactivated, decreasing the photosynthetic efficiency. The high temperature coupled with high respiration and evaporation pushes the plant to permanent wilting when the temperature exceeds 46°C. The extent of crop loss by heat stress can ruin the income for farmers. High temperature stress induces several alterations in physiological, biochemical and molecular components in rice crop leads to reduction in crop production. It is vital to know the mechanisms, which govern the various seed filling events under heat stress environments, to formulate strategies to improve heat tolerance in crop. In this review, we present research on heat stress affect the various process in plant growth and development that may help to understand the mechanisms that make plants tolerant or susceptible to heat stress.

Key words : *Heat stress, growth, crops, photosynthesis.*

The increasing temperatures are resulting in heat stress for various agricultural crops to limit their growth, metabolism, and leading to significant loss of yield potential worldwide. At Present, heat shocks due to the rising atmospheric temperatures are becoming one of the major limiting factors to crop productivity around the globe. This rising temperature may cause a change in the growing periods and the distribution of the agricultural crops (1). Heat stress adversely affects normal plant growth and development depending on the sensitivity of each crop species. Heat stress is defined as the increase in temperature beyond a threshold level for a period of time sufficient to cause irreversible damage to plant growth and development (Table-1). Growth and development of the plants is also greatly affected by the series of morphological, biochemical and physiological changes resulted by high temperature stress (Wahid et al., 2007). At very high temperature, severe cellular injury and even cell death may occur within minutes, which could be collapse of cellular organization (Schoffl et al., 1999). At moderately high temperatures, injuries or death may occur only after long-term exposure. Direct injuries due to high temperatures include protein aggregation and denaturation, and increased fluidity of membrane lipids. Indirect or slower heat injuries include inactivation of enzymes in chloroplast and mitochondria, inhibition of protein synthesis, protein degradation and loss of membrane integrity (Howarth, 2005). In this review, we present research on all these levels of investigation that may help to understand the mechanisms that make plants tolerant or susceptible to heat stress.

Mechanisms for heat tolerance : Plants have evolved several mechanisms that enable them to tolerate higher temperatures. These adaptive thermotolerant mechanisms reflect the environment in which a species has evolved.

Four major aspects of thermotolerance have been studied :

Biochemical and metabolic levels

Membrane stability

Production of heat shock proteins

Photosynthesis and productivity during high temperature stress.

Biochemical and metabolic levels : Plants must be protected from heat-induced oxidative stress so that they can survive under high heat. Tolerance to heat stress in crop plants has been associated with an increase in antioxidative capacity (Figure-1). The impairment of metabolic function during heat stress results in increased production of reactive oxygen species (ROS), which in turn causes secondary damage to proteins and membranes. The production of the ROS has been reported under high temperature stress (2). During heat stress accumulation of ROS has been associated with both the light reactions and the Calvin cycle reactions. The reaction centre of PSII is particularly vulnerable, producing superoxide radicals, hydroxyl radicals and hydrogen peroxide under heat stress. Antioxidant enzymes and non-enzyme systems serve to limit the formation of the most damaging ROS, such as singlet oxygen, and detoxify the cells through ROS-scavenging. Although some antioxidant systems are impaired at high temperatures, others are up-regulated and can be considered component of the heat stress response (5).

Several key phytohormones including ABA, salicylic acid (SA), and ethylene (ET) also increase their levels under heat stress, while others decrease, such as

cytokinin (CK), auxin (AUX), and gibberellic acids (GAs), fluctuations that ultimately cause premature plant senescence. For example, the abscission of reproductive organs, an important effect of heat stress, is known to be caused by increased ABA and ET levels and reduced levels and transport of AUXs. Similarly, an altered AUX biosynthesis in developing anthers was related to pollen sterility (6). A comparable variation in CK content was also found to be the cause of reduced kernel filling in cereals (7).

Membrane stability : In order to tolerate high temperatures, plants must maintain membrane fluidity within a biologically functional range (membrane thermostability). The degree to which membrane fluidity increases with temperature is dependent on membrane composition. Lipids that have unsaturated fatty acid chains, short fatty acid chains or a low sterol content generally form membranes that are more fluid and less stable at high temperatures. The sensitivity of membranes to heat stress can reduced by increasing the proportion of saturated lipids or by altering the composition of specific lipids. Changes in lipid composition during acclimation to high temperature, including increases in the proportion of saturated lipids, have been described in cyanobacteria (8) and a number of plants from both warm and cool regions (9). Some of the changes in the physical properties of membranes are regulated by the activity of heat shock proteins, but others are not (10).

