

REVIEW

**EFFECT OF HEAT STRESS IN TROPICAL LIVESTOCK AND
DIFFERENT STRATEGIES FOR ITS AMELIORATION**

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Stress is a broad term, generally used in negative connotation and is described as the cumulative detrimental effect of a variety of factors on the health and performance of animals. Heat stress occurs in animals when there is an imbalance between heat production within the body and its dissipation. Heat stress is one of the wide varieties of factors which causes oxidative stress *in-vivo*. Reactive oxygen species (ROS), the major culprits for causing oxidative stress, are constantly generated *in vivo* as an integral part of metabolism. ROS may cause oxidative stress when their level exceeds the threshold value. They trigger progressive destruction of polyunsaturated fatty acids (PUFA), ultimately leading to membrane destruction. Body employs antioxidants to quench these free radicals. The enzymatic antioxidants like superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) act by scavenging both intracellular and extracellular superoxide radical and preventing lipid peroxidation of plasma membrane. Non-enzymatic antioxidants include vitamins like vitamins C, A and E, proteins like albumin, transferrin, glutathione (GSH) etc. Antioxidant nutrient supplementation especially vitamins C, A and E, zinc and chromium can be used to attenuate the negative effects of environmental stress.

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Simply defined, thermoregulation is the means by which animal maintains its body temperature. It involves a balance between heat gain and heat loss. Metabolic heat includes that necessary for maintenance plus increments for exercise, growth,

lactation, gestation and feeding. High rates of these activities will result in more heat gain from metabolism. In addition to the heat gained from metabolism, heat is also gained from environment. Stress is a condition which arises when an animal

suddenly faces a change in its environment. It may occur due to a variety of factors, temperature being one of them. Increased ambient temperature may lead to enhanced heat gain as compared to heat loss from the body and may cause heat stress in animals.

Effect of heat stress on animals

Under heat stress, a number of physiological and behavioral responses vary in intensity and duration in relation to the animal genetic make up and environmental factors. Climatic, environmental, nutritional, physical, social or physiological stressors are likely to reduce welfare and performance of animals (Freeman, 1987). Heat stress is one of the most important stressors especially in hot regions of the world. Adaptation to heat stress requires the physiological integration of many organs and systems viz. endocrine, cardiorespiratory and immune system (Altan *et al.*, 2003). The heterophil/lymphocyte (H/L) ratio has been accepted as a reliable index for determining stress in animals which is found to increase as a result of stress (Gross and Siegel, 1983; Mc Farlane and Curtis, 1989).

Heat stress reduces libido, fertility and embryonic survival in animals. Primary effect of environmental stress in neonates is increased disease incidence associated with reduced immunoglobulin content in plasma. Heat stress in late gestation reduces fetal growth and alters endocrine status of the dam. Carryover effects of heat stress during late gestation on postpartum lactation and reproduction are also detectable (Collier *et al.*, 1982).

Heat stress in livestock in tropical countries

A major part of our country is characterized as humid tropic and is subjected to extended periods of high ambient temperature and humidity. The primary non-evaporative means of cooling (viz. conduction, convection and radiation) becomes less

effective with rising ambient temperature and hence under such conditions, an animal becomes increasingly reliant upon evaporative cooling in the form of sweating and panting to alleviate heat stress (Kimothi and Ghosh, 2005). Thermal stress lowers feed intake of animal which in turn reduces their productivity in terms of milk yield, body weight and reproductive performance.

Changes during heat stress

Metabolic changes

High ambient temperature can adversely affect the structure and physiology of cells causing impaired transcription, RNA processing, translation, oxidative metabolism, membrane structure and function (Iwagami, 1996). Cells generate small amounts of free radicals or reactive oxygen species (ROS) during their normal metabolism. Although low levels of ROS are essential in many biochemical processes, accumulation of ROS may damage biological macromolecules i.e. lipids, proteins, carbohydrates and DNA (Mates *et al.*, 1999). External factors such as heat, trauma, ultrasound, infections, radiations, toxins etc. can lead to increased free radicals and other ROS and may lead to oxidative stress (Halliwell *et al.*, 1992).

