Journal of Agroecology and Natural Resource Management
Print ISSN: 2394-0786, Online ISSN: 2394-0794, Volume 1, Number 2; November-January, 2014 pp. 51-55

http://www.krishisanskriti.org/janrm.html

Biospectrum of Heat Adaptations

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Abstract: *Global warming has increased temperature of earth and oceans causing the heat stress to all living beings. Heat stress manifestations are visible in living kingdom. The cells or thermoregulatory centres in the living beings can sense the change in the temperature and can adapt to these changes. Based on degree of adaptability living beings have been classified as endotherms, ectotherms, eurytherms and stenotherms. If we see evolutionary pathway we realize that different animals use different mode of adaptations. It is interesting to note that there are similarities and uniqueness in heat stress adaptations among various species. Adaptations can be broadly categorized as behavioral, physiological and molecular ones. Behavioral adaptations include essentially avoiding the heat stress by moving to comfortable microclimate. Physiological adaptations include activation of thermoregulatory processes such as evaporation, perspiration, panting and also changes in physiological functions of cardiovascular, respiratory, digestive, excretory and nervous system. At the molecular level body tries to cope up by releasing various hormones like cortisol, aldosterone and thyroid hormones. At genetic level the transcription of certain genes (anti oxidative, anti apoptotic etc) is altered. Major role in heat stress acclimation is played by heat stress proteins (HSPs, Chaperones etc) family members like HSP 70,72,73, HSP 90, HSP 105 etc. These chaperones are used as biomarkers of heat stress and acclimation. However these biomarkers are limiting in giving the deep insight for understanding the process of thermoregulation. Thus we need to identify other biomarkers which are representatives of heat stress and acclimation alone and are not seen in other stress conditions. Learning about the etiology of heat stress and acclimatization will help to achieve heat acclimation by change in life style or by intervention with diet / drugs. Thus making it possible to move from heat stress to heat de-stress!*

1. INTRODUCTION

Heat stress due to increased temperature is a serious threat to the living beings. Global warming causing extreme heat events are already a significant public health problem all over the world. Heat related illnesses are serious and can lead to death. In fact, extreme heat events are responsible for high mortality rate annually than hurricanes, lightning, tornadoes, floods, and earthquakes combined. Both occasional and prolonged high temperatures can cause morphological, anatomical, physiological, biochemical and molecular changes in both flora and fauna.

Heat stress effects the plant growth and development causing reduced yield and quality. However some plants show heat tolerance. High temperature causes inactivation of enzymes in chloroplast and mitochondria, protein degradation, and loss of membrane integrity due to an increase in fluidity of membrane lipids, production of reactive oxygen species (ROS) etc. As a part of heat stress defense strategy of the cell heat shock proteins (HSPs) are produced[3]. In many plant species HSPs improve the membrane permeability, photosynthesis, assimilate partitioning etc.

Animals have been divided into various categories depending on how they adapt or regulate their body temperature as homeotherms, poikilotherms and heterotherms. Depending on the source of their heat they are referred as endotherms and ectotherms. Animals like mammals and birds which create their own heat are endotherms and the animals like fishes, amphbians and reptiles receive heat primarily from external sources are the ectotherms. Homeotherms and heterotherms are also being referred as endotherms, and poikilotherms as ectotherms. However, it is not always true. For example, insects are poikilotherms but not true ectotherms as they produce their own heat in order to fly. Animals which have the ability to tolerate a wide range of temperature are the eurytherms (example: cat, dog etc) and animals which can tolerate only a narrow range of temperature are the stenotherms (example: python, crocodile etc)

Thermoregulation is one of the most explicit forms of homeostasis. The nervous system of invertebrates consists of richly interconnected networks containing many neurons with varied physiological properties. As temperature varies the neuronal functions are affected. To stabilize the neuronal function, invertebrates adapt by changing the composition of neuronal cell membranes. They change the ratio of saturated and unsaturated fatty acids in the membranes which affects the generation and transmission of action potential.

