

## Review

# Heat Stress in Dairy Cattle Production: A View from the Perspective of Sustainable Development

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## Abstract

Climate change is transforming heat stress (HS) from a seasonal challenge into a major constraint on the sustainability of dairy cattle production. Increasing temperatures, more frequent and prolonged heat waves, and reduced opportunities for nocturnal recovery adversely affect animal health, welfare, productivity, reproductive performance, and resource-use efficiency. This narrative review provides an integrated critical synthesis of current evidence on HS in dairy cattle within a sustainable development framework. It examines the biological mechanisms underlying disruption of homeostasis together with the consequences for milk production, product quality, health, welfare, reproduction, and developmental programming. The review further evaluates biomarkers of thermal adaptation, precision livestock farming, and environmental, nutritional, managerial, genetic, and technological mitigation strategies, considering their biological effectiveness, practical applicability, economic feasibility, and environmental trade-offs. Rather than addressing these components independently, the review integrates their interactions within an original conceptual framework and comparatively evaluates adaptation strategies to support practical decision-making. Current evidence indicates that sustainable climate adaptation requires integrated, context-specific approaches combining environmental management, nutrition, genetic improvement, precision monitoring, and digital technologies. Future progress will increasingly depend on advances in omics, artificial intelligence, and predictive livestock management to enhance climate resilience while improving productivity, animal welfare, and environmental sustainability.

**Keywords:** heat stress; dairy cattle; climate change; sustainable development; animal welfare; thermotolerance; precision livestock farming; climate resilience



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## 1. Introduction

Climate change is intensifying heat stress (HS), thereby reshaping the productivity, welfare, resource-use efficiency, and sustainability of dairy cattle production. Rising temperatures, greater climatic variability, more frequent and prolonged heatwaves, and reduced nocturnal cooling are increasing the frequency, duration, and severity of thermal challenges worldwide. Consequently, HS has become one of the principal constraints limiting the long-term sustainability and resilience of dairy production systems [1,2].

HS develops when metabolic and environmental heat loads exceed an animal's capacity for effective heat dissipation. The resulting response extends far beyond a transient increase in body temperature and involves coordinated thermoregulatory, behavioural, endocrine, metabolic, oxidative, and immune adjustments. Although these responses initially

preserve thermal homeostasis, their prolonged activation progressively disrupts physiological functions and diverts nutrients and energy away from production, reproduction, and health maintenance [3,4].

The consequences of HS extend beyond impaired productivity. Heat stress compromises milk yield and composition, reproductive efficiency, immune competence, animal welfare, and overall health, while increasing evidence indicates that its effects may persist after thermal exposure has ceased and may even influence subsequent generations through developmental programming [5,6]. These biological disturbances translate directly into economic losses, increased resource requirements, and environmental challenges associated with sustaining dairy production under increasingly variable climatic conditions. HS should therefore be regarded not only as a physiological disturbance but also as a major sustainability challenge affecting production efficiency, resource use, economic viability, environmental performance, and societal expectations regarding animal welfare [2,7].

Although numerous reviews have examined individual aspects of HS, including physiological adaptation, oxidative stress, immunometabolism, nutrition, reproduction, precision livestock farming, genetic improvement, and climate adaptation, these topics have generally been addressed independently. Consequently, limited attention has been paid to the interactions among biological responses, management decisions, technological innovations, resource requirements, and sustainability outcomes that collectively determine the resilience of dairy production systems. This fragmentation limits the development of integrated strategies capable of simultaneously improving animal health, productivity, resource-use efficiency, and long-term sustainability [4,8].

This narrative review addresses this gap by critically integrating current evidence on the biological mechanisms of HS, its consequences for productivity, product quality, health, welfare, and reproduction, together with advances in nutritional, environmental, technological, management, and genetic adaptation strategies within a unified sustainability framework. Rather than considering these components separately, the review evaluates their interactions and their collective contribution to improving the resilience and long-term sustainability of dairy cattle production [9,10].

This narrative review aims to critically synthesise current knowledge on HS in dairy cattle production from the perspective of sustainable development. It examines climate-driven thermal exposure, the biological mechanisms underlying disruption of homeostasis, and the consequences for productivity, product quality, health, welfare, and reproduction. The review further evaluates current biomarkers, predictive monitoring approaches, and adaptation strategies in terms of their biological effectiveness, practical applicability, economic feasibility, resource requirements, and environmental trade-offs. Finally, it identifies key knowledge gaps and future research priorities for developing climate-resilient dairy production systems.

## 2. Methodological Approach

### 2.1. Literature Search Strategy

This narrative review synthesises current scientific evidence on heat stress (HS) in dairy cattle production from the perspective of sustainable development. The evidence base for this narrative review was developed through continuous monitoring of scientific publications, citation tracking of influential studies, and structured searches of major international databases, including Scopus, Web of Science, ScienceDirect, SpringerLink, and PubMed. The final literature search was completed in June 2026.

Search strategies combined controlled vocabulary and free-text terms related to heat stress, climate change, dairy cattle, thermoregulation, physiological adaptation, productivity, product quality, health, reproduction, animal welfare, biomarkers, precision livestock

farming, genetic adaptation, mitigation strategies, and sustainability. Search queries were adapted to the indexing systems and search functionalities of the individual databases to maximise retrieval of relevant publications.

## 2.2. Eligibility Criteria and Literature Selection

The review primarily included peer-reviewed articles published in English-language journals and assigned a Digital Object Identifier (DOI). Priority was given to studies published within the previous five years to reflect current advances in understanding the biological, technological, and sustainability aspects of HS, whereas earlier landmark publications were included where necessary to provide historical context and support fundamental concepts.

Publications were considered eligible when they directly addressed the effects of HS on animal physiology, productivity, health, welfare, reproduction, management, adaptation strategies, or sustainability. Studies outside the scope of the review, duplicate records, publications lacking sufficient methodological information for critical interpretation, and papers without DOI identifiers were excluded.

Following literature screening and removal of duplicate records, the final evidence base comprised 221 publications, which were organised into thematic sections covering climate change, biological mechanisms of HS, productivity and product quality, health and welfare, biomarkers, precision livestock farming, adaptation strategies, genetics, and sustainability.

