INTAS ANIMAL HEALTH

For Vet Professionals Only

Face2Vet
A Clinical Update

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Summer Stress and it's Management

Summer Stress, Dairy Distress

Dear Vets,

Summer stress is a major environmental constraint affecting the health, welfare and productivity of dairy animals, particularly in tropical and subtropical regions. Dairy animals are highly susceptible to climatic stressors such as elevated ambient temperature, intense solar radiation, high relative humidity and reduced wind velocity, which collectively disturb their thermoregulatory balance.

Dairy animals operate efficiently within a narrow thermoneutral zone; however, when environmental conditions exceed this range, their capacity to dissipate metabolic heat becomes compromised. High producing dairy animals are vulnerable due to increased endogenous heat production associated with ruminal fermentation and milk synthesis. Exposure to excessive thermal load activates a series of behavioural, physiological, cardiovascular and endocrine responses aimed at maintaining normothermia. When these adaptive mechanisms fail, hyperthermia develops, resulting in profound alterations in metabolic function, immune competence, milk production and reproductive efficiency.

Given the increasing prevalence of heat stress under changing climatic conditions, a comprehensive understanding of thermoregulatory mechanisms, endocrine adaptations, associated production and reproductive consequences and effective management strategies are essential for sustaining ruminant health and optimising dairy production systems.

This Face2Vet focuses on the summer stress and its management in dairy animals. We invite you to read this issue and share your valuable experiences and insights by scanning the below QR code or by contacting us *via* email at face2vet@intaspharma.com.

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Introduction

Stress is a reflexive biological response of animals to adverse environmental conditions, and its consequences may range from mild discomfort to severe physiological disturbances and even death. Among the various forms of environmental stress, summer or heat stress (HS) has become one of the most serious challenges threatening animal welfare and the sustainability of livestock production systems worldwide. This threat is particularly pronounced in tropical and subtropical regions, such as India, where rising ambient temperatures associated with climate change are increasingly common.

Dairy animals maintain normal physiological functions within a defined thermoneutral zone (TNZ), generally ranging from 16°C to 25°C, during which core body temperature is maintained between 38.4°C and 39.1°C. However, when ambient temperatures exceed this comfort range, the animal's ability to dissipate metabolic heat becomes limited, resulting in heat stress. Under such conditions, animals exhibit physiological and behavioural alterations, including increased body surface temperature, respiration rate, heart rate and rectal temperature. These responses negatively affect feed intake, milk production, reproductive efficiency and immune competence, ultimately increasing susceptibility to diseases.

Despite significant advances in housing design, management practices and cooling technologies, heat stress continues to impose substantial economic losses and welfare concerns in livestock production systems. As global temperatures continue to rise, understanding the physiological basis, indicators and adaptive mechanisms of heat stress, as well as its management and control strategies are essential for developing sustainable and resilient dairy production systems.

Etiology

Environmental Factors

- Environmental conditions are widely recognised as the primary etiological determinants of heat stress in farm animals.
- Among these, elevated ambient temperature is the most critical factor, particularly when it exceeds the species-specific upper limit of TNZ (Fig. 1).

- Severity of heat stress amplified when elevated relative humidity markedly reduces the efficiency of evaporative heat loss through sweating and panting, resulting in the accumulation of excess body heat.
- Solar radiation substantially increases radiant heat gain, especially in animals exposed to direct sunlight.
- Low air movement or poor wind flow reduces heat loss from the animal's body, leading to increased heat stress.
- The interaction of ambient temperature, relative humidity, wind speed and solar radiation collectively determines the intensity of heat stress experienced by livestock (Fig. 2).
- Prolonged or repeated exposure to such adverse climatic conditions results in chronic thermal strain.
- This persistent burden compromises animal welfare by disrupting physiological functions, impairing productivity and reproductive efficiency and increasing susceptibility to disease.

