



Effect of Heat Stress on Crop Production

(*J. S. Desai and N. A. Desai)

C. P. College of Agriculture, SDAU, S. K. Nagar, Gujarat-385506

*Corresponding Author's email: jigardesai182@gmail.com

A complicating factor in low temperature injury is the change of cell's water from the liquid to the solid state. At higher temperature, sudden change to vapour are not found under natural conditions. The slower vaporization at normal temperature is, however, the possible cause of injury. The simplest explanation of single cause (membrane damage) of both direct and indirect injury is that the chloroplast membrane is most sensitive to high temperature, the mitochondrial less and the plasma membrane least sensitive.

Indirect injury due to starvation would occur at temperature just high enough to damage the chloroplast membranes. Bio-chemical lesions, toxicity and protein net breakdown would occur at slightly higher temperature due to mitochondrial membrane damage. Finally, direct injury would occur at still higher temperature due to destruction of the integrity of the plasma membrane.

Injury

Secondary heat induce drought injury: Higher temperature increases the rate of cuticular transpiration. There are two reasons for the sharp rise in transpiration with the rise in environmental temperature, (a) direct effect of temperature on diffusion constant of water and (b) difference in vapour pressure gradient between the leaf and the external atmosphere. Therefore, prolonged high temperature stress often results in injury due to desiccation.

Primary indirect: There are four possible kinds of indirect high temperature injury, any one of which may conceivably inhibit growth in mild doses and actually injure the plant in large doses : starvation, biochemical lesions, protein hydrolysis and toxicity.

The first strain produced by high temperature is kinetic - an increase in reaction rates. Reverse relative reaction rates may lead to (1) an increase in net breakdown and therefore may produce a decrease in concentration or complete absence of an essential metabolite at heat-injuring temperatures. The resulting injury may be starvation, biochemical lesions and protein hydrolysis (2) conversely, it may produce an increase in concentration of a toxic substance normally not detectibly present or in too low a concentration to be injurious.

(1) Starvation: The temperature at which respiration and photosynthesis are equally rapid is called temperature compensation point. If the plants temperature rises above the compensation point, the plant's reserve will begin to deplete. Sufficiently longer time of such high temperature would ultimately lead to starvation and death. As the temperature rises above the compensation point, respiration rate continues to increase and photosynthesis to decrease. The deficit increase particularly rapidly in plants with an active photorespiration (C_3 plants) in addition to normal dark respiration. The existence of compensation temperature is due to the lesser resistance to high temperature of photosynthetic system than the respiratory apparatus.

(2) Biochemical lesions: Accumulation of intermediate substance necessary for growth is inhibited by high temperature. e.g. vitamins, cofactor, etc.

(3) **Protein hydrolysis:** This injury may occur due to net loss of protoplasmic proteins (under slightly more heat stress).

(4) **Toxicity:** There is little evidence that toxicity is associated with high temperature injury. Toxicity may be as a result of respiratory disturbances. e.g. increased anaerobic respiration.

Primary direct: Membranes *viz.*, plasma, nuclear, chloroplast, mitochondrial, ER and golgi are the loci of injury. This is supported by leakage of many substances from heat shocked cells. All the membranes consist of two substances *viz.*, proteins and lipids. Heat injury involves both lipid and protein changes. Membrane damage is the cause of direct heat injury. A loss of semi-permeability or inactivation of the active uptake system at high temperature could be due to either (1) lipid liquefaction, *i.e.*, excessive fluidity of lipids leading to disruption of the lipid layer or (2) denaturation and aggregation of the membrane protein.

Many proteins are denatured at heat killing temperatures. Rapid heat killing may occur if denatured proteins are aggregated. Heat injury results in an increased SS content of proteins at the expense of SH groups disturbing SS:SH ratio. Absence of SH groups could lead to SS-induced aggregation at high temperature.

Plant Adaptation to Heat Stress

Survival in hot, dry environments can be achieved in a variety of ways, by combinations of adaptations. Plant adaptation to heat stress includes avoidance and tolerance mechanisms which employ a number of strategies.