Because this study was designed as a narrative review rather than a systematic review or meta-analysis, no formal risk-of-bias assessment or structured quality appraisal tool (e.g., SANRA) was applied. Instead, publications were selected according to pre-defined eligibility criteria and critically evaluated with respect to their methodological transparency, scientific contribution, and relevance to the objectives of the review. Where conflicting findings were identified, they were critically interpreted in the context of differences in study design, climatic conditions, production systems, animal populations, and methodological approaches.

## 2.3. Scope of the Review

This review examines HS as a multidimensional challenge affecting the biological, productive, economic, environmental, and welfare dimensions of sustainable dairy cattle production. It integrates current evidence on climate change, mechanisms of HS, productivity and product quality, animal health and welfare, reproductive performance, biomarkers, precision livestock farming, adaptation strategies, genomic approaches, and sustainability. Rather than considering these components independently, the review evaluates their interactions and their collective contribution to improving the resilience and long-term sustainability of dairy production systems.

To facilitate interpretation of this interdisciplinary perspective, the review is organised into interconnected thematic sections that progressively examine climate drivers, biological mechanisms, productive and health-related consequences, adaptation strategies, sustainability implications, and future perspectives. These relationships are integrated within the original conceptual framework presented in Figure 1, which illustrates the causal pathways, adaptation mechanisms, sustainability trade-offs, and feedback loops that collectively shape the resilience and sustainability of dairy production systems and provide the structural basis for the organisation of the review.

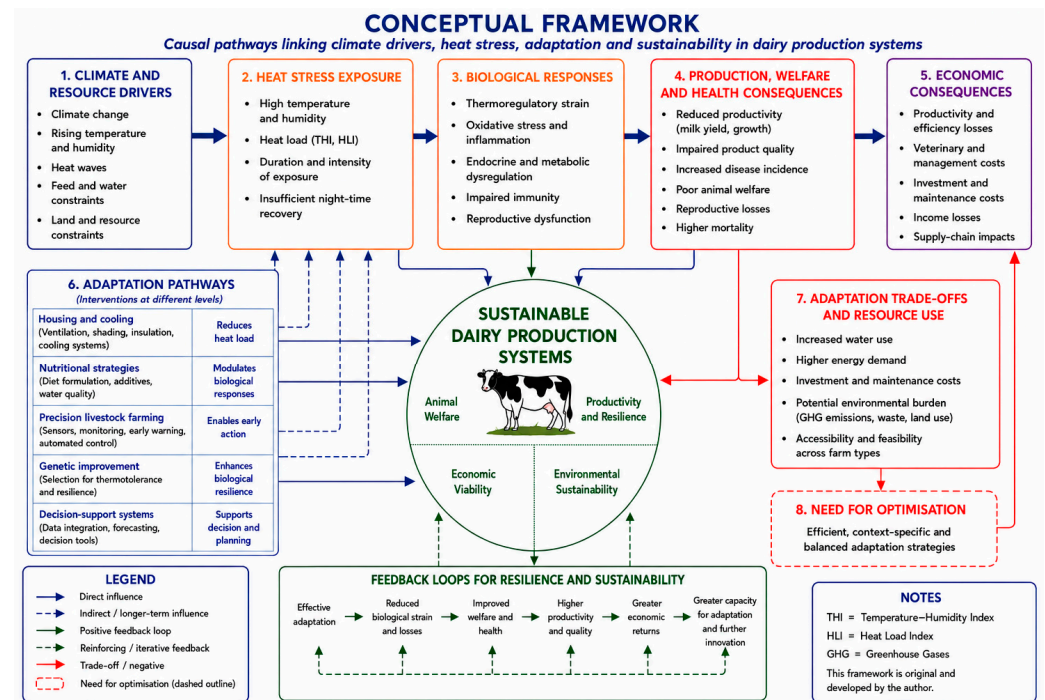


**Figure 1.** Original conceptual framework illustrating the conceptual relationships among climate and resource drivers, heat stress exposure, biological responses, production, welfare and economic consequences, adaptation pathways, sustainability trade-offs, and feedback loops in sustainable dairy production systems. Source: Developed by the author.

## 2.4. Limitations

Several limitations should be considered when interpreting this review. First, owing to its narrative design, the review does not include quantitative meta-analysis or formal statistical synthesis of published findings. Consequently, conclusions are based on critical interpretation and integration of the available evidence rather than quantitative estimates of pooled effects.

Second, substantial heterogeneity exists among published studies with respect to climatic conditions, production systems, breeds, experimental designs, heat-stress indicators, and measured outcomes, which limits direct quantitative comparison across investigations. Variability in reporting standards, together with differences in the balance between original research articles and secondary review literature, may also contribute to inconsistencies in reported effect sizes and complicate evidence synthesis.

Finally, although literature searches were conducted across multiple international databases, differences in database coverage, indexing practices, and publication availability may have influenced the completeness of the evidence base. Accordingly, this review should be interpreted as an integrative critical synthesis of current knowledge intended to identify major advances, remaining knowledge gaps, and priorities for future research rather than as a quantitative ranking of individual adaptation strategies.

## 3. Heat Stress in Dairy Production Systems: Biological Impacts, Adaptation Strategies and Sustainability Challenges

### 3.1. Climate Change as a Driver of Thermal Challenges in Livestock Production

Climate change has become one of the principal global drivers reshaping livestock production systems and their long-term sustainability. Rising average air temperatures, altered precipitation patterns, increasing climatic variability, and the growing frequency and duration of heatwaves are creating new challenges for animal productivity, health,

welfare, and the economic resilience of livestock enterprises. Consequently, climate change is increasingly recognised not merely as an environmental issue but as a systemic factor influencing the efficiency, adaptability, and sustainability of animal production systems [2].

One of the most significant emerging trends is the progressive expansion of the geographical distribution of HS. In traditionally hot regions, both the intensity and duration of thermally challenging periods continue to increase, whereas temperate areas are experiencing HS with increasing frequency despite historically favourable climatic conditions. Long-term observations in Australia have demonstrated substantial seasonal and interannual variation in thermal load affecting cattle, including prolonged periods of unfavourable environmental conditions [11]. Similarly, studies conducted in Canada indicate a gradual increase in the risk of HS in dairy cattle within regions that were previously considered relatively unaffected by excessive heat [12]. Collectively, these findings demonstrate that HS is no longer confined to tropical and subtropical livestock production but has become an increasingly widespread challenge across diverse climatic zones.

