Effects of Heat Stress on Production Performance and Health of Water Buffalo and the Underlying Mechanisms (Postprint)

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Abstract

Southern China predominantly exhibits hot and humid climatic conditions, where high ambient temperature represents the primary constraint on productivity for buffaloes inhabiting tropical and subtropical regions. This review synthesizes research advances regarding optimal environmental conditions for buffalo growth, the impacts of heat stress on production performance and health, and the underlying mechanisms, thereby facilitating improved assessment of heat stress effects on buffalo production and health to promote sustainable development of the buffalo industry.

Full Text

Research Progress on the Effects of Heat Stress on Production Performance and Health of Water Buffaloes and Its Underlying Mechanisms

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Abstract: High temperature and humidity characterize the climate of southern China, where elevated ambient temperature represents a primary constraint on the productivity of buffaloes in tropical and subtropical regions. This review synthesizes research progress on optimal environmental conditions for buffaloes, the impacts of heat stress on their production performance and health, and the

underlying mechanisms. The objective is to provide a better framework for evaluating heat stress effects on buffalo production and health, thereby promoting sustainable development of the buffalo industry.

Keywords: water buffalo; heat stress; temperature humidity index; production performance; health

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China possesses abundant water buffalo (*Bubalus bubalis*) resources. According to 2014 FAO statistics, China had 23.3478 million water buffaloes, accounting for approximately one-fifth of the global population and ranking third worldwide. Historically, water buffaloes served primarily as draft animals, making substantial contributions to agricultural development. With increasing mechanization, their role has gradually shifted toward meat, dairy, and dual-purpose production. Buffaloes are well-adapted to hot, humid climates in muddy marshlands, thriving in tropical and subtropical countries. They efficiently convert low-quality roughage into high-value products like milk and meat, reducing environmental impacts from crop byproducts. However, the influence of climate change on buffalo growth and production efficiency cannot be ignored, as numerous studies have confirmed that environmental factors reduce ruminant performance, particularly in dairy cattle. Climate variables including temperature, humidity, and radiation pose potential threats to livestock growth and production, with thermal environmental elements comprising temperature, relative humidity, solar radiation, and wind speed. As global warming intensifies, high ambient temperature will become the main constraint on buffalo productivity in tropical and subtropical regions. This review summarizes the effects of heat stress on buffalo production performance and health along with their mechanisms, providing new research directions for healthy and efficient buffalo production and theoretical foundations for mitigating or eliminating environment-induced heat stress.

1 Overview of Heat Stress in Water Buffaloes

Taxonomically, water buffaloes (family Bovidae, genus *Bubalus*) belong to the same family but different genus than cattle (family Bovidae, genus *Bos*). Based on chromosome number differences, buffaloes are classified into riverine type (2n=50) and swamp type (2n=48). Despite being more susceptible to heat stress when directly exposed to summer sunlight without access to mud wallowing, water immersion, or cold showers, buffaloes remain among the most heat-adapted livestock species. Although their black skin absorbs more heat, numerous melanin granules in the epidermis trap ultraviolet radiation, and sebaceous glands secrete protective oils, resulting in body temperatures typically lower than those of dairy cattle. Early international reports suggested ideal tropical climate conditions for meat buffalo growth and reproduction as 13-18°C temperature, 55-65% relative humidity, 5-8 km/h wind speed, and moderate light levels. However, due to regional climate variations and buffalo breed differences, establishing locally appropriate comfort indices for buffalo production

requires further investigation.

Heat stress constitutes a primary factor causing reproductive damage in tropical and subtropical countries, necessitating effective mitigation strategies. High temperatures induce significant changes in buffalo biological functions, including reduced feed intake, decreased feed utilization efficiency, disrupted metabolic balance, abnormal hormone secretion, altered enzymatic reactions, and abnormal blood metabolites, ultimately impairing production and reproductive performance. Direct radiation exposure rapidly exacerbates heat stress, prompting animals to actively seek shade. During heat stress, buffaloes exhibit specific signs: increased red patches hidden between the chest, abdomen, and legs; tongue protrusion; panting; salivation; conspicuous bloodshot eyes; reduced feed intake; decreased milk yield; and elevated surface and rectal temperatures.

