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The hidden dangers of winter: A brief review how cold stress affects cattle production

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Abstract

Cold stress significantly impacts cattle health and productivity, especially in regions with harsh winter conditions. Exposure to temperatures outside the thermoneutral zone can lead to alterations in hematobiochemical parameters, including elevated blood glucose and liver enzyme levels, as well as impaired immune function. Cold stress also disrupts reproductive efficiency by causing hormonal imbalances, delayed estrus, and reduced conception rates. Additionally, it negatively affects milk yield and composition. Adaptations at the cellular level, such as changes in gene expression and protein synthesis, help cattle cope with cold exposure. Mitigation strategies, including improved shelter, nutrition, and health management, are essential to minimize the effects of cold stress and maintain livestock productivity.

Keywords: Cold stress, hematobiochemical parameters, cell responses and reproductive efficiency, mitigation strategy

Introduction

Every sphere of life has been impacted by global warming and has significantly impacted both the human and livestock realms and altered our planet's perfectly balanced environment. Incidences of cyclones, floods, landslides, draughts, snowstorms, and vector-borne diseases have all increased. Unfavourable changes in the weather and temperature are becoming an ongoing cause of stress. Animals behave normally and perform at optimal levels in the thermoneutral zone in terms of production and reproduction. When an animal's performance is below the lower critical temperature and above the upper critical temperature, it suffers. The performance of the animal is affected when the temperature is both above and below the upper critical temperature (Manzoor *et al.*, 2021) ^[13]. As homo-thermic animals, cows must keep their body temperature at 38 °C. Animals housed in the thermoneutral zone don't require additional energy to maintain a comfortable body temperature. Moreover, cows experience cold stress when temperatures fall below the lower critical temperature as depicted in (Fig1), which is the lower end of this range. This has an impact on the production and efficiency of cows. According to Angrecka and Herbut *et al.*, (2015) ^[1], animals with dry coats require 1% more energy for every degree of temperature drop below the LCT, while animals with wet coats require 2% more. The primary environmental issue dairy cattle housed in semi-open barns, free stall barns, and pastures in cold climates face is cold stress. Laven and Holmes (2008) and Angrecka and Herbut (2015) ^[1] has depicted environmental temperature is influenced by sun radiation, wind speed, and relative humidity rate, which might result in cold stress. Depending on factors including breed, age, gender, hair mantle, skin structure, size, and body surface, cattle attempt to adapt cold by exhibiting physiological and behavioural changes (Demirören 2007) ^[5]. In dairy cattle, the LCT is approximately 5 °C for calves, 14 °C for adults and dry animals, and 25 °C for lactating cows. As stated by the IUPS Thermal Commission (2003), "any change in the thermal relation between a temperature regulator and its environment which, if uncompensated by temperature regulation, would result in hyper- or hypothermia" is the definition of thermal stress, which is the result of exposure to conditions outside of the TNZ. In general, *Bos taurus* is more suited to cool- to temperate climates, but *Bos indicus* is more suited to tropical climates due to its lower metabolic rate.

Here in this review impact of cold stress in cattle is detailed briefly.

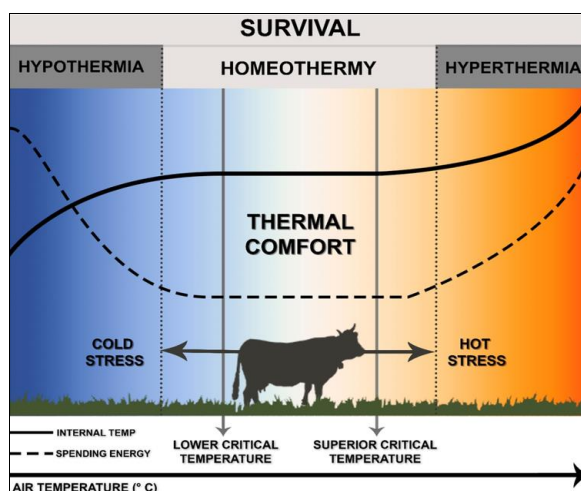


Fig 1: Representation of thermoneutral zone showing upper and lower critical temperature, zones of cold stress and heat stress in cattle (LE d O Aparecido *et al.*, 2021) [2].

