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## Heat Stress in Dairy Cattle: Pathophysiology, Production Consequences, and Management

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### Abstract:

Heat stress is a major constraint on dairy productivity globally, exacerbated by ongoing climate change. When the temperature-humidity index (THI) exceeds 68, dairy cattle experience disruption to feed intake, milk yield, reproductive efficiency, immune function, and metabolic homeostasis. Effective mitigation requires integration of environmental cooling, feeding management modifications, and targeted nutritional strategies including energy-dense rations, electrolyte supplementation, rumen buffering, and antioxidant support. This review synthesizes current evidence on the pathophysiology of heat stress and consolidates practical recommendations for dairy producers.

**Keywords:** heat stress; dairy cattle; temperature-humidity index; nutritional management; thermoregulation; milk production; reproduction

### Introduction:

Climate change is intensifying the frequency and severity of heat stress events in livestock production worldwide. The Intergovernmental Panel on Climate Change (IPCC) has documented a mean surface temperature rise of 0.2°C per decade, with projections of a further 1.4-5.8°C increase by 2100 (IPCC, 2007). For the dairy industry, this trajectory translates directly into elevated production losses. Dairy cattle maintain core body temperature within 38.4-39.1°C, a state achievable within the thermoneutral zone (TNZ) of 16-25°C (Yousef, 1985). Above this range, thermal homeostasis is compromised and a cascade of physiological and metabolic disturbances follows. The temperature-humidity index (THI), a composite metric integrating ambient temperature and relative humidity, is the standard tool for quantifying heat load. Production impairment begins at THI  $\geq$  68, with milk yield declining by approximately 0.41 kg/cow/day per unit increase in THI above 69 (Spiers *et al.*, 2004). Upadhyay *et al.* (2009) estimated annual heat-stress-related milk losses in India alone at 1.8 million

tonnes, equivalent to Rs. 2661.62 crores per year. Though these figures are not scaled up for current inflation but they underscore the urgency of evidence-based amelioration strategies.

### Pathophysiology of Heat Stress:

#### ➤ Thermoregulatory and Metabolic Responses:

When heat load exceeds dissipation capacity, rectal temperature rises above 39.0°C and respiration rate exceeds 60 breaths per minute, which reliably predict the impairment of milk yield and fertility (Kadokawa *et al.*, 2012). Accelerated respiration drives increased CO<sub>2</sub> expiration, reducing blood carbonic acid concentration and ultimately inducing respiratory alkalosis. Renal bicarbonate excretion follows as a compensatory mechanism, depleting the body's buffering reserves (Schneider *et al.*, 1984). Concurrently, salivary flow to the rumen declines due to drooling which is a thermoregulatory behaviour, reducing endogenous rumen buffering and predisposing animals to subclinical and clinical acidosis. Rumen fermentation is further disrupted: acetate production decreases while propionate and butyrate rise, rumination time falls, and rumen motility is reduced. Hypothalamic suppression of appetite directly reduces dry matter intake (DMI), declining by up to 40% at ambient temperatures of 40°C in temperate conditions (Baile and Forbes, 1974).

#### ➤ Oxidative Stress and Immune Suppression:

Heat stress elevates reactive oxygen species (ROS) production across multiple tissues, imposing oxidative stress that overwhelms endogenous antioxidant defences in high-producing animals. Significantly elevated malondialdehyde, catalase, and superoxide dismutase (SOD) activity have been documented in heat-stressed lactating cattle during summer compared to spring seasons (Yattoo *et al.*, 2014). Immunologically, elevated cortisol suppresses neutrophil L-selectin expression, impairing pathogen clearance and elevating susceptibility to mastitis, respiratory disease, and other infections. White blood cell counts increase by 21-26% while red blood cell counts decline by 12-20% in thermally stressed cattle, reflecting broad haematological disruption (Abdel, 1987).

### Consequences For Milk Production and Reproduction:

#### ➤ Milk Yield and Composition:

The quantified impact of heat stress on milk production is substantial. Bouraoui *et al.* (2002) reported that a rise in THI from 68 to 78 reduced DMI by 9.6% and milk output by 21%. Gaafar *et al.* (2011) found that 305-day milk yield declined by 39% when summer THI reached 78.53, compared to a winter THI of 59.82. The mechanism of yield loss is multifactorial. Approximately 36% of the decline is attributable to reduced DMI and negative energy balance (NEB); the remainder reflects direct metabolic adaptation, wherein elevated basal insulin concentrations redirect glucose away from the mammary gland to peripheral non-mammary tissues (Baumgard and Rhoads, 2013). Maintenance energy requirements simultaneously increase by up to 30% in heat-stressed animals. Milk composition

also deteriorates; fat, SNF, and protein percentages decline significantly above THI 72, with reductions in casein fraction and casein index additionally reducing milk processing quality (Kadzere *et al.*, 2002).