Due to their black skin, low sweat gland density, and thick epidermis that reduces evaporative cooling capacity, buffaloes are more sensitive to hot environments when exposed to solar radiation. Room temperature changes cause greater fluctuations in buffalo rectal temperature than in other cattle. Buffaloes excrete water through urination at higher rates than other bovines. Their evolutionary adaptation to humid environments makes them more dependent on external water sources. Immersion in water proves more effective than spraying for reducing body temperature, alleviating stress, restoring physiological responses to normal ranges, and maintaining milk production and health.

2 Effects of Heat Stress on Growth, Reproduction, Lactation, and Immune Performance

Heat stress is the main factor reducing buffalo productivity in tropical and subtropical regions. High ambient temperature combined with high humidity easily causes animal discomfort, exacerbates stress levels, and inhibits physiological and metabolic activities. Generally, a Temperature Humidity Index (THI) exceeding 78 indicates severe heat stress in buffaloes. Heat stress conditions trigger a series of acute biological changes, including suppressed feeding, metabolic disorders, and reduced protein and energy utilization efficiency. These changes impede growth and reproductive performance while disrupting acid-base balance, hormone metabolism, and immune response, thereby affecting buffalo production.

2.1 Effects on Growth Performance

Temperature changes affect growth rate through feed intake, as heat stress reduces nutrient intake to decrease heat increment, resulting in insufficient nutrient supply and reduced production performance. In 32-39°C environments, buffalo excretion of mineral elements potassium, sodium, and calcium increased by 37%, 23%, and 30% respectively compared to 25-32°C conditions, with serum mineral content decreasing significantly. Planting trees in pasture areas effectively reduces solar radiation intensity, providing comfortable environmental

conditions and increasing production benefits. Short-term heat stress reduces dry matter intake in Murrah buffaloes with concurrent negative nitrogen balance, while long-term heat stress improves dry matter digestibility. The relationship between heat stress duration and digestibility/rumen passage rate is complex; under heat stress, the buffalo digestive tract exhibits environmental adaptability by reducing motility and slowing chyme passage rate, demonstrating that high temperature environments can promote feed nutrient digestibility. Previous studies often used non-alternating constant temperature models that cannot fully reflect actual production temperature variations, necessitating more detailed investigation of temperature change impacts in practical production.

2.2 Effects on Reproductive Performance

Buffalo reproductive performance is significantly temperature-dependent. Testicular temperature must be 2-6°C below body temperature to ensure semen quality; impaired heat dissipation causes testicular lesions and deteriorates semen quality. The primary factor for reduced summer fertility is high temperature affecting reproductive tract tissue cell function, increasing silent estrus probability, and influencing follicular development, hormone secretion, uterine blood flow, and endometrial function. Black skin and sparse hair cause buffaloes to absorb more heat in hot weather, while poor heat dissipation mechanisms and few sweat glands lead to metabolic heat excess and heat stress. This means heat stress affects reproductive capacity and pregnancy rates by increasing free radical production, reducing antioxidant generation, and elevating cortisol levels. Heat stress occurs when combined environmental parameters exceed the animal' s thermoneutral zone; research indicates THI 75 represents the threshold affecting pregnancy rates in Murrah buffaloes. When summer THI reaches 75-81, pregnancy rates decrease from 45% to 28%, showing significant seasonal variations in estrus, conception, and calving rates—highest in winter, reduced in autumn and spring, and lowest in summer. Particularly under high summer temperatures with high relative humidity and poor thermoregulatory efficiency, buffalo reproductive performance is severely affected. Improving reproductive rates has become a major goal for dairy buffalo industry development, significantly enhancing farm sustainability and production efficiency. Variations among studies may relate to heat stress duration, intensity, and genetics, requiring further research on relationships between heat stress parameters and buffalo reproductive performance.