Climate change is also altering the characteristics of heatwaves themselves. In subtropical regions of Brazil, increases in both the frequency and duration of heatwaves have intensified thermal stress in high-producing Holstein cows, highlighting the growing climatic risks even within established dairy production areas [13]. Comparable trends have been reported following the analysis of seven decades of climatic data in Italy, where significant increases in both the frequency and persistence of heatwaves have already affected the production environment of small ruminants [14]. These findings indicate that the severity of HS depends not only on peak ambient temperature but also on the duration of thermal exposure, the recurrence of extreme events, and the opportunity for nocturnal recovery. The cumulative nature of thermal load therefore explains why prolonged exposure to moderately elevated temperatures may exert greater biological consequences than brief episodes of extreme heat.

The vulnerability of livestock production systems, however, is far from uniform. In South Asian countries, the temperature–humidity index (THI) exceeds thresholds associated with HS in ruminants during a substantial proportion of the year [15,16]. In small island states of the South Pacific, including Vanuatu, high ambient temperatures interact with elevated humidity, climatic variability, and limited technological capacity for adaptation, thereby increasing overall vulnerability [17]. Conversely, intensive dairy production systems are particularly susceptible because high-producing animals generate considerable metabolic heat and depend heavily on stable environmental conditions. These observations demonstrate that climatic risk is determined not solely by weather conditions but by the interaction between climate, genotype, production level, housing system, and the availability of adaptive resources.

The effects of climate change on livestock production also occur indirectly through reduced feed and water security. Increasing temperatures, prolonged droughts, and increasingly unpredictable precipitation patterns diminish forage yield, reduce feed quality, and restrict water availability. These challenges are especially pronounced in arid and semi-arid regions, where resource scarcity simultaneously intensifies physiological strain on animals while limiting the capacity of farms to implement cooling technologies, optimise nutritional management, and adopt other adaptive measures [1,18]. Consequently, the direct effects of elevated environmental temperatures and the indirect consequences of resource limitation reinforce one another, amplifying the overall impact of HS.

Taken together, current evidence indicates that HS is progressively evolving from a short-term seasonal phenomenon into a systemic production risk. Its impact is determined not only by the occurrence of extreme heat events but also by the duration of heatwaves, insufficient nocturnal recovery, regional vulnerability, and increasing constraints on feed

and water resources. For modern dairy production systems, these developments highlight the need to move beyond reactive responses to individual heat events towards long-term climate risk management integrating continuous environmental monitoring, predictive approaches, animal adaptation, and the progressive modernisation of production systems.

### 3.2. A Challenge to Animal Homeostasis

Maintenance of homeostasis is fundamental to animal survival, health, and productivity. In high-producing dairy cows, however, this balance is continuously challenged by the need to dissipate the large amounts of metabolic heat generated during lactation. Consequently, HS should not be viewed simply as exposure to elevated ambient temperatures but as a systemic biological response that develops when total heat gain exceeds the animal's capacity for heat dissipation, triggering coordinated physiological, metabolic, endocrine, and immune adaptations [3,19].

The severity of HS is determined not only by ambient temperature but also by relative humidity, solar radiation, air velocity, housing conditions, nocturnal cooling, and endogenous heat production. Consequently, assessment of HS has evolved from reliance on individual climatic variables to integrated biometeorological indices and thermodynamic models that more accurately describe the balance between heat production and heat loss [19,20]. Thermodynamic modelling indicates that, at approximately 40% relative humidity and an air velocity of  $1.4 \text{ m s}^{-1}$ , the thermal balance of dairy cows begins to deteriorate at ambient temperatures close to  $20^\circ\text{C}$ . Endogenous heat production increases by approximately 27.3% in cows producing  $18.5 \text{ kg of milk day}^{-1}$  and by 48.5% in cows producing  $31.6 \text{ kg day}^{-1}$ , explaining why high-producing animals experience HS under environmental conditions that remain tolerable for lower-producing cows [20].

The earliest response to HS involves activation of thermoregulatory mechanisms that maximise heat dissipation while minimising additional heat production. Increased respiratory rate, peripheral vasodilation, redistribution of blood flow towards the skin, greater water intake, reduced physical activity, and behavioural adjustments collectively enhance evaporative and convective heat loss. Among these responses, increased respiratory rate is one of the earliest and most sensitive indicators of HS, often preceding measurable increases in core body temperature. These coordinated responses are orchestrated primarily by the hypothalamus, which integrates thermal signals and regulates autonomic, cardiovascular, endocrine, and behavioural functions. During prolonged HS, however, the efficiency of these compensatory mechanisms progressively declines, leading to cumulative heat storage and ultimately thermoregulatory exhaustion [20,21].

As physical thermoregulation becomes less effective, maintenance of homeostasis increasingly depends on neuroendocrine adaptation. Activation of the hypothalamic–pituitary–adrenal (HPA) axis stimulates the sequential release of corticotropin-releasing hormone (CRH), adrenocorticotrophic hormone (ACTH), and cortisol, promoting metabolic flexibility through mobilisation of energy reserves, regulation of water and electrolyte balance, and modulation of immune function [22,23]. Nevertheless, endocrine responses vary substantially with the intensity and duration of HS, production level, physiological status, and individual thermotolerance, indicating that endocrine plasticity is itself a key determinant of heat resilience [22,23].

Among endocrine mediators, prolactin has received increasing attention because its functions extend far beyond lactation. In addition to supporting mammary activity, prolactin contributes to thermoregulation, maintenance of epithelial barrier integrity, water balance, and cellular protection during hyperthermia. Its widespread distribution in the hypothalamus, mammary gland, gastrointestinal tract, and immune cells highlights its role as an integrative regulator linking endocrine, metabolic, and immune adaptation. Conse-

quently, elevated circulating prolactin concentrations despite declining milk production should be interpreted as part of a coordinated adaptive response rather than solely as a consequence of lactogenic regulation [23].

As HS persists, compensatory mechanisms gradually become overwhelmed, promoting oxidative stress, which is now recognised as a central driver of HS-induced dysfunction [24,25]. Excessive production of reactive oxygen species (ROS) damages lipids, proteins, and nucleic acids while activating the NF- $\kappa$ B signalling pathway and suppressing the antioxidant regulator Nrf2. This imbalance amplifies inflammation, weakens antioxidant defences, and establishes a self-perpetuating cycle of oxidative damage [25]. Mitochondria are among the earliest cellular targets, with heat-induced dysfunction reducing ATP synthesis, disrupting calcium homeostasis, and impairing oxidative phosphorylation. These changes subsequently trigger endoplasmic reticulum stress, autophagy, apoptosis, and the synthesis of heat shock proteins (HSPs), which initially preserve protein stability but become insufficient during prolonged HS [25].