1. Haemato-biochemical profile of cattle in cold stress

The blood biochemical profile is crucial for diagnosing and detecting any abnormal conditions in livestock. Cold stress in cattle can lead to significant changes in their hematobiochemical profile, which plays a vital role in diagnosing potential health problems and understanding the effects of environmental stress. Moreover, due to the exposure of cold temperatures, cattle experience various biochemical and haematological alterations, such as fluctuations in blood glucose levels, protein concentrations, and electrolyte balance, along with changes in red and white blood cell counts. Therefore, to adjust this condition animal's body maintain body temperature and energy levels by increasing required metabolic demands. Basically, during cold stress cortisol levels get elevated which is a common indicator of stress, alongside abnormal enzyme activity leads to potential liver function disruptions. Furthermore, by monitoring these hematobiochemical markers, farmers and veterinarians can gain a clearer picture of a cattle's health status, enabling them to detect early signs of stress or illness and implement effective management practices.

Effect of Cold Stress on Haematological Parameters:

Number of studies have explored how cold stress influences the haematological profile of cattle. Kumar *et al.* (2017) [11] observed that cattle exposed to cold stress exhibited an increase in RBC count and haemoglobin concentration, indicating an adaptive response to enhance oxygen delivery to

tissues. Moreover, the study also highlighted an increase in neutrophil count, suggesting heightened immune system activation. However, the researchers also reported a decrease in lymphocyte count, which is consistent with stress-induced immune suppression. This study underscores the importance of these haematological changes in supporting the animal's survival during cold weather, but it also indicates the potential risk of impaired immune function.

Biochemical Shifts During Cold Stress: In winter, the impact of cold stress on the biochemical profile of cattle leads to increase blood glucose and liver enzyme activities. Cold stress increases the metabolic demands for energy, leading to elevated blood glucose levels as the body mobilizes stored reserves to maintain body heat. The study also noted a rise in the activity of liver enzymes like AST and ALT, suggesting possible liver stress due to the cold exposure. Additionally, cortisol levels were found to increase under cold stress, further indicating the body's response to environmental challenges. These biochemical changes are important for understanding how cattle cope with stress, and they help in monitoring their overall health. The level of serum significantly higher serum total protein and albumin concentration during the summer season compared to winter season could be due to the dehydration caused by enhanced respiratory rate during the summer months (Tejaswi *et al.*, 2019) [18].

Immune Response Under Cold Stress: Cold stress can suppress the immune function of cattle, making them more susceptible to infections. According to studies cattle exposed to cold stress had reduced levels of lymphocytes and lower serum immunoglobulin concentrations, signalling an impaired immune response. Cattle under such stress were also more prone to respiratory infections, highlighting the vulnerability of their immune systems during harsh weather conditions. The findings emphasize the need for proactive management practices to support cattle's immune health during cold spells (Kim *et al.*, 2023) [10].

Electrolyte Imbalances in Cold-Stressed Cattle: Cold exposure in cattle is commonly associated with disruptions in the balance of key electrolytes such as sodium, potassium, and chloride. These imbalances often arise from reduced water intake during colder weather, which can lead to dehydration. This dehydration, in turn, intensifies the physiological stress on the animal. To prevent these issues and ensure cattle remain properly hydrated, it is essential to provide access to clean, unfrozen water, especially during winter months (Prasana *et al.*, 2022) [15].

Table 1: Impact of cold stress on Hematobiochemical parameters in cattle

Hematobiochemical Parameter	Effect of Cold Stress	Effect of Cold Stress
Red Blood Cell (RBC) Count	Increase	Cold stress stimulates erythropoiesis (RBC production) to enhance oxygen delivery. Kumar <i>et al.</i> , 2017 observed higher RBC counts as a compensatory response.
Hemoglobin Concentration	Increase	Elevated RBC count leads to increased haemoglobin, optimizing oxygen transport. Kumar <i>et al.</i> , 2017 confirmed this finding as part of the stress response.
White Blood Cell (WBC) Count	Variable: Increase or Decrease	WBC count can either increase or decrease depending on stress severity. Increased neutrophils but decreased lymphocytes, indicating immune system activation and suppression.
Neutrophil Count	Increase	An increase in neutrophils indicates immune system activation due to stress, as observed by Kumar <i>et al.</i> , 2017.
Lymphocyte Count	Decrease	Lymphocyte count decreases due to immune suppression during cold stress, as reported by Kumar <i>et al.</i> , 2017.
Monocyte Count	Variable: Increase or Stable	Monocyte counts can either increase or remain stable, reflecting immune response variability under cold stress.
Platelet Count	Variable Increase	Platelet counts may increase due to inflammation or tissue damage, as noted by Velayudhan., 2023.
Hematocrit (PCV)	Increase or Stabilize	Haematocrit levels may rise due to increased RBC production in response to cold stress (Kumar <i>et al.</i> , 2017).
Mean Corpuscular Volume (MCV)	Variable	MCV may increase or remain stable depending on RBC production adjustments, with variation in response to cold stress.