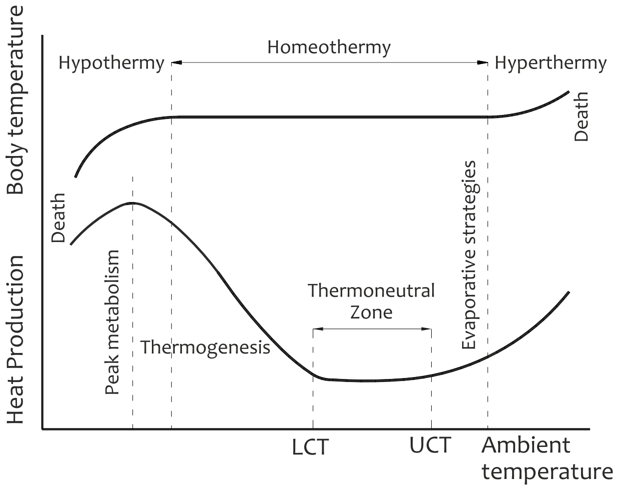

Fig. 1: Effect of ambient temperature on animal metabolism and body temperature (Sanz, 2018)
LCT: lower critical temperature;
UCT: upper critical temperature

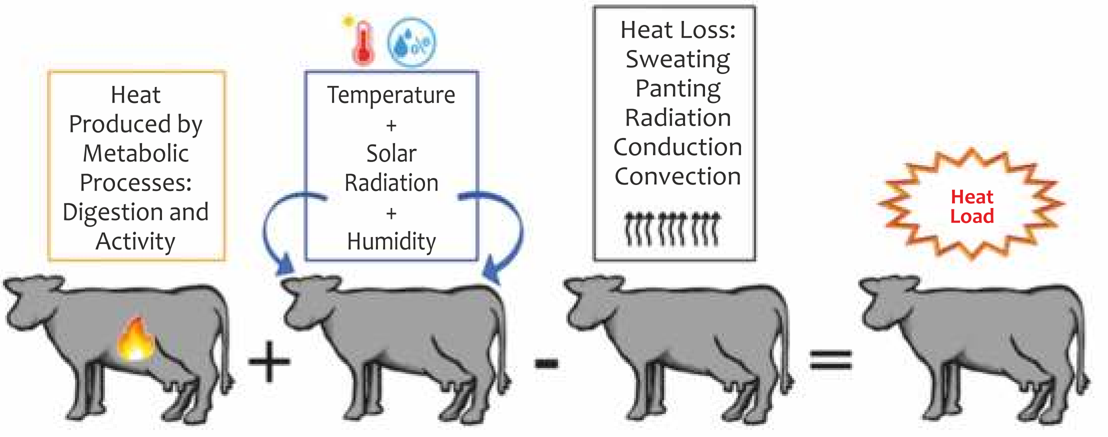

Fig. 2: Summer stress in dairy animals (Toledo *et al.*, 2019)

Animal Related Factors

- Being homeothermic, farm animals possess inherent thermoregulatory mechanisms that allow them to withstand heat stress to a certain extent.

Table 1: Factors affecting dairy animal's susceptibility to heat stress

Factor	More Prone to Heat Stress	Less Prone to Heat Stress
Breed	Exotic / crossbred cows	Indigenous (local) breeds
Parity (age)	Older cows	Younger cows
Milk production level	High-producing cows	Low-producing cows
Stage of lactation	Early lactation	Late lactation / dry cows
Feed intake level	High feed intake	Low to moderate feed intake
Ration composition	High in fibre and crude protein	Balanced ration
Body condition score	High body condition (fat)	Low to moderate body condition
Behaviour	Active / aggressive cows	Calm cows
Housing conditions	Poorly ventilated housing	Well-ventilated housing
Pasturing conditions	Non-shaded pasture	Shaded pasture

Pathogenesis

Heat stress in dairy animals develops gradually. It starts with normal protective body responses to heat, but if heat exposure is severe and lasts for a long time, it can progress to serious illness and even death. This condition passes through four overlapping stages, during which responses that are helpful at first may later become harmful (Fig. 3).