Alteration of lipid composition through gene manipulation has been shown to increase heat tolerance in Arabidopsis, soybean and tobacco (11). The structure and fluidity of lipid membranes is dependent on their composition and on temperature. An increase in temperature will result in an increase in the fluidity of lipid membranes as the hydrogen bonding between adjacent fatty acids become weak. This increase in fluidity is associated with an uncontrolled increase in membrane permeability as the activity of membrane bound proteins is disrupted. Indeed, this uncontrolled membrane permeability is used as an assay to test for damage due to heat stress.

Membrane-associated processes, such as photosynthesis and membrane transport, are typically the first to be inhibited during exposure to high temperature (5). The high temperature sensitivity of PSII is thought to be due, at least in part, to its close association with the thylakoid membrane. In addition to these direct effects on metabolic function the changes in membrane fluidity during heat stress act as a signal to initiate other stress responses in the cell (12).

Heat shock proteins : Within minutes of temperature rising above the optimal, a sub-set of specific stress response genes are actively up-regulated known as heat shock proteins (HSP). HSP occur in all organisms. In plants, they show differential expression in many tissues and cell compartments. Based on their molecular masses these proteins have been classified into six major families i.e. Hsp100, Hsp90, Hsp70, Hsp60, Hsp40 and small heat shock proteins (sHsps). sHsps have subunit molecular masses of 12-43 kDa and are characterized by the presence of a highly conserved stretch of 80-100 amino acids in their C-terminal domains called the “á-crystallin domain” (ACD) that is flanked by less conserved (except a few stretches) N-terminal domain (NTD) and C-terminal extension (CTE) (13). Some members of the sHsp family such as Hsp27, áA- and áB-crystallin form large oligomeric species. These HSPs help in coping with heat stress by improving photosynthesis, partitioning of assimilate, nutrient and water use efficiency and membrane thermal stability (2). HSP utilize a novel transcription factor to respond directly to heat, and their levels have been shown to rise along with temperature until the lethal threshold temperature is reached.

Many HSP are thought to act as chaperone proteins, protecting other proteins from denaturation by reducing misfolding, unfolding, and aggregation. Chaperone activity also helps maintain the translocation of proteins across cell membranes. Immuno-localization studies have determined that HSPs normally associate with particular cellular structures, such as cell wall, chloroplasts, ribosomes and mitochondria (14). When maize, wheat and rye seedlings were subjected to heat shocks (42°C), whereas five mitochondrial LMW-HSPs (28, 23, 22, 20 and 19 kDa) were expressed in maize, only one (20 kDa) was expressed in wheat and rye, suggesting the reason for higher heat tolerance in maize than in wheat and rye (15). The expression profiles of 23 rice sHSPs under vegetative, developmental and stress conditions were reported (16).

Photosynthesis : Generally, inhibition of photo-synthesis is seen as a critical factor in heat stress. Net photosynthesis is typically the first process to be inhibited at high temperatures (5). As temperature rises above optimum, gross photosynthesis is inhibited while respiration and photorespiration increase. The combined effect of these three processes is a marked reduction in net photosynthesis during moderate heat stress.

Light dependent chemical reactions taking place in the thylakoid and the carbon metabolism taking place in the stroma are the main sites of damage as a result of the high temperature stress. Increased temperature of the leaf and photon flux density effects the thermo-tolerance adjustment of the PSII (17). The PSII is very much responsive to temperature and its activity is greatly influenced and even partially terminated under high

Table-1 : Threshold high temperatures for some crops.

Crop plants	Threshold temperature (°C)	Growth stage	References
Wheat	26	Post-anthesis	Stone and Nicolas (1994)
Corn	38	Grain filling	Thompson (1986)
Tomato	45	Reproductive	Rehman et al. (2004)
Pearl millet	35	Seedling	Ashraf and Hafeez (2004)
Tomato	30	Emergence	Camejo et al. (2005)
Brassica	29	Flowering	Morrison and Stewart (2002)
Groundnut	34	Pollen production	Vara Prasad et al. (2000)
Cowpea	41	Flowering	Patel and Hall (1990)
Rice	34	Grain yield	Morita et al. (2004)

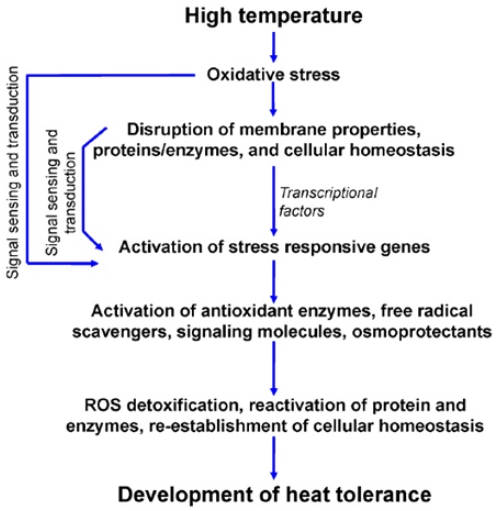

Fig-1 : Heat induced signal transduction mechanism and development of heat tolerance in plants.