2. Cell response towards cold stress

Cells respond to cold stress through a series of complex adaptations aimed at maintaining homeostasis and protecting cellular integrity. Cells exposed to cold may trigger stress pathways involving p38 MAP kinase, which mediates stress-induced apoptosis and adaptation responses. Apoptosis may follow cold exposure upon rewarming, depending on factors such as exposure time, minimum temperature, and cell type. For instance, apoptosis occurs in Burkitt lymphoma cells after rewarming from a brief exposure at 1°C, suggesting that both temperature and duration critically impact cold-induced apoptosis. At extreme cold, ice crystal formation damages cellular structures, leading to necrosis through direct physical disruption of membranes and organelles, which is irreversible (Pradhan *et al.*, 2008) ^[16].

Cold stress triggers a series of cellular mechanisms that lead to changes in gene expression and protein synthesis, as well as unique responses during rewarming. One of the primary effects of cold exposure is the inhibition of transcription and translation, which reduces overall protein synthesis. This slowdown in protein production serves to conserve energy and may prevent the synthesis of misfolded proteins, which are more likely to accumulate under cold conditions (Pradhan *et al.*, 2008) ^[16]. In addition to this general inhibition, cold stress also inhibits RNA degradation in certain cells, allowing for the accumulation of specific cold-responsive proteins. For example, in bacteria, cold shock proteins (CSPs) are upregulated, and in hepatoblastoma cells, the expression of ATPase subunits is increased (Pradhan *et al.*, 2008) ^[16]. This adaptive mechanism helps cells manage the metabolic and stress responses to cold.

A key feature of cold-induced gene expression is the presence of cold-responsive elements in the promoter regions of certain genes. These elements are critical for activating cold-responsive genes, such as the cold-inducible RNA-binding protein (CIRP), which contains cold response elements that mediate its increased transcription during cold exposure (Chowdhury *et al.*, 2023) ^[4]. Such elements allow cells to rapidly adjust gene expression in response to the environmental stressor. Additionally, cold exposure can lead to alternative splicing of pre-mRNA, generating protein variants better suited to function under cold stress. For instance, in neurofibromatosis type 1, cold exposure alters the splicing pattern, potentially producing proteins that are more stable or functional at low temperatures (Pradhan *et al.*, 2008) ^[16]. Moreover, cold stress can enhance translation efficiency for certain mRNAs, allowing for more effective protein synthesis at lower temperatures. This is achieved through specialized sequences, such as internal ribosome entry sites (IRESs), that enable translation initiation without the usual reliance on the 5' cap structure. An example of this is RNA-binding motif protein 3 (RBM3), a cold shock protein that is translated more efficiently under cold conditions due to the presence of these IRES elements (Sahib *et al.*, 2024) ^[17]. These mechanisms collectively enable cells to better manage the physiological stress induced by cold exposure, ensuring that essential proteins are synthesized and adapted for the low-temperature environment.

In conclusion, cold stress induces a complex array of cellular responses, from inhibiting general protein synthesis to activating specific cold-responsive genes and proteins. These adaptations are crucial for cellular survival under cold conditions and also reflect the dynamic nature of gene expression and protein synthesis during cold stress and rewarming. After cold exposure, cells typically activate a

stress response as temperatures begin to normalize, initiating processes that help restore protein synthesis and promote cellular repair. One of the key responses is the activation of the cell stress response. Severe cold can cause protein denaturation, triggering cell stress signals, such as the phosphorylation of MAP kinases like p38. However, during hypothermic conditions, the low temperature itself may inhibit the full expression of heat shock proteins (HSPs), which are crucial for managing protein damage. Upon rewarming, the body reactivates these stress responses. For example, heat shock factor 1 (HSF-1) binds to heat shock elements (HSEs) in the promoter regions of stress-responsive genes, initiating the expression of HSPs like HSP70 and HSP90, which are involved in refolding damaged proteins and restoring cellular function (Sahib *et al.*, 2024) ^[17].

Another significant cellular event following cold exposure is the generation of free radicals upon rewarming. The transition from low to normal temperatures can lead to the production of reactive oxygen species (ROS), resulting in oxidative stress. ROS can damage cellular structures, including lipids, proteins, and DNA, prompting further stress responses such as the activation of heat shock proteins. This oxidative stress explains why certain heat shock proteins, such as HSPs, only appear during rewarming and not during the cold exposure itself, as the oxidative damage from rewarming is a distinct stressor that requires specific cellular responses (Pradhan *et al.*, 2008) ^[16].