Stage 1

Adaptive Responses

- Reduced feed intake
- Altered feeding behaviour (night feeding)
- Increased standing and bunching behaviour
- Increased water intake
- Sweating and panting
- Peripheral vasodilation (blood to skin)
  (Protective but initiates electrolyte loss & visceral hypoperfusion)

Stage 2

Organ Dysfunction and Endotoxemia

- Prolonged hyperthermia
- GI hypoxia and epithelial damage
- Increased gut permeability (leaky gut)
- Endotoxin (lipopolysaccharide) translocation
- Loss of saliva → Leads to ↓ sodium and bicarbonate
- ↓ rumen pH → ruminal acidosis
- Liver detoxification overwhelmed
- Renal hypoperfusion and injury → renal dysfunction

Stage 3

Systemic Inflammatory Response

- Endotoxemia
- Systemic inflammatory response syndrome (SIRS)
- Cytokine storm
- Oxidative stress (ROS, RNS)
- Endothelia injury →Coagulation dysfunction →Disseminated Intravascular coagulation
- Tissue ischemia

Stage 4

Multiple Organ Failure and Death

- Progressive inflammation, oxidative damage, coagulation failure and hypoxia
- Multiple organ dysfunction syndrome (MODS)
- Acute respiratory distress syndrome (ARDS)
- Septic shock
- Circulatory failure, reduced cardiac output, severe hypotension and metabolic collapse
- Death

Fig. 3: Pathogenesis of heat stress

Impact of Summer Stress

Summer stress in animals is commonly evaluated using the temperature–humidity index (THI). Owing to their high sensitivity to summer stress, animals experience a wide range of productive, reproductive and health related challenges during periods of elevated THI (Table 2). These stress induced impairments collectively lead to significant economic losses for dairy farmers worldwide.

Table 2: THI level and its effects

THI	Stress Level	Effects
<72	None	No observable stress: cows remain comfortable with normal feeding, production and reproduction.
72-79	Mild	Cows cope with mild stress by seeking shade, breathing faster and increasing blood flow to the skin. Milk production is generally not affected.
80-89	Moderate	Respiratory rate and saliva secretion increase noticeably. Feed intake decreases, water intake increases and body temperature rises. Milk yield and reproductive efficiency start to decline.
90–98	Severe	Cows experience severe discomfort due to very high body temperature, heavy panting and excessive salivation. Milk production and reproduction are significantly reduced.
>98	Danger	High risk of animal collapse and death.

The following formula is used to calculate temperature–humidity index (THI).

$$\mathbf{THI = (1.8 \times T + 32) - (0.55 - 0.0055 \times RH) \times (1.8 \times T - 26)}$$

Where

- **T** = Ambient temperature (°C)
- **RH** = Relative humidity (%)

Physiological Responses

Dairy animals are highly susceptible to heat stress due to increased metabolic heat production associated with high milk yield, and exposure beyond the thermoneutral zone triggers physiological and endocrine responses to maintain homeostasis.

Thermoregulatory and Cardiovascular Responses

- Increased heart rate promotes peripheral blood flow, aiding heat transfer from the core to the body surface.
- Activation of evaporative cooling mechanisms, such as panting and sweating, enhances heat loss but results in increased body fluid loss.
- Thus, animals increase water intake to maintain hydration.
- At high temperature and humidity, cooling mechanisms fail, leading to impaired heat dissipation and development of hyperthermia.
- To limit further heat production, animals reduce dry matter intake, which helps control body temperature but ultimately compromises productivity.

Digestive and Metabolic Consequences

- Reduced feed intake minimises metabolic heat load, but it can negatively affect gastrointestinal function.
- A decrease in rumen pH increases the risk of ruminal acidosis, potentially due to enhanced proliferation of lactic acid–producing bacteria.
- Reductions in DMI also impair intestinal morphology which compromise gut barrier integrity (leaky gut), facilitating translocation of pathogens and endotoxins across the intestinal epithelium and into systemic circulation (Fig. 4).
- Activation of the immune system following pathogen translocation diverts metabolic energy away from growth, reproduction and milk production toward immune defence mechanisms.