The consequences of oxidative stress extend far beyond the cellular level. Organs with high metabolic demands, including the liver, mammary gland, skeletal muscle, and intestinal epithelium, are particularly susceptible to mitochondrial dysfunction and oxidative injury, resulting in impaired nutrient utilisation, reduced milk synthesis, and compromised immune competence. Experimental studies further demonstrate that enhancing antioxidant capacity attenuates increases in rectal temperature, respiratory rate, and oxidative biomarkers, reinforcing the concept that oxidative stress is not merely a consequence of HS but a key mechanism linking thermal challenge to systemic dysfunction [24,25].

HS also profoundly reshapes systemic metabolism by redirecting nutrients from productive functions towards the maintenance of homeostasis. Consequently, declines in milk yield cannot be explained solely by reduced feed intake but reflect active metabolic reprogramming driven by endocrine adaptation, oxidative stress, and cellular responses [25–28]. Although dry matter intake (DMI) typically decreases by 10–15% (approximately 2.0–3.75 kg day<sup>−1</sup> in high-producing cows), pair-feeding experiments consistently show that heat-stressed cows produce less milk than thermoneutral cows receiving the same amount of feed, demonstrating that hyperthermia directly disrupts metabolic regulation independently of nutrient intake [25,27].

During HS, glucose, amino acids, and lipids are preferentially diverted towards thermoregulation, antioxidant defence, tissue repair, and immune function rather than milk synthesis and reproduction. Although this metabolic redistribution is essential for survival, it inevitably compromises productive efficiency. The mammary gland is particularly vulnerable because sustained lactation depends on a continuous supply of oxygen and metabolic substrates. Whereas moderate HS may transiently increase mammary blood flow, severe thermal load (THI > 79) markedly reduces tissue perfusion, glucose uptake, oxygen delivery, and nutrient clearance, thereby limiting secretory activity independently of feed intake [27].

At the molecular level, hyperthermia further impairs mammary function by suppressing amino acid transport and inhibiting the mechanistic target of rapamycin (mTOR) signalling pathway, a central regulator of protein synthesis. Expression of the amino acid transporters *SLC7A5*, *SLC38A2*, and *SLC38A3*, together with the  $\beta$ -casein gene *CSN2*, is reduced, providing direct molecular evidence that HS suppresses milk protein synthesis. Consequently, reductions in milk yield and protein concentration result from the combined effects of altered nutrient partitioning, impaired mammary perfusion, and inhibition of anabolic signalling rather than decreased nutrient intake alone [26,28].

Beyond host metabolism, the gastrointestinal microbiome has emerged as an important regulator of thermal adaptation. HS induces substantial remodelling of ruminal microbial

communities independently of feed restriction, demonstrating that hyperthermia directly alters microbial ecology [25,29,30]. Among the most consistent changes are reductions in fibrolytic bacteria such as *Fibrobacter*, accompanied by shifts in metabolically important genera, including *Prevotella* and *Succiniclacticum*. These alterations modify carbohydrate fermentation, nitrogen metabolism, volatile fatty acid production, and overall energy harvest, ultimately influencing nutrient availability to the host [29,30].

Microbial remodelling also alters the production of short-chain fatty acids and other signalling metabolites involved in maintaining intestinal barrier integrity, hepatic metabolism, and systemic immunity. Increased intestinal permeability may facilitate translocation of bacterial components into the circulation, further amplifying inflammatory signalling initiated by oxidative stress. Chronic HS is additionally associated with reduced microbial diversity and extensive functional reorganisation of pathways involved in amino acid metabolism and energy production, whereas specific microbial profiles appear to enhance thermotolerance [25,29–31]. These findings indicate that the microbiome is not merely a passive target of HS but an active determinant of heat resilience and a promising target for nutritional interventions aimed at improving climate adaptation [29–31].

Collectively, these findings demonstrate that metabolic reprogramming during HS extends well beyond alterations in energy metabolism to encompass coordinated interactions among endocrine regulation, cellular signalling, mammary function, and the gastrointestinal microbiome. Together, these processes form the mechanistic bridge between the initial physiological response to HS and the immune remodelling that characterises prolonged thermal stress [25,31].

HS profoundly alters immune function, although its effects extend far beyond the traditional concept of immunosuppression. Increasing evidence indicates that HS induces immune remodelling, characterised by the concurrent activation of pro-inflammatory and regulatory pathways rather than uniform suppression of immune activity [25,32–35]. Oxidative stress provides the principal link between metabolic dysfunction and immune activation through NF- $\kappa$ B-dependent signalling, promoting the production of inflammatory mediators, including interleukin-1 $\beta$  (IL-1 $\beta$ ), interleukin-6 (IL-6), and tumour necrosis factor- $\alpha$  (TNF- $\alpha$ ). Simultaneously, anti-inflammatory mechanisms are activated to limit tissue damage, and the balance between these opposing responses largely determines whether adaptation is maintained or progresses to chronic inflammation [25,34].

HS also alters the function of peripheral blood mononuclear cells (PBMCs), macrophages, monocytes, and other immune cell populations by modifying cytokine production, heat shock protein expression, and the activity of genes involved in cellular defence [32–34]. The magnitude of these responses varies considerably among animals, indicating that thermotolerance depends not only on thermoregulatory efficiency but also on immune plasticity. Importantly, immune alterations frequently persist after environmental temperatures return to normal, providing a biological basis for the delayed onset of mastitis, metabolic disorders, and reproductive dysfunction during the post-HS recovery period [32,33].

These findings underpin the emerging concept of immunometabolism, which recognises metabolism and immunity as components of an integrated regulatory network [35]. During HS, endocrine adaptation, oxidative stress, mitochondrial dysfunction, nutrient partitioning, microbiome remodelling, and immune responses interact through interconnected signalling pathways that collectively determine the animal's capacity to maintain homeostasis. Metabolic disturbances directly influence immune function, while inflammatory mediators reciprocally regulate energy metabolism and nutrient allocation, explaining why prolonged HS simultaneously compromises productivity, health, welfare, and reproductive performance [25,35].