Altan *et al.*, 2003 have demonstrated that heat stress increased lipid peroxidation which was associated with production of large number of free radicals which are capable of initiating peroxidation of polyunsaturated fatty acids. Ralhan *et al.* (2004), also reported that lipid peroxidation is significantly increased during reticulo-ruminal impaction in buffaloes. Heat stress may lead to increased production of transition metal ions (TMI), which can make electron donations to oxygen forming superoxide or H₂O₂ which is further reduced to an extremely reactive OH radical causing oxidative stress (Agarwal and Prabhakaran, 2005).

Antioxidants, both enzymatic (viz. superoxide dismutase, glutathione peroxidase & catalase) and nonenzymatic (vitamins C, E and A, glutathione, pyruvate etc) provide necessary defence against oxidative stress generated due to high ambient temperature. Catalase detoxifies H_2O_2 produced during different metabolic processes and also in stressful conditions by reducing it to H_2O and O_2 (Fridovich, 1978). Superoxide dismutase (SOD) in conjugation with catalase and glutathione peroxidase (GPx) scavenges both intracellular and extracellular superoxide radicals and prevents lipid peroxidation (Agarwal and Prabhakaran, 2005). GPx reacts with peroxides and requires glutathione (GSH) as the reductive substance donating an electron. GSH reduces oxygen toxicity by preventing O_2^- formation (Yoda *et al.*, 1986). Rampal *et al.*, (2002) reported that catalase activity is reduced in oxydementon-methyl induced oxidative stress in buffaloes. Sharma *et al* (2004) also reported similar findings in molybdenum induced oxidative stress in crossbred calves. Heat stress in lactating animals results in dramatic reduction in roughage intake, gut motility and rumination which in turn contribute to decreased volatile fatty acid production and may contribute to alteration in acetate: propionate ratio. Rumen pH also declines during thermal stress (Collier *et al.*, 1982). Electrolyte concentrations, in particular Na^+ and K^+ are reduced in rumen fluid of heat stressed cattle. The decrease in Na^+ and K^+ are related to increase in loss of urinary Na^+ and loss of skin K^+ as well as decline in plasma aldosterone and increase in plasma prolactin (Collier *et al.*, 1982). Enhanced heat dissipation during heat stress may also lead to electrolyte losses through sweat, saliva, polypnea and urine. This may lead to fall in plasma Na^+ , K^+ and Cl^- concentration (Coppock *et al.*, 1982). Scheneider *et al.* (1984) reported that heat stress in

lactating dairy cows caused significant loss of serum Na^+ and K^+ . West (1999) also reported similar fall in serum electrolyte concentration in dairy cows subjected to heat stress. Dale and Brody, (1954) suggested that a heat stressed animal, particularly a lactating cow, might experience metabolic ketosis as energy input would not satisfy energy need and thus accelerate body fat catabolism accumulating ketone bodies if they are not rapidly excreted. These ketone bodies deplete blood alkali reserves, possibly potentiating respiratory alkalosis. Thermal stress alters dietary protein utilization and body protein metabolism (Ames *et al.*, 1980).

Hormonal changes

It has been recognized that certain environmental stressors have the potential to activate the hypothalamo-pituitary-adrenal cortical axis (HPA) and sympatho-adrenal medullary axis (Minton,1994). There is increase in plasma concentration of cortisol and corticosterone and less frequently an increase in plasma epinephrine and nor epinephrine concentration in heat stressed animals (Minton,1994). Magdub *et al.*, (1982) reported that during heat stress there were significant reduction in concentrations of triiodothyronine (T_3) and thyroxine (T_4) in plasma and in milk of lactating cows. However, a significant increase in T_3 but not in T_4 level was observed during heat stress in cross bred cattle (Singh *et al*, 1984). Collier *et al.*, (1982) reported that thermal stress reduced birth weights of Holstein calves. Reduced birth weight of calves was associated with lower concentrations of estrone sulfate in plasma of heat stressed animals. Because estrone sulfate is produced by the gravid uterus and conceptus, its reduction indicates reduced conceptus function during thermal stress. Concentration of progesterone in plasma was also reported to elevate in heat stressed cycling cows by the same team