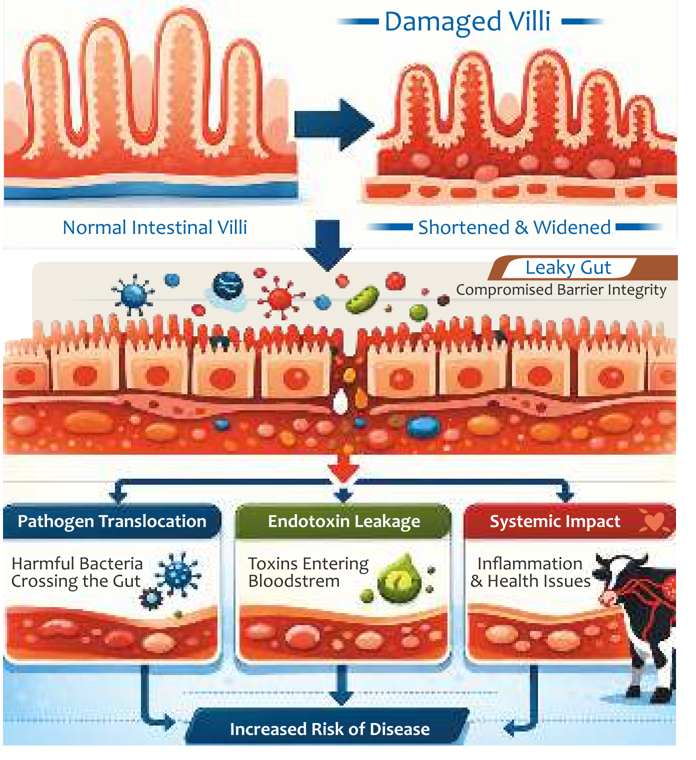

Fig. 4: Intestinal barrier dysfunction and its systemic consequences

Endocrine Adaptations

- Circulating thyroid hormone (T3 and T4) concentrations decline, leading to a reduction in basal metabolic rate and endogenous heat production.
- Acute heat stress increases prolactin and cortisol levels. Prolactin helps improve sweating and heat loss, while cortisol supports metabolism and immune regulation during stress.
- Chronic heat stress may lead to reduced or unchanged levels of cortisol, growth hormone and thyroxine due to prolonged water and electrolyte loss from continuous sweating and increased breathing, affecting normal hormone regulation.
- Additionally, chronic heat stress has been associated with increased progesterone concentrations, possibly due to reduced conversion of progesterone to cortisol, contributing further to suppressed circulating cortisol levels which leads to reduced stress adaptability.

Effect on Production

Summer stress has a substantial negative impact on milk production and represents a major source of economic loss for the dairy industry.

Biological Mechanisms Underlying Reduced Milk Production

- One major contributing factor is the reduction in dry matter intake (DMI), which limits the availability of energy and nutrients required to sustain normal milk synthesis. Studies have reported reductions in DMI ranging from 40 to 48% during heat stress, corresponding to decreases in milk yield of 25 to 53% compared with cows maintained under thermoneutral conditions. However, reductions in DMI alone do not fully explain the magnitude of milk yield losses observed during heat stress.

- Heat stress during late gestation increases mammary epithelial cell death and reduces cell proliferation.
- Impaired pre-partum mammary development can lower milk yield in the subsequent lactation.
- Heat stress also alters gene and microRNA expression in mammary gland, upregulating pathways require for cellular survival and downregulating pathways linked to milk synthesis.

Effects on Milk Composition and Quality

- Heat stress also negatively affects milk composition and quality.
- Numerous studies have demonstrated reductions in milk fat and protein yields during heat stress, along with increases in somatic cell count.
- The decline in milk components is largely attributed to reduced DMI; however, additional metabolic changes induced by heat stress also contribute to these effects.
- Altered amino acid metabolism is also limiting protein synthesis and reducing milk protein output (Fig. 5).