2.3 Effects on Lactation Performance

Higher milk yield requires higher feed intake, but heat stress reduces intake to decrease heat production, contradicting high-yield objectives. High-producing buffaloes generate more metabolic heat during milk production, making them more susceptible to heat stress with negative impacts on body condition and milk yield. THI is widely used to evaluate heat effects on cattle. When THI is 35-72, buffalo milk production is unaffected by heat stress. High temperatures

affect mammary cell development, with higher milk yields in autumn-winter than spring-summer, though mammary cell mitochondrial DNA and morphological structure remain unchanged. Under heat stress, increasing THI reduces milk yield by 35-40%; high-producing buffaloes may lose 8-9 L daily, while low-producing buffaloes experience smaller impacts.

2.4 Effects on Immune Performance

Environmental factors such as temperature affect immune and antioxidant functions. Strong solar radiation in tropical and subtropical regions can damage skin tissue and potentially cause skin tumors. Studies show heat stress reduces buffalo immune function and increases disease susceptibility. Heat stress enhances macrophage phagocytosis and increases macrophage numbers. Calves born in summer have lower serum immunoglobulin levels than those born in February-March. Heat stress reduces serum immunoglobulin levels and suppresses immune function by decreasing lymphocyte proliferation. Reduced immune function during heat stress increases disease risk compared to normal conditions.

3 Mechanisms of Heat Stress in Water Buffaloes

Buffaloes are morphologically well-suited for tropical hot-humid environments, but without proper protection, their health is severely affected by environmental temperature and humidity, particularly during summer. Research on buffalo heat stress mechanisms primarily uses skin characteristics, respiratory rate, rectal temperature, sweating, and panting capacity as adaptation parameters. Neuroendocrine and renal function changes during heat stress have also been investigated.

3.1 Hormone-Mediated Pathways

The thyroid and adrenal glands play important roles in environmental adaptation. During heat stress, thyroid hormone concentration and metabolic heat production decrease while blood cortisol concentration increases. Studying hormone changes during metabolism better elucidates animal health changes under heat stress. Buffaloes show lower ovarian follicle estradiol-17 synthesis in summer. Heat stress causes hyperprolactinemia, reducing luteinizing hormone secretion and estradiol production during estrus. Progesterone deficiency during hot seasons reduces ovarian embryo survival in the uterus. This endocrine pattern ([Figure 1: see original paper]) may partially reduce sexual activity and reproduction rates in summer. Cortisol plays an important role during stress and affects immunity. Studies show buffaloes are most sensitive to heat stress, with cortisol concentrations increasing even under gradual heat stress in temperate environments. Blood cortisol concentration is 9.07-12.53 ng/mL at low temperatures but increases significantly as heat intensifies. However, hormonal regulation of heat stress is limited, requiring investigation of buffalo stress response mechanisms using multiple molecular biology techniques including electrophoresis and

chromatography.

3.2 Cell Apoptosis Pathways

High temperature adversely affects buffalo oocyte yield, quality, and development. Although blastocyst rates remain unaffected across seasons, follicle and oocyte numbers and blastocyst quality decrease during hot seasons. In autumn-winter, the pituitary becomes more sensitive, causing follicular accumulation and increasing small follicle numbers, but these small follicles respond poorly to gonadotropins and are more likely to undergo atresia during development. Mechanisms by which environmental temperature affects oocyte maturation may involve reduced intracellular protein synthesis, cytoskeletal disruption, microfilament/microtubule structure damage, and oocyte apoptosis induction. Post-stress ovarian damage to maternal mRNA replication and transcription mechanisms subsequently affects gene expression before and after embryonic genome activation. Studies show summer high temperatures affect buffalo oocyte division and blastocyst development. Heat shock protein (HSP) mechanisms during heat stress in domestic animals are well-documented. HSPs are a multigene family with molecular masses ranging 10-150 ku, found in all major cell types, enabling gradual cellular adaptation to environmental changes and playing key thermoregulatory roles in environmental stress adaptation. HSP70 mRNA shows significant differences between heat and cold treatments in buffalo oocytes; upregulated expression promotes apoptosis and serves as a heat stress severity marker in bovine embryos. Temperature elevation increases oocyte HSP70 mRNA expression mediated by cumulus cells, with more pronounced effects in immature oocytes.

