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Impact of heat stress on tropical livestock and various approaches for mitigation

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Abstract

Stress, often viewed negatively, encompasses various factors that can adversely affect the health and performance of animals. Heat stress arises when there's an imbalance between the body's heat production and its dissipation, contributing to oxidative stress. Reactive oxygen species (ROS), generated as a natural byproduct of metabolism, can lead to oxidative stress if their levels exceed a certain threshold. This can result in the gradual degradation of polyunsaturated fatty acids (PUFA), ultimately damaging cell membranes. The body employs antioxidants to counteract these free radicals. Enzymatic antioxidants such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) scavenge intracellular and extracellular superoxide radicals, preventing lipid peroxidation of cell membranes. Non-enzymatic antioxidants, including vitamins C, A, and E, as well as proteins like albumin and glutathione (GSH), also play a crucial role. Supplementation with antioxidant nutrients, particularly vitamins C, A, and E, zinc, and chromium, can mitigate the adverse effects of environmental stressors.

Keywords: Heat stress, tropical livestock, superoxide dismutase, catalase

Introduction

Thermoregulation is the process through which animals regulate their body temperature, maintaining a balance between heat gain and heat loss. Metabolic heat, which includes energy required for basic bodily functions as well as additional needs like exercise, growth, lactation, gestation, and feeding, contributes to heat gain. Higher rates of these activities result in increased heat production. Besides metabolic heat, animals also gain heat from their environment. Stress occurs when animals experience a sudden change in their environment, which can be triggered by various factors, including temperature fluctuations. Elevated ambient temperatures can lead to greater heat gain compared to heat loss from the body, potentially inducing heat stress in animals.

Effect of heat stress on animals

Heat stress induces a range of physiological and behavioral responses in animals, which vary in intensity and duration depending on genetic makeup and environmental factors. Various stressors, including climatic, environmental, nutritional, physical, social, or physiological factors, can diminish animal welfare and performance. Particularly in hot regions, heat stress is a significant stressor. Adapting to heat stress requires coordinated physiological responses from multiple organ systems, including the endocrine, cardio-respiratory, and immune systems (Freeman, 1987 and Altan *et al*., 2003) [6, 1].

The heterophil/lymphocyte (H/L) ratio serves as a reliable indicator of stress in animals, as it tends to increase in response to stressors. Heat stress adversely affects libido, fertility, and embryonic survival in animals. Neonates exposed to environmental stress experience higher disease incidence due to reduced immunoglobulin content in plasma (Gross and Siegel, 1983; Mc Farlane and Curtis, 1989) [8, 14]. During late gestation, heat stress reduces fetal growth and alters the endocrine status of the dam. Furthermore, the repercussions of heat stress during late gestation extend to postpartum lactation and reproduction, indicating carryover effects (Collier *et al*., 1982) [4].

Heat stress in livestock in tropical countries

In the humid tropics, high temperatures and humidity reduce the effectiveness of non-evaporative cooling methods like conduction, convection, and radiation. Animals rely more on evaporative cooling, such as sweating and panting, to alleviate heat stress (Kimothi and Ghosh, 2005) [9]. Thermal stress lowers feed intake, reducing productivity in terms of milk yield, body weight, and reproductive performance.

Strategies for ameliorating heat stress

Shelter management

Management tools can modify the microenvironment to enhance heat dissipation and relieve heat stress in animals. Scientifically constructed sheds provide a comfortable environment, with shading being a cost-effective method to reduce heat accumulation. However, shade alone does not affect air temperature or humidity, necessitating additional cooling in hot, humid climates (Kimothi and Ghosh, 2005) [9]. Cooling ponds and sprinklers can also be used, though their efficiency is limited.

Genetic modification

There is genetic variation among animals in their cooling

capability, indicating that animals with greater heat tolerance can be selected through genetic means. Crossbreeding presents another opportunity for enhancing heat tolerance (Kimothi and Ghosh, 2005) [9]. However, extensive crossbreeding studies have demonstrated minimal heterosis for heat tolerance (Branton *et al*., 1979) [3]. Further research is necessary to examine the variability in heat tolerance among high-yielding animals. It is possible that improved herds could be developed by selecting for both milk yield and heat tolerance under local conditions.

