International Journal of Science, Environment and Technology, Vol. 6, No 4, 2017, 2252 – 2257

ISSN 2278-3687 (O)
2277-663X (P)

# PHYSIOLOGICAL AND METABOLIC ADAPTATION OF LIVESTOCK TO HEAT STRESS

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**Abstract:** India lying in the warm climatic zones of the world is subjected to extended periods of high ambient temperature and relative humidity. Despite of these high ambient temperatures, high direct and indirect solar radiation and humidity in tropical, sub-tropical and arid areas, native cattle sustain well by adapting to the environment through certain physiological and metabolic alterations in their body. These include alteration in water and electrolyte metabolism, changes in physiological parameters, exhibition of nycthermal pattern of acid-base balance, variations in secretion rate of various hormones and biochemical parameters. Hormones play a key role in thermal adaptation by involving with nutrient partitioning and homeorhesis or for homeostatic regulation, augmented by thermal stressor. All these adaptive physiologic responses are more pronounced in the native cattle *Bos indicus* than the exotic *Bos taurus* making them a thermotolerant breed.

**Keywords:** Heat stress, Physiological changes, Metabolic changes, Hormones, Zebu Cattle.

## Introduction

Cattle being homeothermic have the ability to regulate internal body temperature. This ability allows them to function in spite of variations in the surrounding environment and they are neither under heat or cold stress when the effective environmental temperature is in the thermoneutral zone. Some physiological processes are modified during heat exposure in an attempt to support heat balance and hence homeothermy is less disrupted. Reactions of homeotherms to moderate climatic changes are compensatory and are directed at restoring thermal balance. Under thermal stress, a number of physiological and behavioral responses vary in intensity and duration in relation to the animal genetic make-up and environmental

*Received June 12, 2017 * Published Aug 2, 2017 * www.ijset.net*

factors through the integration of many organs and systems viz. behavioral, endocrine, cardio-respiratory and immune system.

**Physiological Responses to heat stress**

Physiological parameters like respiration rate, skin temperature, udder skin temperature and pulse rate gives an immediate response to the climatic stress. Sweating, high respiration rate, vasodilation with increased blood flow to skin surface, high rectal temperature, reduced metabolic rate and altered water metabolism are the physiologic responses that are associated to combat heat stress.

Sethi (1994) found that physiological responses like rectal temperature (RT), pulse rate(PT) and respiration rate (RR) reflect the degree of stress imposed on animals by climatic parameters. The ability of an animal to withstand the rigors of climatic stress under warm conditions has been assessed physiologically by means of changes in body temperature, respiration rate and pulse Rate. In a hot environment the maintenance of the physiological responses of animals depends mainly on the cooling derived from the evaporation of water from respiration and salivation.

An evaporative heat loss from the respiratory tract is regarded as one of the primary mechanisms for maintenance of heat balance. This respiratory response arises from direct heat stimulation of peripheral receptors which transmit nervous impulses to the thermal centre in the hypothalamus. The cardio-respiratory centre is then stimulated to send impulses to the diaphragm and intercostals muscles for further respiratory activity. Chikamune and Shimizu (1983) observed a highly significant correlation between RR and seasonal air temperature in swamp buffalo and Holstein cows when the relative humidity (RH) was kept constant, whereas no such significant correlation was observed between RR and RH when the air temperature was kept constant. Respiration rate was indicator of heat stress in the hot environment and gave significant correlations with circulating corticoids concentration (Kumar *et al.,* 2005). Also by exhibiting a nycthermal pattern of acid-base balance, which is manifested by a high respiratory rate leading to respiratory alkalosis in the hot day time hours and lower urine pH and greater urine ammonia concentrations in the cool night time hours, animals can tolerate to ultra circadian temperature variations.

**Endocrine responses of livestock to heat stress**

The endocrine system involved heavily in coordination of metabolism is substantially altered because of thermal stress. The hormones associated with adaptation to heat stress are

prolactin (PRL), growth hormone (GH), thyroid hormones, glucocorticoids, mineralocorticoids, atecholamines and antidiuretic hormone (ADH).

Prolactin is vital for mammogenesis, lactogenesis and to varying degrees for galactopoiesis . Concentrations of plasma PRL are elevated during thermal stress in dairy cows. However, the function of this elevated PRL is not yet clear. Collier *et al*., (1982) proposed that elevated PRL is involved in meeting increased water and electrolyte demands of heat stressed cows.

Growth hormone is a calorigenic hormone produced from the anterior pituitary gland and does not function through a target gland but exerts its effects on almost all tissues of the body. The plasma GH levels declined from 18.2ng/ml at thermo-neutrality to 13.5ng/ml in heat stressed Jersey cows. Igono (1985) reported that GH content in milk of low, medium and high production groups declined when THI exceeded 70. Plasma GH reductions that occurred with heat stressed cows did not occur in thermoneutral conditions for cows fed restricted intakes that were similar to those consumed during heat stress . The decreased GH leads to less calorigenesis aimed in maintenance of heat in the body. In addition to calorigenesis, GH also enhances heat production by stimulating thyroid activity. Therefore, a reduced secretion of this hormone is all the more necessary for survival of the homeotherm in high ambient temperatures.