Moreover, certain heat shock proteins exhibit temperature-dependent induction patterns. For example, proteins like APG-1 and HSP105 require a specific sequence of temperature shifts for their induction. These proteins are induced when cells are exposed to a cold temperature (e.g., 32°C) followed by a mild rewarming phase (e.g., 39 °C). This unique response suggests that these genes are regulated by HSF-1, which activates gene expression in response to temperature changes, but they are also possibly regulated by an unidentified repressor that becomes inactive at low temperatures but reactivates during specific warming conditions. This dual regulation allows the cell to finely tune the expression of these proteins in response to both the severity and rate of temperature shifts, highlighting the intricate control of gene expression during rewarming (Pradhan *et al.*, 2008) ^[16].

In conclusion, the mechanisms that follow cold exposure and rewarming involve a complex series of stress responses, including the activation of protein refolding processes, the generation of free radicals, and the induction of heat shock proteins in a temperature-dependent manner. These responses are critical for cellular recovery and protection against the damage caused by both the cold and the rewarming process, ensuring that cells can restore their normal functions and survive the stresses associated with extreme temperature fluctuations.

3. Cold stress disrupts reproductive efficiency

Cold stress has significant effects on the reproductive efficiency of livestock, impacting both male and female reproductive systems. The physiological changes induced by cold exposure can disrupt hormonal regulation, impair gamete development, and ultimately reduce reproductive performance. One of the key impacts of cold stress is hormonal imbalance. Cold exposure can disrupt the endocrine function, particularly affecting the secretion of gonadotropins, which are crucial for regulating reproductive cycles. In females, reduced secretion of hormones such as luteinizing

hormone (LH) and follicle-stimulating hormone (FSH) can interfere with ovulation, estrus cycles, and the maintenance of pregnancy. In males, these hormonal changes can lead to decreased testosterone production, impairing spermatogenesis and overall fertility (Chowdhury *et al.*, 2023) ^[4].

In addition to disrupting gonadotropins, cold stress also leads to an increase in corticosteroid levels, particularly cortisol. Elevated cortisol levels, a hallmark of stress, suppress the secretion of reproductive hormones and further interfere with reproductive function. Cortisol inhibits the normal functioning of the hypothalamic-pituitary-gonadal axis, which is critical for regulating reproductive processes. This suppression of hypothalamic and pituitary function can lead to a reduction in reproductive efficiency, including delayed estrus, poor conception rates, and increased early pregnancy loss in females, as well as decreased semen quality and libido in males (Pradhan *et al.*, 2008) ^[16]. These hormonal disruptions highlight the significant impact of cold stress on the reproductive health of livestock, affecting their ability to reproduce effectively and maintain optimal productivity.

i) Female reproduction

a) Delayed or Suppressed Estrus Cycles: Cold stress can prolong the interval between estrus cycles, making it challenging to time breeding accurately. In cattle and sheep, estrus frequency can decrease as energy is diverted from reproductive processes to maintaining body temperature (Sahib *et al.*, 2024) ^[17].

b) Embryonic Development: For pregnant females, cold stress may reduce blood flow to the uterus, compromising nutrient and oxygen supply to the developing embryo. This can lead to lower pregnancy rates and increase the risk of early embryonic mortality (Pradhan *et al.*, 2008) ^[16].

c) Reduced Conception Rates: In dairy cattle, cold stress has been associated with decreased conception rates, as the body prioritizes thermoregulation over reproduction. Reduced fertility under cold conditions can result in extended calving intervals, impacting herd productivity (Chowdhury *et al.*, 2023) ^[4].

ii) Male Reproduction

a) Spermatogenesis: Cold stress can impair spermatogenesis, particularly if prolonged exposure diverts blood flow and reduces testicular temperature stability, essential for optimal sperm production. Studies have shown that extreme cold can reduce sperm motility and increase sperm abnormalities, affecting overall semen quality in bulls and rams (Sahib *et al.*, 2024) ^[17].

b) Libido Reduction: The energy demands associated with cold stress can lead to reduced libido or mating behaviours in male livestock. Lower libido and compromised semen quality decrease successful mating rates in natural breeding setups (Pradhan *et al.*, 2008) ^[16].