In the case of vertebrates, particularly homeotherms possess sophisticated ability to control their body temperature because of the presence of **thermoregulatory centre.** In humans the thermoregulatory centre exists in the brain. It is the preoptic

anterior hypothalamus (POAH). This gets input from the skin, visceral organs and the brain. It sends out output to the skin, muscles and the sweat glands. It maintains the physiological temperature of 37°C

Heat transfer between the body of the organism and the macroenvironment occurs by conduction, convection, radiation and evaporation. Very high temperatures can cause severe cellular injury and cell death within short time, thus leading to a catastrophic collapse of cellular organization[10]. However, high level of heat tolerance is seen in biological kingdom[13] because of series of adaptations which are genetically encoded but the evolutionary events leading to such phenomenon remain obscure.

2. EFFECTS OF HEAT STRESS

Heat stress shows multiple effects on almost all the internal organs resulting in serious health ramifications.

Table 1: Effects of Heat Stress

Brain	Decreased cerebral perfusion, Destruction of purkinje cells responsible for motor coordination in the cerebellar cortex. There are changes which results in memory loss, loss of concentration, causes dizziness and delirium.
Heart	To maintain increased blood flow to the skin the cardiac output may increase upto 8L/min. The heat stress would increase sympathetic stimulation which may increase the heart rate.
Gut	There is decreased splanchnic blood flow. There is decreased blood flow to the Gut which leads to nitrosative and oxidative stress. Increased anaerobic metabolism and acidosis. There is increased permeability of the epithelial cells of the gut as the tight junctions of the epithelial cells become leaky.
Liver	There is reduced blood supply to the liver The power of liver to detoxify endotoxins also decreases. There can be acute liver failure.
Kidney	As the core body temperature rises above 40°C, the renal blood flow is greatly reduced, leading to kidney failure. Reduced filtration and urinary flow.
Muscle	Severe dehydration leads to Rhabdomyolysis. It is the breakdown of muscle tissue that leads to the release of muscle fiber contents into the blood. These substances are harmful to the kidney and often cause kidney damage. Skeletal muscle's plasma membrane becomes severely injured and the contents of the cells are leaked into the blood leading to myoglobinuria.
Blood	Biophysical and biochemical changes in the blood. Dehydration increases viscosity of blood which affects its fluidity and circulation Heat stress also affects the coagulation capacity of the blood.

3. HEAT ILLNESS

When heat is combined with other stressors such as physical work, loss of fluids, fatigue or some other medical conditions, it may lead to heat related illnesses like heat syncope, heat cramps, heat exhaustion and heat stroke.

Table 2: Types of Heat illness

Heat Syncope	Syncope means "fainting." First signs include dizziness and light-headedness. Generally it occurs in the beginning of summers before the circulatory system adapts to heat.
Heat Cramps	Cramping (muscular pain) occurs after the body loses a significant amount of salt. It affects both active muscles (arms, legs) and involuntary (usually abdominal) muscles.
Heat Exhaustion	Occurs upon prolonged exposure to high temperature and dehydration. (extremely tired) Symptoms include nausea, dizziness, weakness headache, blurred vision and profuse sweating.
Heat Stroke	The blood to the brain stops. Core temperature rises beyond 40.5°C Symptoms include dry skin, cessation of sweating, and loss of consciousness.

4. HEAT STRESS AND ACCLIMATION

Heat stress occurs when the body's thermoregulatory systems fails to regulate the internal temperature. Whereas acclimation is the process in which an individual organism adjusts to a gradual change in its environment (such as a change in temperature, humidity, photoperiod, or pH), allowing it to maintain performance across a range of environmental conditions**.**

Heat acclimation reduces the core body temperatures, the heart rate, and the physiological strain during exercise/heat stress[9]. In acclimation the endurance capacity of the individual to heat stress in work increases[2,8]. The acclimation happens in a biphasic manner. In the initial responses are called as short term heat acclimation (STHA). There after in the long term heat acclimation (LTHA) the temperature and physiological function of most organs come back to normal.

Temperature-adaptive physiological variation plays important roles in latitudinal biogeographic distribution. Differences among species in thermal tolerance limits and in the capacities to adjust these limits may determine how organisms are affected by climate change.