The thyroid gland secretes triiodothyronine ($T_3$) and tetraiodothyronine/ thyroxine ($T_4$). These hormones are the primary determinants of basal metabolic rate and have a positive correlation to weight gain or tissue production. The response of $T_3$ and $T_4$ to heat stress is slow and it takes several days for levels to reach a new steady state. A decline in the plasma concentrations of T from 2.2 to 1.16ng/ml has been reported (Johnson, 1980) whereas a reduced thyroid activity in thermal acclimated cattle has been reported by Gale. This decline in thyroid hormones along with decreased plasma GH level has a synergistic effect to reduce heat production.

Acute and chronic thermal stress shows differing responses on glucocorticoid concentrations, being elevated in former but not in later. Alvarez and Johnson reported an increase in glucocorticoids level from 2.4 to 3.9 µg/100 ml (62%) by the second hour of heat exposure, reached a peak of 5.4 µg/100 ml (120%) at 4 hours, then declined gradually to the normal of 2.4µg/100 ml at 48 hours and stayed at this level inspite of continued heat stimulus. The initial rise in plasma glucocorticoids is due to activation of the adrenocorticotropin (ACTH) releasing mechanism in the hypothalamus by thermoceptors of the skin whereas the later decline to normal, inspite of continuing heat stimulus, indicates a negative glucocorticoid

feedback and a decrease in the glucocorticoid binding transcortin . The glucocorticoids work as vasodilators proteolysis and lipolysis, hence, providing energy to the animal to help offset the reduction of intake.

In the bovine species, the simultaneous relationship among thermal stress, plasma aldosterone concentration and urine electrolyte excretion has been documented (El-Nouty *et al*.,1980). Plasma aldosterone concentration was the same during the first few hours of heat exposure however, with prolonged exposure; it was 40% lower and declined rapidly during later hours of exposure. This decline in aldosterone concentration is due to a fall in serum K levels because of its increased excretion in sweat and is explained on the basis of a major difference between ruminants and nonruminants with respect to location of Na and K loss during thermal stress. Nonruminants produce sweat high in Na and low in K concentrations; but this is vice versa for ruminants.

The concentration of catecholamines is elevated during both acute and chronic thermal stress. Alvarez and Johnson (1973) have reported an average increase of 45 and 42% in short and 91 and 70% in long heat exposures for epinephrine and norepine-phrene, respectively. Allen and Bligh (1969) have reported that catecholamines activate sweat glands of cattle and are involved in regulating sweat gland activity. Data regarding the catecholamines is still lacking An increase in plasma osmolality, or a decrease in blood volume, leads to ADH secretion from the pituitary gland; which in turn acts on the kidneys to retain water. Increased water losses in the respiratory tract and at the skin of thermal-stressed animal invokes increased secretion of ADH which is associated with a need to conserve water and increase water intake.

**Biochemical changes**:

Oxidative stress resulting from increased production of free radicals and reactive oxygen species (ROS), increases the antioxidant defence. Antioxidants are those nutrients which are required to cleanse cells of ROS. Vitamin E, vitamin A and vitamin C are classic examples of antioxidants. Antioxidant in low concentrations significantly delays or inhibits oxidation of oxidizable substrates. Following antioxidants got impaired during heat stress such as Enzymatic (SOD, CAT, GPX), Non-enzymatic (Albumin, L-cysteine, homocysteine and Protein sulfhydryl groups) and Non-enzymatic low molecular weight antioxidants (ascorbic acid, glutathione, uric acid α-tocopherol, β-carotene and retinol) (Kumar *et al.,*2007).Tissue defence mechanisms against free-radical damage generally include vitamin C, vitamin E, and βcarotene as the major vitamin antioxidant sources (Bernabucci *et al.,*2002). In addition,

several metalloenzymes which include glutathione peroxidase (Se), catalase (Fe) and superoxide dismutase (Cu, Zn, and Mn) are also critical in protecting the internal cellular constituents from oxidative damage (Weiss, 2006).

**Conclusion**

Heat stress is a cause of great concern among livestock owners in tropical countries. Adaptation to heat stress is a homeorhetic process which involves changes in nutrient acquisition and metabolism, hormonal signals which affect responsiveness of target tissues to environmental stimuli and imbalances in acid-base chemistry of the animal. These alterations are same in *Bos taurus* and *Bos indicus* breeds of cattle but are less pronounced in *Bos indicus* making it better adaptive to heat stress. Hence the consequences of heat exposure on production of milk and meat are also less for *Bos indicus.* Shelter management alone cannot combat heat stress in livestock. Dietary supplementation of salts and exogeneous antioxidants should be tried to cope up with heat stress.

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