workers. During short term exposure to high ambient temperature, the concentrations of glucocorticoids and catecholamines were found to be elevated (Thompson *et al.*, 1963). El Nouty *et al.*, (1980) documented the simultaneous relationship among thermal stress, plasma aldosterone level and urine electrolyte concentration in bovines. During prolonged heat exposure plasma aldosterone level was reported to decline. Concurrent with this, there were significant fall in serum and urinary K⁺. El Nouty *et al.*, (1980) also suggested that a fall in serum K⁺ depressed aldosterone secretion, which may also have reduced urinary K⁺ excretion. Wetterman and Tucker, (1974) reported an increase in plasma prolactin concentration during thermal stress in dairy cows. Alteration in prolactin secretion may be associated with altered metabolic state of heat stressed animals. One possibility is that prolactin is involved in meeting increased water and electrolyte demands of heat stressed animals.

Immunological changes

Cell mediated immunity

Since stressors have been associated with increased circulatory concentration of glucocorticoids, they also have been linked with decreased functioning of the cells of the immune system. Blecha *et al.* (1984) found that when cattle were exposed to stressful conditions, lymphocyte proliferative responses to concanavalin A (Con A) were reduced. High ambient temperature causes functional and metabolic alterations in cells and tissues including cells of immune system. In such conditions, the administration of antioxidants has proved useful for improvement of several immune functions (Victor *et al.*, 1999). The immune cell functions are associated with production of ROS such as that involved in the microbial activity of

phagocytes or lymphoproliferative response to mitogens (Goldstone and Hunt, 1997). However excessive production of ROS due to heat stress renders harmful effect on cells of immune system. When exposed to oxidative stress, polymorphonuclear leukocytes (PMNs) change their pattern of oxygen uptake sharply while releasing large amounts of superoxide anion into the cell environment. PMNs play an important role as mediators of tissue destructive events in inflammatory diseases, ranging from rheumatoid arthritis and myocardial reperfusion injury to respiratory distress syndrome (Sharma *et al.*, 2002).

Humoral immunity

Heat stress reduced serum IgG₁ in calves associated with an increased cortisol concentration (Stott *et al.*, 1976), and extreme cold stress also reduced colostral immunoglobulin transfer (Olsen *et al.*, 1980). Thus, environmental extremes can influence disease resistance in dairy calves.

Strategies for ameliorating heat stress

The effects of heat stress are costly to dairy farmers, but there are opportunities to recover some of the losses due to hot weather. Physical modifications of environment, genetic development of breeds that are less sensitive to heat and nutritional management are the three major key components to sustain production in hot environment (Beede and Collier, 1986).

Shelter management

With the help of managerial tools, it is possible to modify the microenvironment to enhance heat dissipation mechanism to relieve heat stress. Sheds if constructed scientifically, provide comfortable environment to animals. There is no doubt that shading is one of the cheapest ways to modify an animal's environment during hot weather. Although shade reduces heat accumulation, there is

no effect on air temperature or relative humidity and additional cooling is necessary for farm animals in a hot humid climate (Kimothi and Ghosh, 2005). Cooling ponds and sprinklers can also be used to cool the environment but none has been proved efficient.

Genetic modification

There is genetic variation among animals for cooling capability, which suggests that more heat tolerant animals can be selected genetically. Cross breeding offers another opportunity (Kimothi and Ghosh, 2005). However, extensive crossbreeding studies have shown little heterosis for heat tolerance (Branton *et al.*, 1979). Additional studies are needed to examine variability in heat tolerance of high yielding animals and. Possibly improved herds could be developed when selected for milk yield and heat tolerance under local conditions.

Nutritional management

Oxidative damage, as a result of heat stress may be minimized by antioxidant defense mechanisms that protect the cells against cellular oxidants and repair system that prevent the accumulation of oxidatively damaged molecules. Antioxidants, both enzymatic and non-enzymatic, provide necessary defense against oxidative stress as a result of thermal stress.