Collectively, current evidence supports a systems-level view of HS in which disruption of thermal balance initiates a coordinated adaptive cascade involving neuroendocrine regulation, oxidative stress, metabolic reprogramming, microbiome remodelling, and immune adaptation [19,25,35]. Rather than acting independently, these processes reinforce one another through interconnected molecular pathways, including NF- $\kappa$ B, Nrf2, mTOR, and heat shock proteins, ultimately determining the animal's capacity to withstand thermal stress. Variation in productive level, metabolic heat production, endocrine responsiveness, antioxidant capacity, immune plasticity, and microbiome composition further explains the marked differences in thermotolerance observed among animals exposed to similar environmental conditions [25,35].

This integrative perspective has shifted both research and livestock management beyond reliance on climatic indices or isolated physiological variables towards multidisciplinary approaches that combine environmental, physiological, biochemical, immunological, and molecular information [19,20]. Advances in genomics, transcriptomics, proteomics, metabolomics, and microbiome research have accelerated this transition by enabling the identification of biomarkers, regulatory pathways, and potential intervention targets for improving resilience to HS [25,29–31,35].

Taken together, the mechanisms described above provide the biological framework for understanding the production consequences of HS in dairy cattle. Disruption of thermoregulation, endocrine signalling, redox homeostasis, metabolism, microbiome composition, and immune function collectively impairs mammary gland activity and systemic physiological performance, ultimately reducing milk yield and quality while compromising animal health, welfare, and reproductive efficiency. These production responses are examined in the following sections of this review [19,25].

### 3.3. A Threat to Productivity and Product Quality

Productivity represents an integrated indicator of animal performance because it reflects the combined outcome of physiological, endocrine, metabolic, and immune processes that maintain homeostasis. Rather than being a direct consequence of elevated ambient temperature, the decline in productivity under HS should be viewed as the result of systemic adaptation, whereby energy and nutrients are redirected from productive functions towards thermoregulation, antioxidant defence, endocrine adaptation, and maintenance of homeostasis. Consequently, production losses may occur before feed intake declines substantially, highlighting HS as one of the major biological and economic constraints on modern livestock production under climate change [36,37].

These mechanisms have been investigated most extensively in dairy cattle, where the high metabolic demands of lactation generate considerable endogenous heat. Under such conditions, even moderate increases in the temperature–humidity index (THI) rapidly challenge the thermoregulatory capacity of high-producing cows. Pair-feeding studies further demonstrate that heat-stressed cows produce less milk than thermoneutral animals receiving the same amount of feed, indicating that production losses result primarily from endocrine and metabolic reprogramming, altered nutrient partitioning, and impaired mammary function rather than reduced feed intake alone [36,37].

Large-scale population studies consistently demonstrate the high sensitivity of milk production to thermal load, although reported THI thresholds vary considerably among studies owing to differences in physiological status, production level, climatic conditions, and study design. Analysis of 387,963 Holstein cows in Canada showed that milk yield declined significantly at approximately THI 68, whereas milk fat and protein responded much earlier, at THI values of about 57 and 60, respectively. Based on these findings, annual losses to the Canadian dairy industry were estimated at approximately CAD 34.5 million,

highlighting the substantial economic impact of both reduced milk yield and deteriorating milk composition [38].

Subsequent studies indicate that the productive response to HS follows a multi-threshold rather than a single-threshold pattern. Three critical THI ranges (47–50, 61–69, and 72–76) have been identified, with progressively greater declines in milk yield beyond each threshold. Above the highest threshold, milk losses increased to 0.04–0.23 kg per additional THI unit, whereas milk fat and protein consistently proved more sensitive than overall milk yield, suggesting that impairment of mammary synthetic activity precedes substantial reductions in milk production [39].

Physiological status further modifies thermal susceptibility. Lactating cows respond to HS at considerably lower THI values than dry cows, while the greatest production losses occur during mid-lactation, when daily milk yield may decline by nearly 20%, compared with approximately 14% during early and late lactation. These findings demonstrate that the productive consequences of HS depend not only on thermal load but also on metabolic intensity and physiological stage [40].

Collectively, current evidence indicates that the productive response to HS is determined by the interaction of thermal load, physiological status, production level, and management conditions. Despite variation in reported THI thresholds, studies consistently show that changes in milk composition precede declines in milk yield, making milk fat and protein among the earliest and most sensitive indicators of heat-induced impairment of mammary function.

Beyond reducing milk yield, heat adaptation is consistently accompanied by deterioration in milk quality. Whereas lactation performance reflects the overall physiological status of the animal, milk composition provides a more direct indicator of mammary synthetic activity. Consequently, changes in milk fat, protein, and other components are increasingly recognised as earlier and more sensitive indicators of HS than declines in milk yield itself.

This differential response reflects the varying susceptibility of mammary synthetic processes to thermal challenge. Disruptions in energy metabolism, mammary perfusion, and milk component synthesis develop before substantial reductions in milk secretion become apparent. As a result, alterations in milk composition often precede measurable production losses.

Field studies conducted under diverse climatic conditions consistently support this pattern. During summer, daily milk yield declined from 31.8 to 24.9 kg (−21.7%), accompanied by reductions in milk fat (3.53% to 3.30%), protein (3.16% to 3.12%), and solids-not-fat (8.29% to 7.95%), together with increases in somatic cell count and bacterial contamination. Significant negative correlations were observed between THI and milk yield, milk fat, protein, solids-not-fat, and milk pH, indicating that HS simultaneously compromises both production and the hygienic and technological quality of milk [41].

Further evidence demonstrates that individual milk components differ in their sensitivity to thermal load. Threshold THI values were approximately 72 for milk yield, 64 for milk fat, and 63 for milk protein, whereas increases in somatic cell count became evident only above THI 78. Beyond these thresholds, each additional THI unit reduced milk yield by up to 0.249 kg/day, milk protein by 0.008 kg/day, and milk fat by 0.006 kg/day per cow. Importantly, the detrimental effects of heat waves persisted for 7–12 days after thermal conditions had returned to normal, while consecutive hot days further amplified production losses, particularly in high-producing multiparous cows. These findings emphasise that the duration of thermal exposure is as important as its intensity [42].