3.3 Oxidative Stress Pathways

Early studies showed heat stress causes oxidative stress in transition buffaloes and reduces antioxidant activity in cow plasma. Bernabucci et al. reported antioxidant status may determine bovine reproductive function. Reactive oxygen species (ROS) and antioxidants may play important roles in follicular development. Megahed et al. noted heat stress may affect animal reproduction by increasing free radicals; elevated malondialdehyde (MDA) levels indicate buffalo fertility is affected by increased free radical production in summer. Additionally, serum MDA content and superoxide dismutase (SOD) activity during the luteal phase are significantly higher than during the follicular phase in summer. High SOD levels are needed to neutralize increased lipid peroxidation in summer follicles. Increased MDA during the luteal phase may enhance lipid peroxidation in luteal cell membranes of small follicles, potentially associated with gonadotropin receptor loss, reduced cyclic adenosine monophosphate (cAMP) formation, and decreased steroidogenic capacity. Small amounts of free radicals and oxides produced during normal metabolism are essential for various biochemical reactions, but accumulation during stress damages macromolecules like proteins and DNA. Thermal environments induce oxidative stress, causing DNA senescence

and cytotoxicity. Mammalian cells possess enzymatic and non-enzymatic antioxidant defense systems against oxidative damage, including SOD, catalase (CAT), glucose-6-phosphate dehydrogenase (G6PD), and glutathione (GSH). Studies show heat stress significantly increases CAT, SOD, and G6PD activities and GSH concentration in buffalo erythrocytes.

3.4 Immune Stress Pathways

All body cells express cytokine receptors, and cytokines participate in most physiological responses. Major inflammatory cytokines such as interleukin (IL)-2, IL-4, IL-6, and tumor necrosis factor (TNF)- stimulate inflammatory reactions through multiple cellular cascades and participate in immune regulation. Cytokines communicate the animal' s biological status to target cells through receptor interactions on cell surfaces. Cellular responses to cytokine stimulation depend on cytokine type and target cell nature, including proliferation, differentiation, and cell function. Inflammatory cytokines play key roles in stimulating systemic inflammatory responses, including increased body temperature, elevated heart rate, and reduced feed intake. Peripheral blood mononuclear cell (PBMC) proliferation improves when isolated from dry periods versus lactation periods. Immune cell damage in late-pregnancy buffaloes under heat stress may be influenced by prolactin (PRL) alterations; heat stress can regulate cellular immune status through PRL, modulating both innate and adaptive immunity. PRL regulates physiological activities by modulating cell proliferation, differentiation, and immune function. Heat stress increases plasma PRL concentration, while high PRL downregulates receptor expression, resulting in higher PRL mRNA expression in lymphocytes under cold conditions. Suppressor of cytokine signaling (SOCS) proteins comprise negative regulators that modulate immunity through feedback inhibition of cytokine signaling and immune cell/cytokine expression. SOCS-1 protein is a necessary regulatory factor for dendritic cell activation of T cells, maintaining immune tolerance by limiting T cell proliferation. Heat stress also increases neutrophil numbers while reducing lymphocyte counts; neutrophils primarily function in phagocytosis, while lymphocytes participate in immune protein synthesis and immune function regulation. In summer, untreated buffaloes show reduced lymphocyte interferon (IFN)- and IL-4 secretion compared to cooled buffaloes, indicating lower immune status. Reduced immune function and decreased neutrophil/immune cell migration and phagocytic capacity during transition periods increase disease susceptibility in dairy buffaloes.

4 Summary

In summary, although buffaloes can survive in hot-humid regions, heat stress effects cannot be underestimated due to their unique physiological characteristics. High temperature and humidity reduce feed intake and milk yield, impair production performance, damage oocytes, affect reproductive performance, induce immune stress, and reduce disease resistance. Therefore, future work should

prioritize investigating optimal growth environmental conditions and thresholds, establishing relevant evaluation indices. Based on these findings, evaluating various physiological responses and production performance changes under heat stress, and utilizing multiple physical measures and nutritional strategies to reduce heat stress hazards and improve buffalo rearing conditions represent important future research directions.

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Note: Figure translations are in progress. See original paper for figures.