5. BEHAVIORAL AND PHYSIOLOGICAL ASPECTS OF HEAT STRESS AND ACCLIMATION IN ANIMALS

In higher organisms hypothalamus is the thermoregulatory part of the brain which signals the animal to make appropriate changes. Animals show behavioral changes like on hot days they will move to shady, wet, cool rocks and transfer their excess body heat away. For example snakes find dark crevices to avoid absorbing heat on hot days. Fishes change their depth in the water column to adjust their temperature.

Honeybees show both "Passive" and "Active" nest temperature regulation. Nest structure and nest site selection

falls in passive mechanism. Clustering of individuals together to handle temperature of the entire hive by evaporative cooling or wing fanning is the active mechanism. Nest thermoregulation also helps to regulate the temperature of the developing larvae. However at higher temperature (upto 46°C.) during flights honeybees use an evaporative cooling mechanism. They repeatedly regurgitate a droplet of fluid and suck it back in.

The processes that can remove the heat are convection from the skin to the air, heat loss through sweat evaporating from skin, and heat radiation to cooler objects near the body. Other animals, such as dogs pant to release saliva for evaporation. Kangaroos lick their front paws to stay cool. In response to heat stress the cattle shows decreased food intake, acidosis in the stomach and decrease in milk yield.

Camel is well adapted to desert life. It starts to sweat when the body temperature exceeds 42°C. The sweat evaporates from the skin, not from the hair, so the heat is drawn from the body, not from the atmosphere. Water is conserved, the source being the alimentary canal. It conserves water and excretes dry feces. Fecal water loss is reduced, so camels excrete dry feces. Water and urea are absorbed to produce a concentrated urine. Camel cells are virtually unaffected by high internal urea concentrations; in this they resemble marine sharks. Camels rehydrate rapidly, being able to drink 200 litres of water in 3minutes. In arid conditions camels are better milk producers than cattle, sheep or goats. In hot climates a cow will give 0.5-l kg milk a day whilst under similar arid conditions a camel will give 4 kg milk a day[16]. The camel also undergoes selective brain cooling by sending the blood cooled by the nasal evaporation which protects its brain from damage and confers greater endurance in heat[1] **.**

Thus it is evident that different animals use various modes of adaptations.

6. MOLECULAR ASPECTS OF HEAT STRESS AND ACCLIMATION

At molecular level changes in gene expression of heat shock proteins and many other proteins ensure protection to survive in heat stress conditions.

6.1 The Heat Shock proteins (HSPS)/Chaperones:

The animals can be exposed to several types of stress. These can be environmental high temperature, low temperature bacterial and viral infection, radiation, heavy metal, salinity, hypoxia, pesticide, oxidative stress, physical activity, aging or genetic etc. In all these stresses the heat shock proteins have been largely implicated in stress and acclimation. These are proteins which participate in normal folding and renaturation of proteins. They participate in protein quality control and maintain the protein homeostasis. The heat stress response occurs when the number of unfolded proteins increases to an extent that they cannot be folded by the normal levels of heat shock protein (HSPs). Thus the cells upregulate the expression of HSPs to maintain the protein homeostasis. During acclimation the HSP levels decrease but remain considerably higher than the non heat stressed controls. The expression of these chaperones are under the regulation of transcription factors like Heat shock Factor (HSF1,2) which can activate the Heat shock elements (HSE) in the nucleus. The HSE increases the transcription of the HSPs. The HSPs implicated in heat stress are HSP70,72,90, and those in heat acclimation are HSP 105. The HSPs can manifest their functions as antioxidative, antiapoptotic, antiageing etc.

The HSPs are found throughout the biological systems like bacteria, plants and animals. Their structures are highly conserved. The *Homo sapiens* and *Drosophila melangaste* share 50% similarity whereas *E.coli* and *Drosophila* share 70% similarity. The expression of HSP is correlated to the level of stress[7].

These chaperones are not specific to any type of stress as it is evident from the fact that the ambient temperature hot or cold stress can both upregulate the HSP. In heat stress these are upregulated immediately on exposure to heat and the levels remain high thereafter. But cold stressed species show upregulation of HSPs on acclimation or return to normal temperature. Moreover it has been seen that cold stress upregulates cold shock proteins viz. CIRP a mitotic protein, RBM3, KIAA0058.