At first glance, these observations appear inconsistent with controlled experiments, in which marked production losses are not always detected during the initial days of heat exposure. However, this discrepancy largely reflects differences in exposure duration rather

than conflicting biological responses. After five days of experimental HS, reductions in dry matter intake and changes in selected physicochemical characteristics of milk were already evident, whereas milk fat, lactose, total solids, somatic cell count, and milk yield remained unchanged. Simultaneously, the use of ventilation and sprinkler cooling increased milk protein concentration and alleviated physiological signs of HS. Thus, controlled experiments primarily capture the early, largely reversible phase of thermal adaptation, whereas field studies reflect the cumulative consequences of prolonged heat exposure [43].

Taken together, these findings demonstrate that milk composition responds to HS earlier than milk yield and therefore provides a more sensitive indicator of impaired mammary function. Moreover, the magnitude of these changes depends not only on the severity of thermal stress but also on its duration. Consequently, evaluating milk yield alone is likely to underestimate the biological and economic impact of HS, whereas integrating milk composition, hygienic quality, and their temporal dynamics offers a more comprehensive assessment of heat-induced impairment.

Deterioration in milk composition represents only the phenotypic manifestation of deeper alterations within the mammary secretory epithelium during HS. While conventional indicators such as milk fat and protein reflect the final outcome of these changes, advances in lipidomics, metabolomics, fatty acid profiling, and transcriptomics have shifted attention towards the cellular and molecular mechanisms underlying heat-induced mammary dysfunction. Consequently, recent research has focused less on describing changes in milk composition and more on understanding the metabolic reprogramming of the mammary gland during acute and chronic heat exposure.

Current evidence indicates that this reprogramming develops progressively. Early HS is characterised by compensatory responses that largely preserve mammary function, whereas prolonged heat exposure induces persistent alterations in energy, lipid, and protein metabolism, ultimately suppressing the synthetic activity of the mammary epithelium.

Differences in the duration of thermal exposure largely explain the apparent discrepancies among studies. Following short-term experimental HS ( $\text{THI } 76 \pm 4.1$  for two days), no significant changes were detected in the overall milk fatty acid profile or major phospholipid classes, with only minor alterations in several short-chain fatty acids that were largely attributable to dietary factors rather than heat stress itself. These findings suggest that mammary lipid metabolism remains largely preserved during the initial stages of thermal adaptation [44].

In contrast, prolonged HS results in progressive metabolic dysregulation rather than direct thermal injury to mammary cells. Reduced insulin-mediated nutrient partitioning, impaired mobilisation of non-esterified fatty acids, altered ruminal fermentation with decreased acetate and butyrate production, and systemic inflammation collectively restrict *de novo* fatty acid synthesis and gradually reduce milk fat concentration. Thus, milk fat depression should be regarded as the consequence of sustained metabolic reprogramming rather than an immediate effect of elevated temperature [45].

Transcriptomic studies have further expanded this concept by demonstrating that chronic HS is accompanied by persistent changes in the expression of genes involved in nutrient transport, lipid synthesis, mitochondrial energy metabolism, antioxidant defence, and cellular stress responses. These molecular alterations reflect a transition from transient functional adaptation to sustained metabolic reprogramming of the mammary epithelium, explaining the persistent suppression of *de novo* fatty acid synthesis, shifts in milk fatty acid composition, and the continued deterioration of milk processing characteristics even after thermal conditions have returned to normal [46].

Long-term field observations closely mirror these molecular findings. During 26 consecutive days of heat exposure ( $\text{THI} \geq 72$ ), milk fat declined from 3.73% to 3.50% (−6.2%),

milk protein from 3.24% to 3.03% (−6.5%), and solids-not-fat by approximately 7%, whereas average daily milk yield remained relatively stable. These results indicate that molecular disturbances accumulate progressively and become evident as changes in milk composition before substantial declines in milk yield occur. Consequently, economic losses may begin to develop well before reductions in production become apparent [47].

The magnitude of these responses is, however, strongly influenced by management practices. Under modern ventilation and cooling systems, correlations between thermal load and milk quality traits remained relatively weak ( $r = -0.21$  to  $-0.28$ ), suggesting that effective cooling substantially mitigates the adverse effects of HS. In contrast, a survey of 53 dairy farms identified much stronger negative associations between air temperature and milk fat ( $r = -0.68$ ), protein ( $r = -0.71$ ), and solids-not-fat ( $r = -0.65$ ), while somatic cell count increased with rising ambient temperature ( $r = 0.70$ ;  $p < 0.05$ ). In addition, total mixed ration (TMR) feeding improved resilience to thermal stress compared with conventional feeding systems. These findings emphasise that the biological consequences of HS are determined not only by its severity but also by the effectiveness of management strategies aimed at maintaining metabolic stability [48,49].

Collectively, evidence from molecular, experimental, and field studies supports a unified model of mammary adaptation to heat stress. Short-term exposure is characterised by predominantly reversible functional responses, whereas prolonged thermal stress induces stable metabolic and transcriptomic reprogramming, resulting in persistent impairment of mammary synthetic activity. These findings further demonstrate that the duration of heat exposure is as important as the absolute THI level in determining the transition from reversible adaptation to sustained deterioration in milk composition and the accumulation of economically significant production losses.

Although the effects of HS on productivity have been investigated most extensively in dairy cattle, growing evidence indicates that the underlying biological principles are broadly conserved across livestock species. Regardless of species, increasing thermal load activates similar adaptive responses aimed at maintaining thermal homeostasis. However, their productive consequences vary according to physiological characteristics, metabolic intensity, production purpose, and thermoregulatory capacity.

A common feature across species is the redistribution of energy and nutrients from productive functions towards thermoregulation and maintenance of homeostasis. In high-producing dairy cows, this is primarily expressed as impaired mammary synthetic activity, whereas in meat-producing animals the earliest manifestations are reduced growth performance and poorer feed efficiency. Thus, the same adaptive mechanisms are reflected through different production traits depending on species-specific biology.

This concept is supported by studies in small ruminants. Under acute heat exposure, goats lost approximately 0.5 kg of body weight for every 1 °C increase in ambient temperature, reflecting the rapid development of negative energy balance. These changes were attributed to reduced feed intake, increased energetic costs of thermoregulation, and suppression of anabolic processes, demonstrating that nutrient partitioning shifts from growth towards maintenance of homeostasis even in the absence of lactation [50].