#### ➤ **Reproductive Performance:**

Reproductive failure under heat stress is mediated through multiple simultaneous pathways. Elevated ACTH and cortisol suppress estradiol-induced sexual behaviour; follicular function is impaired by hyperthermia; granulosa cell aromatase activity declines, reducing estradiol synthesis and the LH surge necessary for ovulation; and endometrial PGF-2 $\alpha$  secretion increases, threatening early pregnancy. Up to 80% of estrus events may go undetected during summer (Rutledge, 2001). Conception rates fall from 40-60% in cooler months to as low as 10-20% in severe summer conditions (Cavestany *et al.*, 1985). Oocyte competence for fertilization and blastocyst development is compromised, with embryos exposed to heat stress during 1-7 days exhibiting elevated embryonic loss by day 42 of gestation. Male fertility is equally affected: scrotal hyperthermia degrades sperm morphology, motility, membrane integrity, and fertilization rate, with heat-stressed spermatozoa achieving fertilization rates of 53.7% compared to 70.2-81.5% in controls (Rahman *et al.*, 2013).

#### **Strategies for Amelioration:**

##### ➤ **Environmental Modifications:**

Environmental cooling is the primary and most effective intervention. Evaporative cooling systems combining soakers or sprinklers with forced ventilation through high-volume, low-speed fans delivering air speeds of 1-2 m/s across resting areas and feed bunks consistently improve milk production, reproductive performance, and feed conversion efficiency (Wolfenson, 2009). Shade provision reduces radiant heat load and is the most cost-effective measure for pasture systems. Crucially, cooling infrastructure must extend to dry cows: heat stress during late gestation reduces mammary cell proliferation, depresses milk yield in the subsequent lactation, and compromises calf immune development and lifetime performance (Tao and Dahl, 2013).

##### ➤ **Feeding Management:**

Timing and frequency of feed delivery significantly influence intake during heat stress. Aligning 60-70% of daily ration delivery to cooler evening and early morning hours, combined with frequent feed push-up, maximises DMI during periods of lower thermal burden. Multiple smaller feed deliveries reduce slug feeding a pattern that drives acute rumen pH depression and minimise concentrate selection and sorting. Adding water or liquid feeds to total mixed rations (TMR) improves cohesion and limits particle sorting. Unrestricted access to fresh, cool water is non-negotiable: heat-stressed cows may consume two to three times their normal intake, and any restriction in supply rapidly compounds DMI depression and production losses.

### ➤ Nutritional Adjustments:

Nutritional intervention targets four goals: increasing dietary energy density, minimising rumen heat of fermentation, restoring electrolyte balance, and supporting antioxidant defences. Fiber fermentation generates the highest heat increment among ruminant substrates; reducing dietary neutral detergent fiber (NDF) modestly while maintaining a minimum of 28-30% NDF to preserve rumen health decreases endogenous thermal burden. High-quality, highly digestible forages accelerate rumen passage and reduce sorting. Starch inclusion should not exceed 26-28% of dry matter (DM), with lower ceilings when highly fermentable sources predominate.

Supplemental fat provided at 2-3% of DM from rumen-inert (calcium soap) sources and palmitic/oleic acid blends delivers dense energy without contributing to rumen heat of fermentation, and is the preferred energy supplement during heat stress. Polyunsaturated fat sources should be minimised to protect butterfat synthesis. Total dietary fat should not exceed 6% of DM. Crude protein should be maintained at  $\geq 18\%$  of DM on a dry basis, with optimisation of ruminally undegraded protein (RUP) to supply metabolizable protein while minimising metabolic heat from nitrogen excretion (West, 1999). Electrolyte losses through sweating and urination require correction via increased dietary sodium (0.40-0.60% of DM) and potassium (1.50-1.60% of DM). When potassium is elevated, magnesium must also be increased to 0.35-0.40% of DM to counter competitive inhibition of absorption. Sodium bicarbonate at 0.5-1.0% of DM restores rumen buffering capacity compromised by respiratory alkalosis and reduced salivary flow. Antioxidant supplementation including Vitamins E and C, zinc, niacin, and betaine, mitigates oxidative stress and supports immune function. Live yeast cultures stabilise rumen fermentation during heat stress by modifying volatile fatty acid profiles and reducing ruminal ammonia concentration (Stella *et al.*, 2007).

### Conclusion:

Heat stress represents a multidimensional threat to dairy productivity, acting simultaneously through reduced feed intake, disrupted metabolism, impaired reproduction, and compromised immunity. As global temperatures rise, its economic toll will intensify. Effective mitigation demands an integrated approach: environmental cooling infrastructure extended to dry cows combined with strategic nutritional adjustments targeting energy density, electrolyte balance, rumen buffering, and antioxidant capacity, and supported by optimised feeding management. Additionally, long-term genetic strategies leveraging heat shock protein gene markers for selection of thermotolerant sires offer a heritable and sustainable complement to these interventions. Dairy producers operating in heat-challenged environments should develop farm-specific heat stress management plans, guided by continuous THI monitoring and in collaboration with qualified nutritionists and veterinarians.

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