6.2 Heat acclimatisation can help in cross tolerance and can maintain the molecular memory:

Heat acclimation is due to molecular changes as it can give cross tolerance to other stress factors[12]**.** It can also leave a molecular memory as the initial acclimation which required 30 day could be achieved in two days only in humans. But there is upregulation of certain gene which confers heat adaptations. But expression of HSP continue to be higher than non-stressed condition[4,5].

6.3 The molecular expression of Non- HSP genes

The molecular aspects of heat stress and acclimation show changes in the gene expression of several Non HSP genes which can regulate the following systems:

- Apoptotic/necrotic response
- Cytoskeleton
- Endocrine profile
- Immune response
- Membrane permeability
- Metabolism
- Protein homeostasis

- Signaling pathways
- Transcription factors

Apoptosis/necrosis:

The inhibition in formation of apoptotic inhibitor like bcl2 promotes apoptosis. The heat stress tissues show pyknotic cell nuclei indicating the presence of apoptosis or necrosis in the tissue cells.

Cytoskeleton and muscle function:

The heat stressed rat heart shows V1 myosin in normal and V2 and V3 myosin in heat acclimated heart. This may adapt the heart to chronic heat[6]. Heat acclimation improves the mechanical and metabolic performance by changing the permeability to calcium from the extracellular source. Mild heat stress leads to reorganization of actin filaments into stress fibers whereas severe heat stress leads to aggregation of cytoskeleton proteins.

Endocrine profiles:

The thyroid hormones, prolactin, somatotrophin, glucocorticoids, and mineralocorticoids, epinephrine, progesterone, estrone are the major hormones whose levels increase during the heat stress and which help the body to adapt to heat stress.

Immune pathway:

The increase in HSPs can upregulate the expression of cytokines[11]. The increase in core body temperature in heat stress is like fever but the mechanism of such increase is different in these situations. In fever the immune response that is the fight between pathogen and the immune components increases the core body temperature. But in heat stress it is the failure of thermoregulatory mechanism to dissipate heat which leads to this increase in core body temperature.

Membrane permeability:

The permeability of gut epithelial cells change as the tight junctions of these cells becomes leaky. This leads to the leak of endotoxins into the intracellular spaces which evoke immune response. The change in membrane permeability leads to drop in cellular pH and changes in ion homeostasis.

Metabolism:

The heat stress can induce tissue specific changes. The heat stress increases the expression of manganese superoxide dismutase (MnSOD) in cardiac myocytes but not in pneumocytes. The energy metabolism shows changes by activating carbohydrate and amino acid metabolism and inhibiting lipid metabolism in heat stress[15]. The hepatic and skeletal tissues show different metabolic preference in heat stress. There is net reduction in ATP production from metabolism.

Protein Homeostasis:

The heat stress can disturb the protein homeostasis. Several proteins denature by the increased core temperature. This signals the cell to decrease protein synthesis. It can also send these proteins for refolding by chaperones to the endoplasmic reticulum. The chaperone levels like HSP 70,72,90,105 etc. are upreguated as body adapts to heat stress and acquires acclimation. Otherwise these unfolded proteins are degraded in the proteosomes by ubiquitin mediated proteolysis. If all this is not possible the cell is signaled for apoptosis or necrosis.

Signaling pathway:

It effects the expression of myc, MAP kinases etc which are the components of the signaling pathways.

Nitric oxide synthase etc which are the components of the signaling pathways. These help in regulating cell cycle, phosphorylation of proteins, vasodilalation.

Transcription factors:

The transcription factors like HSF1.2, P53, AP-1 P21 is affected in heat stress and acclimation indicating the signaling pathways which may affect the protein homeostasis, cell cycle regulation, apoptosis etc.

7. BIOMARKERS OF HEAT STRESS AND ACCLIMATION:

Chaperones like HSP 70, 72, 90 have been considered as biomarkers of heat stress and HSP 105 have used as biomarkers of acclimation. However, these proteins can also been seen to rise in viral infection, cold, ageing, metal toxicity, hypoxia etc. Thus we see that these biomarkers are markers of stress and not necessarily heat stress and acclimation.