Prolonged HS also compromises product quality across livestock species. In meat-producing animals, chronic thermal stress alters post-mortem glycolysis, muscle pH decline, water-holding capacity, colour stability, antioxidant status, and protein denaturation, thereby increasing the incidence of PSE (pale, soft, exudative) and DFD (dark, firm, dry) meat. As in dairy cattle, HS therefore affects not only production volume but also product quality and technological value [51].

The severity of these losses is strongly influenced by species-specific thermoregulatory capacity and the effectiveness of management interventions. In pigs, where physiological

heat dissipation is inherently limited, evaporative cooling increased final body weight by approximately 5 kg, reduced stocking density contributed a further 3 kg, and combining both strategies improved body weight by nearly 9 kg after an 81-day finishing period. Animals managed under the most effective cooling strategy reached 89.9 kg compared with 80.6 kg under conventional conditions, accompanied by lower hair cortisone concentrations, indicating that improved productivity reflected both enhanced growth and reduced chronic physiological stress [52].

Commercial poultry exhibits even greater susceptibility to HS because the absence of sweat glands and exceptionally high metabolic heat production substantially restrict heat dissipation. Consequently, heat exposure rapidly reduces feed intake, growth rate, feed efficiency, and breast muscle yield while increasing mortality and the incidence of heat-associated myopathies. Nevertheless, local cooling systems markedly reduce both mortality and muscle pathology, demonstrating that appropriate management can substantially alleviate production losses even in highly susceptible species [53,54].

Taken together, evidence from diverse livestock systems indicates that interspecies differences relate primarily to the rate and phenotypic expression of thermal adaptation rather than to its fundamental biological mechanisms. Across species, increasing thermal load consistently redirects physiological resources from production towards survival, making reduced productivity and declining product quality the earliest and most consistent manifestations of HS, although their specific expression depends on species biology and production system [55].

Overall, productivity should be regarded not merely as an economic outcome but as an integrated biological indicator of the animal's adaptive capacity under thermal stress. Importantly, production losses represent only the visible consequence of broader physiological, metabolic, and molecular disturbances. A comprehensive understanding of HS therefore requires productivity to be evaluated alongside animal health, reproductive performance, and welfare, which are discussed in the following sections of this review.

### 3.4. A Challenge to Animal Health, Welfare, and Reproduction

The consequences of HS extend far beyond impaired thermoregulation and reduced productivity. As discussed in the previous sections, prolonged thermal exposure triggers integrated endocrine, metabolic, and immune disturbances that progressively manifest as clinical disorders affecting mammary health, locomotion, reproduction, pregnancy, and fetal development. Rather than representing independent outcomes, these conditions should be viewed as interconnected stages of systemic maladaptation.

Unlike immediate production losses, many health consequences develop gradually and persist beyond the period of heat exposure. Their effects may become evident during the subsequent lactation or even in the next generation, emphasising that the biological burden of HS extends from early immunometabolic disruption to long-term physiological programming.

Among all physiological stages, transition cows are particularly vulnerable because the high metabolic demands of the periparturient period coincide with limited adaptive capacity. Within the framework of immunometabolism, negative energy balance, elevated concentrations of non-esterified fatty acids (NEFA), and ketone bodies directly alter immune-cell function, thereby increasing susceptibility to postpartum disease [56].

Large cohort studies strongly support this concept. In dairy cows, prepartum NEFA concentrations  $\geq 160 \mu\text{mol/L}$  were associated with increased risks of hyperketonaemia (OR = 2.0), displaced abomasum (OR = 12.3), metritis (OR = 9.4), and clinical mastitis (OR = 5.8) [57]. Similarly, analysis of 13,120 calvings demonstrated that increasing THI significantly increased the incidence of retained placenta, metritis, and ketosis, particularly fol-

lowing periods of sustained heat exposure [58]. Together, these findings indicate that postpartum disorders should be interpreted as manifestations of systemic immunometabolic dysregulation rather than isolated clinical diseases.

The mammary gland is among the organs most susceptible to this systemic imbalance. Whereas alterations in milk composition reflect impaired secretory activity, increases in somatic cell count and mastitis indicate disruption of local immune defence. Consistent with the mechanisms outlined in Section 3.2, HS impairs mammary immunity through oxidative stress, inflammatory signalling, altered gene expression, microbiome disruption, and reduced neutrophil function. Importantly, heat exposure during the dry period exerts prolonged effects that persist into the subsequent lactation, while cooling during this period improves postpartum neutrophil function and passive immune transfer to calves, demonstrating that immunological consequences extend beyond the period of thermal exposure itself [59,60].

Field studies further indicate that mastitis develops through the interaction between impaired host defence and environmental conditions. Rising ambient temperature increases somatic cell count and mastitis risk while simultaneously altering barn microclimate and bedding characteristics. Bedding moisture, pH, and surface temperature were identified as major determinants of subclinical mastitis, indicating that the effects of HS are partially mediated through changes in the housing environment and microbial exposure rather than through direct thermal injury alone [61].

Collectively, current evidence supports a dual-pathway model of mastitis development under HS. Systemic immunometabolic dysfunction increases host susceptibility, whereas deterioration of the housing microenvironment enhances pathogen challenge. Their interaction explains the marked increase in mammary disease observed during prolonged periods of thermal stress.

HS affects animal welfare through a progressive transition from adaptive behavioural responses to persistent functional impairment. Because welfare integrates behavioural, physiological, and pathological changes, it provides a more comprehensive assessment of the biological consequences of HS than individual clinical indicators.

The earliest manifestations of HS are behavioural adaptations that enhance heat dissipation, including reduced feed intake and physical activity, altered daily activity patterns, prolonged standing, shorter lying and rumination times, and increased competition for access to water, shade, or cooling devices. Although initially compensatory, these responses become detrimental during prolonged HS by increasing mechanical loading of the claws and predisposing animals to lameness.

The delayed nature of these disorders is particularly noteworthy. Following severe HS (THI = 88), the prevalence of sole ulcers increased from 15.2–18.6% to 28.1% three months after the heat event before subsequently declining to 12.9% ( $p < 0.05$ ), demonstrating that clinically important consequences may emerge long after environmental temperatures have returned to normal [62].