4. Effect of cold stress on milk yield and milk composition

The protein and total dry matter rate in milk, or SCC, of Simmental dairy cattle are impacted by cold stress (Kaygusuz and Akdağ, 2021) ^[9]. Additionally, the existence of a negative phenotypic association between milk fat and total dry matter rate was confirmed. Reduced energy intake from forage may result in a decrease in milk protein and milk fat rates, which in turn may lead to a decrease in total dry matter (Laven and Holmes 2008, Bickert and Mattiello 2016) ^[12, 3]. Low environmental temperature may also speed up basal

metabolism and speed up digestion. According to Dinçel and Dikmen (2013) ^[6], variations in milk somatic cell count are caused by seasonal changes.

5. Nutrition and Energy Allocation

Cold stress increases metabolic rates, causing livestock to use more energy to maintain body heat, which can lead to reduced body condition. For both males and females, insufficient energy can impair reproductive functions, as the body conserves resources for survival rather than reproduction. Adequate nutrition and energy reserves are essential during cold periods to support reproductive processes (Chowdhury *et al.*, 2023) ^[4].

6. Management Practices to Mitigate Cold Stress

Providing adequate shelter can protect livestock from extreme cold, reducing the extent of cold stress on reproductive functions. Increasing dietary energy intake helps animals meet the higher metabolic demands of cold weather, supporting reproductive health. Providing heated areas, bedding, and proper ventilation can further reduce the impact of cold stress on reproductive efficiency in livestock (Pradhan *et al.*, 2008) ^[16].

7. Management strategies to mitigate cold stress

The effects of cold stress are avoided by raising the barn's temperature, lowering its humidity, improving ventilation, providing bedding to protect against cold floors, lukewarm drinking water, hay and concentrate for increased heat generation and energy balance, dry teats to prevent frostbite, heat lamps, calf jackets, and warm blankets.

Various mitigation strategies are mentioned below

1. Nutritional management: The energy content in feed rations must be increased to 77% total digestible nutrients (TDN), compared to the usual 70-72% and the crude protein (CP) content from 14.5% to 17.5% to support better growth and milk production. Including about 20% fiber in the feed, compared to 17%, can help increase the fat percentage in milk and reduce the negative effects of cold temperatures on animals. Animals kept outdoors during the colder season require approximately 15-20% more feed than those kept in confinement housing to maintain their energy levels and body condition throughout the season (Graunke *et al.*, 2011) ^[7]. Providing concentrates such as feed blocks, UMBB licks, cubes, meals, and cakes can help deliver essential nutrients. Offer a range of cultivated fodders, including paddy, oat, maize, berseem, lucerne, and turnip, to supply a balanced diet rich in essential nutrients. Chaffing straw can reduce wastage by 15-20%, improving feed efficiency by conserving this valuable resource. Applying fertilizer-grade urea at a rate of 4-5% raises the digestibility by roughly five units; however, if the straws are ensiled for ten days, the digestibility is doubled.

1. Shelter Management: Proper sheds should be provided to protect animals from prevailing chilly winds. Heating facilities, such as room heaters, should be installed, and curtains can be used to help retain warmth within the shed. Ventilation in the shed should be appropriate and free from drafts, with relative humidity ideally maintained between 40% and 80%. Bedding should be clean and dry, with a thickness of 4-6 inches for large animals and 2 inches for smaller animals, placed on a

concrete floor to reduce body heat loss. The shed should be cleaned at least twice a day to ensure proper disposal of waste and to minimize the buildup of ammonia gases. Calf jackets and blankets are useful for keeping calves warm and reducing their exposure to cold temperatures.

2. **Health Management:** Vaccination schedules, nutritional supplementation, and deworming protocols should be strictly followed to maintain animal health. Wet and muddy conditions, as well as feed contamination from manure, should be avoided to prevent the incidence of coccidiosis. Using a teat dip powder can reduce the risk of frostbite on teats during cold winter months. Regular checking must be done for bruises on the soles, trim overgrown hooves, and take steps to prevent laminitis and lameness in the animals.

Conclusion

Cold stress significantly impacts cattle across multiple aspects of their health and productivity. Exposure to low temperatures triggers changes in their haematological and biochemical profiles, affecting metabolic functions and immune responses. These physiological shifts can lead to reproductive challenges, including hormonal imbalances that disrupt fertility in both males and females. Additionally, cold stress can reduce milk yield and alter its composition, further affecting dairy production. On a cellular level, cattle adapt through modifications in gene expression and protein synthesis to maintain homeostasis. To mitigate these effects, it is essential to implement effective management strategies, such as improving shelter, adjusting nutrition, and ensuring optimal health care. By understanding and addressing the consequences of cold stress, farmers can improve cattle welfare and maintain productivity during colder periods.

Conflict of interest

Authors do not have any conflict of interests to declare.

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