Summer stress

↓

Reduced feed intake and increased energy maintenance

↓

Metabolic and hormonal shifts (↑ Cortisol, ↓ Insulin sensitivity)

↓

Amino acid mobilization (Muscle proteolysis)

↓

Increased circulating animo acid

↓

Amino acids used as energy to support maintenance and thermoregulatory demands

↓

Reduced mammary uptake, fewer amino acid in mammary gland

↓

limiting protein synthesis and Reduced milk protein output

Fig. 5: Mechanisms underlying reduced milk protein

Effect on Reproduction

- Summer stress adversely affects reproductive performance in dairy animal across all stages.
- Heat stress markedly suppresses estrus expression, with up to 80% of heats going undetected despite normal ovulation.
- This is due to weakened behavioural and physiological signs such as reduced mounting activity and decreased cervical mucus.
- As a result, insemination timing becomes less precise, leading to more services per conception as THI increases.

Summer stress also directly impairs ovarian function by;

- Reducing luteinizing hormone and estradiol synthesis, impairing follicular development, causing ovarian inactivity and smaller follicles, and ultimately lowering conception rates.
- Beyond estrus and conception, it also compromises oocyte quality and early embryonic development.
- Disrupted progesterone levels hasten oocyte maturation and impair nuclear and cytoplasmic development, reducing oocyte competence and increasing fertilization failure.
- Elevated uterine temperature and reduced uterine blood flow further impair gamete viability and embryo survival.
- As a result, early embryonic growth, corpus luteum quality and embryonic attachment are negatively affected, leading to increased early embryonic mortality during periods of thermal stress (Fig. 6).

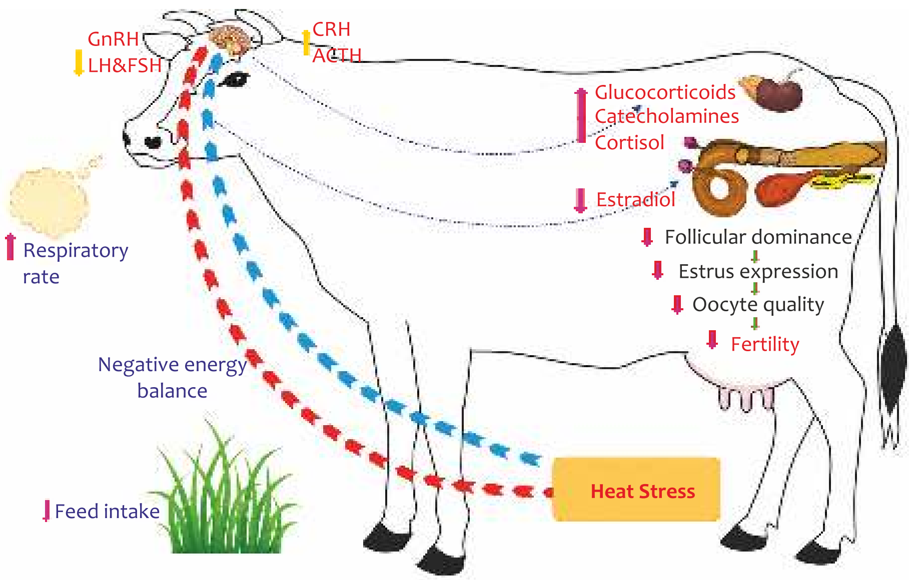

Fig. 6: Impact of heat stress on female reproductive performance
(Krishnan *et al.*, 2017)

Heat stress during late gestation has lasting consequences for both the dam and offspring.

- Chronic elevation of prolactin during heat stress induces downregulation of prolactin receptors in mammary tissue, reducing lactogenic signalling and epithelial cell proliferation.
- Calves born to heat-stressed dams exhibiting significantly lower birth weights, shortened gestation length and impaired placental function.

Effect on Health and Immune Response

In addition to its detrimental effects on productivity and reproduction, heat stress markedly compromises the health of dairy animal. Numerous studies have demonstrated increased disease incidence and mortality during periods of summer/heat stress.

Although the mechanisms underlying these health challenges are multifactorial, a major contributing factor is the profound effect of heat stress on function of immune system.

Effect on Adaptive Immune Responses

- In the adaptive immune system, heat stress disrupts the balance between T-helper 1 (Th1 - cell-mediated immunity) and T-helper 2 (Th2 - humoral immunity) immune responses, causing a shift toward Th2 dominance.
- This imbalance is partly mediated by cortisol, which interacts with DNA regulatory elements and suppresses the transcription of genes involved in Th1 activation and pro-inflammatory cytokine production.
- Consequently, the cell-mediated immune response (CMIR) which is crucial for defence against intracellular pathogens is impaired.