8. CONCLUSION

The heat stress affects the human mental and physical performance, the milk yield of the cattle, the productivity of plants etc., this leads to tremendous loss to the economy. We have understood the phenomenon of heat stress and acclimation to large extent. But there is a need to further understand the etiology of heat stress so that we can understand not only the molecular mechanism of heat adaptability completely with its uniqueness but also heat related illnesses better and can design drug/lifestyle to achieve acclimatization faster and reduce the economic and health loss.

9. ACKNOWLEDGEMENTS

The project entitled "Biomarkers and Heat Acclimation" is supported by a grant from the University of Delhi and DRDO.

REFERENCES

[1] Elkhawad, A. O. (1992). Selective brain cooling in desert animals: The camel (*Camelus dromedarius*). *Comparative Biochemistry and Physiology Part A: Physiology*, *101*(2), 195-201.

[2] Fruth, J. M., & Gisolfi, C. V. (1983). Work-heat tolerance in endurance-trained rats. *Journal of Applied Physiology*, *54*(1), 249-253.

[3] Hall, A.E. (2001). Crop Responses to Environment. CRC Press LLC, Boca Raton, Florida

[4] Horowitz, M. (2001). Heat acclimation: phenotypic plasticity and cues to the underlying molecular mechanisms. *Journal of Thermal Biology*, *26*(4), 357-363.

[5] Horowitz, M., Kaspler, P., Marmary, Y., & Oron, Y. (1996). Evidence for contribution of effector organ cellular responses to the biphasic dynamics of heat acclimation. *Journal of Applied Physiology*, *80*(1), 77-85.

[6] Horowitz, M, Peyser,Y.M, Muhlrad, A. (1986) Alterations in cardiac myosin isoenzymes distribution as an adaptation to chronic environmental heat stress in the rat. J of Molecular and Cellular Cardiology Volume 18, (5,) , Pages 511–515.

[7] Kregel, K. C. (2002). Invited review: heat shock proteins: modifying factors in physiological stress responses and acquired thermotolerance. *Journal of Applied Physiology*, *92*(5), 2177-2186.

[8] Kuennen, M., Gillum, T., Dokladny, K., Bedrick, E., Schneider, S., & Moseley, P. (2011). Thermotolerance and heat acclimation may share a common mechanism in humans. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *301*(2), R524-R533.

[9] Matthew K , Trevor G , Karol D , Edward B , Suzanne S , Pope(2011) Thermotolerance and heat acclimation may share a common mechanism in humans American Journal of Physiology - Regulatory, Integrative and Comparative Physiology Vol. 301(. 2),R524-R533

[10] Schöffl, F., Prandl, R., & Reindl, A. (1999). Molecular responses to heat stress. Molecular responses to cold, drought, heat and salt stress in higher plants. *RG Landes Co., Austin, Texas*, 81-98.

[11] Selkirk, G. A., McLellan, T. M., Wright, H. E., & Rhind, S. G. (2008). Mild endotoxemia, NF-κB translocation, and cytokine increase during exertional heat stress in trained and untrained individuals. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *295*(2), R611-R623.

[12] Shinozaki, K., & Yamaguchi-Shinozaki, K. (2000). Molecular responses to dehydration and low temperature: differences and cross-talk between two stress signaling pathways. *Current opinion in plant biology*, *3*(3), 217-223.

[13] Somero, G. N. (2005). Linking biogeography to physiology: evolutionary and acclimatory adjustments of thermal limits. *Front Zool*, *2*(1), 1-1.

[14] Tetievsky, A., Cohen, O., Eli-Berchoer, L., Gerstenblith, G., Stern, M. D., Wapinski, I., & Horowitz, M. (2008). Physiological and molecular evidence of heat acclimation memory: a lesson from thermal responses and ischemic cross-tolerance in the heart. *Physiological genomics*, *34*(1), 78-87.

[15] Windisch, H. S., Kathöver, R., Pörtner, H. O., Frickenhaus, S., & Lucassen, M. (2011). Thermal acclimation in Antarctic fish: transcriptomic profiling of metabolic pathways. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *301*(5), R1453-R1466.

[16] Yagil, R. (1985). The desert camel. *Comparative physiological adaptation*. Karger.