temperature stress (18). C_4 plants do not suffer from the increase in photorespiration and so can maintain a higher photosynthetic optimum. The imbalance between photosynthesis and respiration is itself damaging, as carbohydrate reserves can become depleted. As temperature rises further, membrane transport and respiration become inhibited, eventually leading to cell death. Both the light reactions and the Calvin cycle are highly sensitive to moderate heat stress. Injury following severe heat stress is perhaps most acute for the light reactions, with even brief exposure resulting in long-term inhibition of photosystem II (PSII). As the activity of PSII is highly temperature sensitive it can be used as an indicator of heat stress and heat injury; measurements of chlorophyll fluorescence have been widely used for this purpose Severe heat stress is still thought to be due to injury of PSII, through direct cleavage of the D1 protein and a range of other mechanisms. Although the thermal sensitivity of PSII is not solely due to the thermal sensitivity of cell membranes, membrane properties are a major regulator of both inhibition and injury of PSII (5).

Net photosynthesis in many plant species is inhibited due to reduction in the activation state of the CO_2 binding enzyme, Rubisco (17). Although the catalytic activity of Rubisco increases with rising temperature, its low affinity toward CO_2 and capability of binding with O_2 limits the increase in net photosynthesis rate (17).

Plant growth and development : High temperatures may cause scorching of the twigs and leaves along with visual symptoms of sunburn, leaves senescence, growth inhibition and discoloration of fruits and leaves (19). As temperature increases within a plant's thermal range, the duration of growth decreases but the rate of growth increases. This response can be seen in a range of tissues including leaves, stems and fruit. A smaller organ size at maturity due to high temperature is associated with smaller cells rather than a change in cell number. This implies that cell enlargement is more sensitive to temperature than is cell division. The reduced duration of development can also limit the number of organs that are produced, e.g. grain number in wheat is reduced when plants are grown at moderately high temperatures (20). High temperature stress reduced number of spikes and number of florets per plant in rice and seed-set in sorghum was also negatively affected under similar conditions (21).

The thermal sensitivity of reproductive processes can be a limiting factor for plant productivity. Heat stress can reduce the duration of reproductive development and severely inhibits floral development, fertilization and post fertilization processes in many species. Pollen viability is particularly vulnerable to heat damage. Severe heat stress inhibits both the photosynthetic source and the reproductive sink, resulting in a significant reduction in the number and size of seeds and/or fruit. Reduction in the activities of source and sink takes place under heat stress which greatly effects the growth and ultimately the economic yield (22). This is a particular problem in fruit and grain crops such as tomato, cowpea, wheat, and maize.

CONCLUSION

High temperature stress has become a major concern for crop production worldwide because it greatly affects the growth, development, and productivity of plants. The responses of plants to heat stress have been studied intensively in recent years; however, a complete

understanding of thermo-tolerance mechanisms remains elusive. Under heat stress conditions, plants accumulate different metabolites (such as antioxidants, osmoprotectants, heat shock proteins [HSPs], etc.) and different metabolic pathways and processes are activated. These changes emphasize the importance of physiological and molecular studies to reveal the mechanisms underlying stress responses. At the field level, managing or manipulating cultural practices, such as the timing and methods for sowing, irrigation management, and selection of cultivars and species, can also considerably decrease the adverse effects of heat stress. Field experiments that explore different biochemical and molecular approaches and agronomic management practices are needed to investigate the actual heat responses and their effects on final crop yield. Molecular approaches that uncover the response and tolerance mechanisms will pave the way to engineering plants capable of heat tolerating and could be the basis for development of crop varieties capable of producing economic yields under heat stress.