Nutritional management

Oxidative damage caused by heat stress can be minimized by antioxidant defense mechanisms, which protect cells against cellular oxidants and employ repair systems to prevent the accumulation of oxidatively damaged molecules. Both enzymatic and non-enzymatic antioxidants play a crucial role in providing necessary defense against oxidative stress induced by thermal stress.

Non enzymatic antioxidants in reducing oxidative stress

Vitamins

Both vitamin C and vitamin E possess antioxidant properties that protect biological membranes against damage from reactive oxygen species (ROS). Vitamin E acts as an inhibitor of lipid peroxidation (Seyrek *et al*., 2004) [17], and has been shown to reduce hepatotoxicity and oxidative stress in rats (Onyema *et al*., 2006) [18]. Similarly, vitamin C is a chain-breaking antioxidant that prevents lipid peroxidation and recycles vitamin E. It also protects against DNA damage from H2O2 radicals but can produce ROS in the presence of transition metal ions (Lutsenko *et al*., 2002) [10]. Both ascorbate and zinc scavenge ROS during oxidative stress (Prasad, 1979) [20], and vitamin C can spare other antioxidants (Frey, 1991) [7]. Ramachandran *et al*. (2002) [15] found that vitamins C, E, and β-carotene alleviate oxidative stress in rats, particularly in the liver. Vitamin C aids in the absorption of folic acid by reducing it to tetrahydrofolate, and its deficiency impairs folic acid use. Additionally, ascorbic acid can restore androgenic and gametogenic activity in ethanol-treated rats (Maneesh *et al*., 2005) [13] and, along with electrolyte supplementation, reduce heat stress in buffaloes (Sunil Kumar *et al*., 2010) [21].

Minerals and trace elements

Zinc and other trace elements like copper (Cu) and chromium (Cr) function as indirect antioxidants. Zinc acts as a catalytic cofactor for Cu/Zn superoxide dismutase (SOD), catalyzing the dismutation of superoxide anion into molecular oxygen and hydrogen peroxide (H2O2), which is then metabolized by glutathione peroxidase (GPx) and catalase (CAT). Deficiencies in Cu and Zn negatively impact the antioxidant defense system and increase oxidative damage (Picco *et al*., 2004) [19]. Cu deficiency specifically reduces the activity of Cu/Zn SOD, CAT, and GPx, and normal Cu levels are necessary for maintaining DNA integrity during oxidative stress.

Supplementation with electrolytes, including sodium (Na+), potassium (K+), and chloride (Cl-), helps combat heat stress in animals. This practice benefits heat-stressed dairy cows by improving milk yield, acid-base balance, and lowering body temperature (Coppock *et al*., 1982) [5]. West *et al*. (1999) [24] found that Na+ and K+ levels remained normal during heat stress with electrolyte supplementation. Sodium and potassium bicarbonate/carbonate supplementation also aids in regulating blood acid-base balance (Sanchez *et al*., 1994) [16].

Amelioration through immunomodulation by dietary supplement

The immune-stimulant effects of antioxidants are influenced by factors such as age, immune status of organisms, and the specific immune functions studied (Victor *et al*., 1999) [23]. Heat stress can be mitigated by an organism's complex antioxidant system (Mac Arthur, 2000) [11], which can be enhanced through dietary supplementation of antioxidants. Vitamins, particularly Vitamin C, and trace minerals such as zinc, are recognized for their significant roles as modulators of antibody responses and enhancers of wound healing in domestic animals (Vegad and Katiyar, 1995) [22]. Bhar *et al*. (2003) [2] investigated the impact of dietary supplementation with Vitamin C and zinc on wound healing rate, antibody response, and growth performance in castrated domestic pigs. Their findings indicated that groups receiving Vitamin C and zinc supplementation exhibited improved antibody responses and wound healing rates. Furthermore, supplementation with vitamins C, E, A, and zinc has been shown to be effective in counteracting the adverse effects of environmental stress (Mac Dowell, 1989) [12].

Conclusion

Heat stress poses a significant challenge for livestock owners in tropical regions, leading to alterations in antioxidant levels and electrolyte concentrations while increasing *in vivo* lipid per-oxidation. Additionally, heat stress suppresses cell-mediated immune responses. Simply relying on shelter management is insufficient to mitigate heat stress in livestock. Dietary supplementation with salts and exogenous antioxidants should be considered as strategies to address heat stress.

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