Effects on Immune Cell Function

Both innate and adaptive immune responses are adversely affected by heat stress.

- It has been consistently shown to reduce lymphocyte proliferation. Lymphocytes, including B and T cells, normally undergo rapid proliferation following pathogen exposure to mount an effective immune response.
- Reduced proliferative capacity limits immune cell multiplication and pathogen defence, thereby severely impairing the animal's ability to control infections and mount an effective immune response.
- In parallel, heat stress impairs neutrophil function, further weakening innate immune defences (Fig. 7).

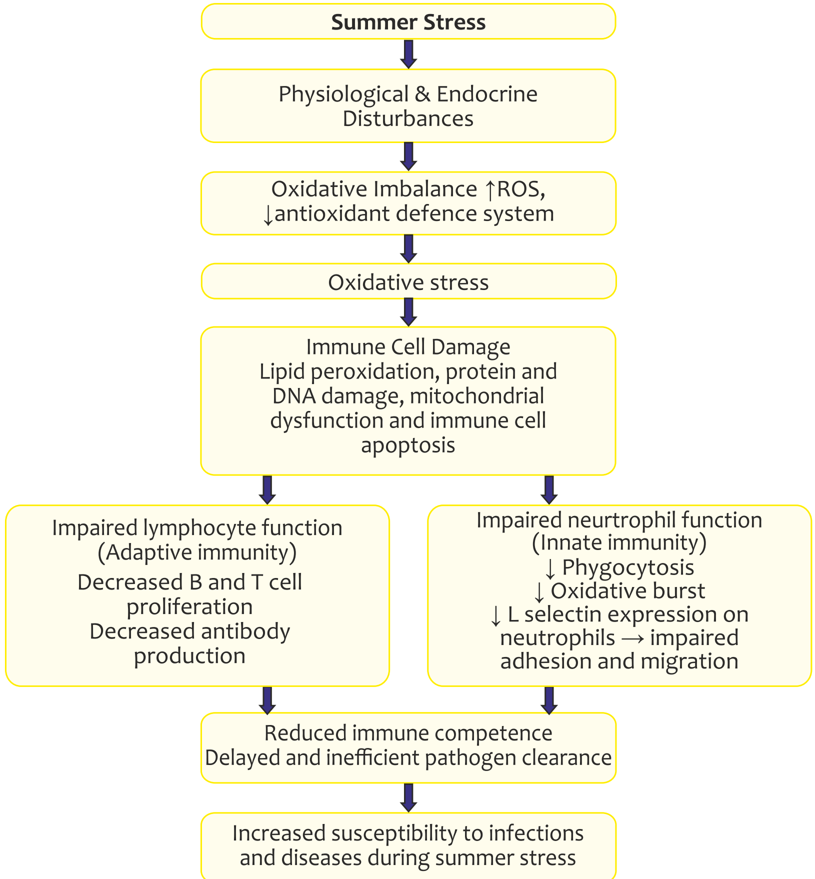

Fig. 7: Mechanisms of summer stress induced immune suppression

Preventive Measures and Management Strategies

Heat stress is a major constraint to livestock productivity, health and welfare, particularly in tropical and subtropical regions. Effective prevention and management require an integrated approach combining environmental modification and nutritional interventions.

Physical Modification of the Environment

- Environmental modification is the most immediate and effective approach for mitigating heat stress in hot climates.
- Provision of shade (natural or artificial) significantly reduces heat load from direct sunlight and improves animal comfort.
  - o Natural shade (trees) is highly effective due to combined shading and evaporative cooling from foliage (Fig. 8a).
  - o Artificial shade structures should use reflective materials (white galvanized or aluminium roofing) and provide adequate space per animal (Fig. 8b).