(1) **Avoidance Mechanisms:** Under high temperature conditions, plants exhibit various mechanisms for surviving which include long-term evolutionary phenological and morphological adaptations and short-term avoidance or acclimation mechanisms.

- Stomata : Closure of stomata and reduced water loss.
- Early maturity : Early maturation is closely correlated with smaller yield losses under high temperature, which may be attributed to the engagement of an escape mechanism.
- Reduced absorption of solar radiation : Plants growing in a hot climate avoid heat stress by reducing the absorption of solar radiation.
- Tomentose : The presence of small hairs (tomentose) that form a thick coat on the surface of the leaf as well as cuticles, protective waxy covering.
- Paraheliotropism : Leaf blades often turn away from light and orient themselves parallel to sun rays (paraheliotropism).
- Rolling leaf blades : Solar radiation is reduced by rolling leaf blades. Physiological role of leaf rolling was the maintenance of adaptation potential by increasing the efficiency of water metabolism in the flag leaves of wheat under high temperature.
- Small leaves : Plants with small leaves are also more likely to avoid heat stress: they evacuate heat to ambient more quickly due to smaller resistance of the air boundary layer in comparison with large leaves.
- Transpiration : In well-hydrated plants, intensive transpiration prevents leaves from heat stress, and leaf temperature may be 6 °C or even 10–15 °C lower than ambient temperature.
- Life histories : Many species have evolved life histories which permit them to avoid the hottest period of the year. This can be achieved by leaf abscission, leaving heat resistant buds, or in desert annuals, by completing the entire reproductive cycle during the cooler months.
- Biochemical adaptations :
 - Biochemical adaptations favoring net photosynthesis at high temperature (in particular C₄ and CAM photosynthetic pathways), although C₃ plants are also common in desert floras.
 - Alteration of membrane lipid compositions.

(2) **Tolerance Mechanisms:** Heat tolerance is generally defined as the ability of the plant to grow and produce economic yield under high temperature. This is a highly specific trait, and closely related species, even different organs and tissues of the same plant, may vary significantly in this respect. Plants have evolved various mechanisms for thriving under higher prevailing temperatures. Some major tolerance mechanisms, including ion transporters, late embryogenesis abundant proteins, osmoprotectants, antioxidant defense, and factors involved in signaling cascades and transcriptional control are essentially significant to counteract the stress effects.

Management Practices

(1) **Date of sowing:** In temperate or subtropical climatic zones, which have seasonal variations in temperature, sowing date can be varied to increase the probability that annual crop species will escape stressfully high temperatures during subsequent sensitive stages of development. For example, sowing dates can be chosen so that reproductive stages that are particularly sensitive to heat do not occur during periods when stressfully hot weather is most likely to occur.

(2) **Choice of crop species and cultivars:** Crop species and cultivars should be chosen that are adapted to the high temperatures likely to occur in the specific location.

(3) **Depth of sowing:** In tropical zones, inadequate plant emergence and establishment can limit the productivity of several warm-season annual crops. The soil surface can become very hot and substantially reduce emergence independently of drought effects. Hot soils retard hypocotyl elongation and this can have a detrimental effect on emergence, which is aggravated by deep sowing of seeds. When soils are hot, seed must be sown at a depth that is neither too deep which constrain hypocotyl emergence nor too shallow and be too close to the very hot surface.

(4) **Irrigation and other management practices:** A degree of escape of high leaf temperatures can be achieved by insuring that maximum transpiration rates are maintained since evaporative cooling can result in leaf temperatures cooler for rapidly transpiring plants compared with slowly transpiring plants. Plants transpire at maximum rates if their root zones have high levels of soil water and adequate aeration.

(5) **Use of exogenous protectants:** In recent decades, exogenous application of protectants such as osmoprotectants, phytohormones, signaling molecules, trace elements, *etc.* have shown beneficial effect on plants grown under high temperature as these protectants has growth promoting and antioxidant capacity.