Non enzymatic antioxidants in reducing oxidative stress

Vitamins

Both vitamin C and vitamin E have antioxidant properties. Antioxidant vitamins have proved to protect the biological membranes against the damage of ROS and the role of vitamin E as an inhibitor –“chain blocker”- of lipid peroxidation has been well established (Seyrek *et al.*, 2004). The effect of vitamin E was studied on monosodium

glutamate induced hepatotoxicity and oxidative stress in rats by Onyema *et al.*,(2006). They successfully demonstrated the ameliorative effect of vitamin E on stressed rats. Like vitamin E, ascorbate is also a chain breaking antioxidant. It prevents lipid peroxidation due to peroxy radicals. It also recycles vitamin E. It protects against DNA damage induced by H₂O₂ radical. Vitamin C has a paradoxical effect as it can also produce ROS by its action on transition metal ions (Lutsenko *et al.*, 2002). Both ascorbate and zinc are known to scavenge reactive oxygen species (ROS) during oxidative stress (Prasad, 1979). Frey (1991), reported that vitamin C has an ability to spare other antioxidants in relieving oxidative stress in human subjects. Ramachandran *et al.*,(2002) demonstrated the effect of dietary component viz vitamin C, E & β-carotene in relieving oxidative stress in rats by measuring the activities of antioxidant enzymes in liver and kidney .They found the effect to be more pronounced in the liver than in kidney. Vitamin C was found to assist in absorption of folic acid by reducing it to tetrahydrofolate, the latter again acts as an antioxidant. Use of folic acid is impaired when vitamin C is deficient. Maneesh *et al.* (2005) reported that oral administration of ascorbic acid restores the androgenic and gametogenic activity of ethanol treated rats. Vitamin C along with electrolyte supplementation was found to ameliorate the heat stress in buffaloes (Sunil Kumar *et al.*, 2010).

Minerals and trace elements

Zinc and other trace elements like Cu and Cr act as typical antioxidants as they work indirectly. Zinc is a catalytic cofactor for Cu/Zn SOD and catalyzes dismutation of superoxide anion, producing molecular oxygen and H₂O₂, the latter product is usually metabolized by GPx and CAT. Several

reports have shown the impact of Cu and Zn deficiency on the antioxidant defence system and oxidative damage to cellular components (Picco *et al.*, 2004). The activity of Cu/Zn SOD, CAT and GPx is decreased in Cu deficient animals. It is also reported that normal Cu levels are necessary to maintain the structural integrity of DNA during oxidative stress. Supplementation of electrolytes is one among the nutritional strategies to combat heat stress in animals. Addition of Na⁺, K⁺ and Cl⁻ is benefited in heat stressed dairy cows in terms of milk yield, acid base balance and lower temperature (Coppock *et al.*, 1982). West *et al.* (1999) reported that Na⁺ and K⁺ status of the body stayed normal during heat stress when supplemented with electrolytes. Supplementation of sodium and potassium in the form of bicarbonate/carbonate also help in better regulation of acid-base balance in the blood (Sanchez *et al.*, 1994).

Amelioration through immunomodulation by dietary supplement

The immunostimulant effect of antioxidant depends on age and immune state of organisms as well as on the kind of immune function studied (Victor *et al.*, 1999). The effect of heat stress can be neutralized by complex antioxidant system that an organism develops (Mac Arthur, 2000). The antioxidant system can be boosted by supplementing antioxidants in diet. Vitamin C and trace minerals like zinc have proved to play a vital role as modulators of antibody response and enhances wound healing in domestic animals. (Vegad and Katiyar, 1995). Bhar *et al.* (2003) conducted an experiment to study the effect of dietary supplementation of vitamin C along with zinc on the wound healing rate, antibody response and growth performance in castrated domestic pigs. The antibody response and wound healing rate were

found to be good to high in groups supplemented with vitamin C and zinc. Studies have shown that supplementation of vitamins C, E & A and zinc are effective in preventing the negative effect of environmental stress (Mac Dowell, 1989).

Conclusion

Heat stress is a cause of great concern among livestock owners in tropical countries. It causes change in the antioxidant level and electrolyte concentration but increases lipid peroxidation *in vivo*. Cell mediated immune response is also decreased due to heat stress. Shelter management alone cannot combat heat stress in livestock. Dietary supplementation of salts and exogenous antioxidants should be tried to cope up with heat stress.

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