Fig. 8(a, b): Natural and artificial shades

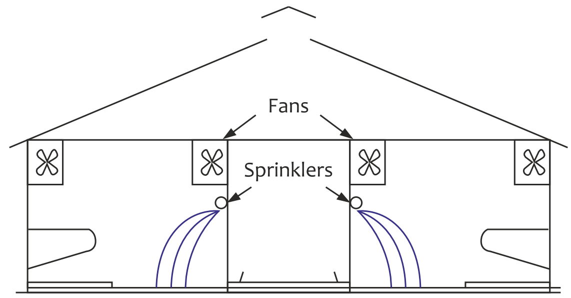

Fig. 9: Fans and sprinklers (Toledo *et al.*, 2019)

- Evaporative cooling systems, including fogging, misting, sprinklers and cooling ponds enhance heat dissipation, particularly when combined with natural or forced air movement (Fig. 9).
- Cooling systems have been shown to improve milk yield, reproductive performance and feed efficiency in dairy animals.

Nutritional Management

- Heat stress reduces dry matter intake (DMI), limiting nutrient availability and reducing nutrient utilisation efficiency.
- Diets should be carefully balanced to maintain homeostasis and prevent nutrient deficiencies.
- Diets should contain about 18% crude protein (DM basis) to support production under heat stress. Overfeeding protein increases heat production due to excess energy use in urea excretion.
- Optimizing rumen undegradable protein (rumen bypass protein) improves milk yield under heat stress conditions.
- Low-fibre and higher-fat rations are beneficial, as fat produces less metabolic heat compared with fibre fermentation.
- Supplementation with antioxidants (vitamins C, E, A) and minerals such as zinc reduces oxidative stress and improves immune function.
- Electrolyte supplementation **(Intalyte Oral)** helps maintain acid-base balance and hydration during heat stress.

Therapeutic Management

Fluid therapy

- Intensive intravenous fluid therapy using an electrolyte solution is recommended for the management of moderate to severe dehydration. This approach effectively corrects fluid and electrolyte imbalances, supports circulatory stability, rapidly restores plasma volume, aids in normalizing body temperature and helps prevent complications such as shock or organ dysfunction. **Intalyte** can be administered @ dosage of 500–2000 ml IV.

Corticosteroids

- Administration of corticosteroids can be done to prevent and manage shock conditions in animals. Isoflupredone **(Inerol)** can be administered @ 5-10 ml, IM according to size of animal and severity of heat stress.

Supportive therapy

- Electrolytes and multivitamins may be used as supportive supplementation to intravenous fluid therapy. This will help during recovery and in mild to moderate cases, to maintain hydration and electrolyte balance. **Intalyte Oral** (Sodium Chloride, Potassium, Calcium Propionate and Magnesium Sulphate fortified with Vitamin A, D3 and E) can be supplemented by dissolving 30 g in 1 litre of drinking water twice daily.
- A multivitamin supplementation to support immunity, improvement in udder health, reduction in oxidative stress during heat stress. **Intavita-NH Oral** (Vitamin A, D3, E, H and Nicotinamide) can be given @ 10 ml daily orally.
- Herbal liver tonic supports hepatic function compromised by metabolic stress, enhances nutrient utilization and promotes faster recovery. **Livotas** can be supplemented @ 30–40 ml orally once daily.
- Rumen buffer supplementation may be used to help stabilize rumen pH, support healthy digestion and rumination and reduce the risk of ruminal acidosis. **Bufzone** (a rumen buffer, yeast and metabolic booster) supplemented @ 200 g daily for 3-4 days orally.

Conclusion

Heat stress is a major constraint to dairy animals' welfare and productivity in tropical and subtropical regions. Prolonged exposure to high temperature and humidity disrupts thermoregulation, leading to reduced feed intake, impaired rumen and gut health, hormonal and immune suppression, and significant losses in milk yield, milk quality, reproductive performance and overall health, especially in high-producing and crossbred or exotic breeds. Heat stress during late gestation also has lasting negative effects on mammary development, future lactation and offspring performance. Effective mitigation requires an integrated approach combining environmental cooling, nutritional interventions and therapeutic support such as electrolytes, vitamins, buffers and liver supplements. With climate change intensifying heat stress risks, comprehensive management is essential to protect animal welfare, reduce economic losses and ensure sustainable dairy production.

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