REFERENCES

1. Porter, J.R. (2005). Rising temperatures are likely to reduce crop yields. *Nature, 436:* 174.
2. Wahid, A., Gelani, S., Ashraf, M. and Foolad, M. 2007. Heat tolerance in plants: An overview. *Environmental and Experimental Botany, 61:* 199–223.
3. Schoffl, F., Prandl, R. and Reindl, A. (1999). Molecular responses to heat stress. In K. Shinozaki & K. Yamaguchi-Shinozaki (Eds.), Molecular responses to cold, drought, heat and salt stress in higher plants. Austin, TX: Landes, R.G. pp. 81–98.
4. Howarth, C.J. (2005). Genetic improvements of tolerance to high temperature. In M. Ashraf & P.J.C. Harris (Eds.), Abiotic stresses: Plant resistance through breeding and molecular approaches New York, NY: Howarth Press. pp. 277–300.
5. Allakhverdiev, S. I., Kreslavski, V. D., Klimov, V. V., Los, D. A., Carpentier, R. and Mohanty, P. (2008). Heat stress: An overview of molecular responses in photosynthesis. *Photosynthesis Research. 98:* 541–550.
6. Sakata, T., Oshino, T., Miura, S., Tomabechi, M., Tsunaga, Y., Higashitani, N., Miyazawa, Y., Takahashi, H., Watanabe, M., Higashitani, A. (2010). Auxins reverse plant male sterility caused by high temperatures. *Proc. Natl. Acad. Sci. 107:* 8569–8574.
7. Banowetz, G., Ammar, K., and Chen, D. 1999. Temperature effects on cytokinin accumulation and kernel mass in a dwarf wheat. *Ann. Bot. 83:* 303–307.
8. Los, D.A. and Murata, N. 2004. Membrane fluidity and its roles in the perception of environmental signals. *Biochim Biophys Acta,* 1666: 142–157.
9. Larkindale, J. and Huang, B. (2005). Effects of abscisic acid, salicylic acid, ethylene and hydrogen peroxide in thermotolerance and recovery for creeping bentgrass. *Plant Growth Regul., 47 :* 17–28.
10. Sharkey, T.D. 2005. Effects of moderate heat stress on photosynthesis: importance of thylakoid reactions, rubisco deactivation, reactive oxygen species, and thermo-tolerance provided by isoprene. *Plant Cell Environ. 28:* 269–277.
11. Alfonso, M., Yruela, I., Almarcegui, S., Torrado, E., Perez, M.A., Picorel, R. (2001). Unusual tolerance to high temperatures in a new herbicide-resistant D1 mutant from *Glycine max* (L.) Merr. cell cultures deficient in fatty acid desaturation, *Planta. 212:* 573-582.
12. Kappe, G., Franck, E., Verschuure, P., Boelens, W.C., Leunissen, J.A. and de Jong W.W. (2003). The human genome encodes 10 alpha-crystallin-related small heat shock proteins: HspB1-10 Cell Stress Chaperones, Spring, *8(1):* 53-61.
12. Mittler, R., Finka, A., and Goloubinoff, P. (2012). How do plants feel the heat? *Trends in Biochemical Sciences, 37:* 118–125.
13. Franck E., Madsen, O., van Rheede, T., Ricard, G., Huynen, M.A. and de Jong W.W. (2004). Evolutionary diversity of vertebrate small heat shock proteins *J. Mol. Evol., 59 :* 792-805.
14. Yang, K.A., Lim, C.J., Hong, J.K., Park, C.Y., Cheong, Y.H., Chung,W.S., Lee, K.O., Lee, S.Y., Cho, M.J. and Lim, C.O. (2006). Identification of cell wall genes modified by a permissive high temperature in Chinese cabbage. *Plant Sci. 171:* 175–182.
15. Korotaeva, N.E., Antipina, A.I., Grabelynch, O.I., Varakina, N.N., Borovskii, G.B. and Voinikov, V.K. (2001). Mitochondrial low-molecular-weight heat shock proteins and tolerance of crop plant's mitochondria to hyperthermia. Fiziol. *Biokhim Kul'turn. Rasten. 29:* 271–276.
16. Sarkar, N. K., Kim, Y. K., and Grover, A. (2009). Rice sHsp genes: genomic organization and expression profiling under stress and development. *BMC Genomics, 10:* 393.
17. Crafts-Brandner, S. J., and Salvucci, M.E. (2002). Sensitivity of photosynthesis in a C4 plant maize to heat stress. *Plant Physiol. 129:* 1773–1780**.**
18. Camejo, D., Rodriguez, P., Morales, M.A., Dellamico, J.M., Torrecillas, A., Alarcon, J.J. (2005). High temperature effects on photosynthetic activity of two tomato cultivars with different heat susceptibility. *J. Plant Physiol. 162:* 281–289.
19. Ismail, A. M., and Hall, A. E. (1999). Reproductive-stage heat tolerance, leaf membrane thermostability and plant morphology in cowpea. *Crop Sci. 39:* 1762–1768.
20. Stone, P.J., Nicolas and M.E. (1994). Wheat cultivars vary widely in their responses of grain yield and quality to short periods of post-anthesis heat stress. *Aust. J. Plant Physiol. 21:* 887–900.
21. Fahad, S., Hussain, S., Saud, S., Khan, F., Hassan, S., Amanullah, (2016). Exogenously applied plant growth regulators affect heat-stressed rice pollens. *J. Agron. Crop Sci. 202:* 139–150.
22. Taiz, L., and Zeiger, E. (2006). Plant Physiology, 4th Edn. Sunderland, MA: Sinauer Associates Inc Publishers.

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