Once lameness develops, it initiates a secondary cycle of pain, metabolic disturbance, and reduced welfare. Although the following studies did not directly investigate HS-induced lameness, they illustrate the systemic consequences of this condition, the risk of which increases under prolonged HS. Lameness was associated with elevated cortisol concentrations ( $r = 0.630$ ;  $p < 0.001$ ), increased milk electrical conductivity, and alterations in blood urea nitrogen, the BUN/creatinine ratio, sodium, and copeptin concentrations, indicating widespread neuroendocrine and metabolic disruption and identifying potential biomarkers for objective welfare assessment [63,64].

Taken together, current evidence supports a sequential model of welfare deterioration during HS. Heat stress initially induces adaptive behavioural responses, prolonged stand-

ing subsequently increases the risk of claw disorders, and the resulting lameness further amplifies systemic metabolic and endocrine dysfunction, creating a self-reinforcing cycle of declining health and welfare.

A similar progression from adaptive responses to systemic dysfunction occurs in the reproductive system, whose function depends on the coordinated interaction of endocrine, metabolic, vascular, and immune pathways. HS disrupts reproduction at multiple stages, from oestrus expression and ovulation to fertilisation, early embryonic development, and maternal–embryonic interactions.

The earliest manifestations include reduced oestrus intensity and duration, impaired oestrus detection, hormonal dysregulation, suppressed follicular development, and disturbed ovulation. Consequently, conception rates decline, while days open and the service period increase, with fertility recovering more slowly than milk production following summer HS [65]. Even a 0.5 °C increase in body temperature has been associated with reduced conception, whereas pregnancy rates may decline by more than 20–30% during HS owing to impaired oocyte quality, endocrine disruption, and increased embryonic loss [66].

Large population studies further demonstrate that reproductive performance is affected before extreme THI values are reached. Analysis of more than two million records from Canadian Holstein cows identified fertility thresholds at approximately THI 66 (range 65.0–68.6 depending on parity and evaluation period), with the greatest sensitivity occurring from three days before to two days after insemination. Cows inseminated under synchronised breeding programmes were considerably more sensitive than those bred after natural oestrus detection (THI threshold 64 vs. 72.8), highlighting that both the intensity and timing of HS determine reproductive success [67].

Beyond endocrine regulation, HS directly compromises female gamete competence and early embryonic development. Elevated temperature impairs oocyte maturation, increases oxidative stress, disrupts meiosis, and reduces blastocyst development. Partial restoration of oocyte maturation and embryo development by supplementation with IGF-1 (100 ng/mL) further emphasises the importance of metabolic and growth-factor signalling pathways involved in reproductive thermotolerance [68].

HS also compromises male fertility, although this aspect has received less attention than female reproductive physiology. Elevated testicular temperature disrupts spermatogenesis, reduces sperm motility, viability, and fertilising capacity, increases sperm abnormalities and oxidative damage, and ultimately compromises semen quality in breeding bulls. Together, these findings indicate that successful reproduction under HS depends on preserving the functional competence of both female and male gametes [66].

However, direct hyperthermia alone cannot fully explain reproductive failure. Experimental studies suggest that widely used *in vitro* exposure to 40–41 °C may overestimate the actual thermal conditions experienced by reproductive tissues *in vivo*. During HS, uterine temperature typically increases by only about 0.5 °C and rarely exceeds 39 °C, even at THI  $\geq$  74. Nevertheless, a further increase of only 0.5 °C above 38.6 °C within the uterine lumen was associated with a 12% reduction in conception rate [69]. These observations indicate that reproductive losses arise not only from direct cellular heat damage but also from impaired ovarian perfusion, altered nutrient and hormone transport, and disruption of the reproductive microenvironment.

Collectively, current evidence indicates that reproductive dysfunction during HS cannot be attributed to a single mechanism. Population studies identify critical windows of thermal susceptibility, experimental models reveal cellular mechanisms of damage, and physiological observations demonstrate that even modest local increases in reproductive tract temperature may have important clinical consequences. Together, these findings

support the concept that reproductive failure during HS results from the combined effects of direct cellular injury and systemic maternal maladaptation.

The reproductive consequences of HS are inherently cumulative and often persist beyond the period of thermal exposure. Recovery of reproductive function may remain incomplete for weeks or months after environmental conditions return to normal. This concept is consistent with autumn low milk yield syndrome (ALMYS), in which reduced milk production, impaired fertility, increased mastitis incidence, and postpartum disorders are interpreted as interconnected manifestations of cumulative physiological exhaustion rather than independent seasonal events [70].

The effects of HS also extend beyond conception and early embryogenesis. During pregnancy, the placenta becomes the primary biological interface through which maternal HS influences fetal development. Hyperthermia, dehydration, impaired uteroplacental perfusion, systemic inflammation, and oxidative stress compromise placental structure and function, reducing the efficiency of oxygen and nutrient transfer to the fetus. Consequently, maternal HS increases the risks of intrauterine growth restriction, shortened gestation, stillbirth, and neonatal mortality, with placental dysfunction representing a central mechanism linking maternal thermal stress to adverse pregnancy outcomes [71].

Experimental evidence confirms these mechanisms. In dairy cows, HS during the dry period (THI 77–79) induced structural and functional placental alterations, shortened gestation by 3.0 days ( $p = 0.04$ ), and reduced calf birth weight by 3.4 kg ( $p = 0.03$ ) despite compensatory increases in placental vascularisation [72]. Similar trends have been reported in sheep, where a 1 °C increase in ambient temperature was associated with approximately a 3.5% reduction in lambing rate and a 0.6% decrease in lamb birth weight [73]. Despite species differences, both experimental and population studies identify the placenta as the principal biological interface linking maternal HS with impaired offspring development.

These observations underpin the concept of developmental programming, whereby prenatal HS influences offspring health, reproductive performance, and productivity long after birth. The long interval between prenatal exposure and postnatal outcomes complicates causal inference and highlights the importance of longitudinal cohort studies.

This delayed impact is particularly evident when HS occurs during the transition and dry periods. In Holstein cows, exposure to THI 71.4–72.7 increased the incidence of retained placenta, metritis, and mastitis, reduced pregnancy rate after first insemination by 10.6%, and increased the risk of culling during the first 90 days of lactation [74]. Similarly, analysis of 12,102 lactations showed that increasing THI during the dry period reduced pregnancy rate after first insemination by 6.1% and overall pregnancy rate by 17.6%, while increasing the number of inseminations per pregnancy and reducing 305-day milk yield [75]. Collectively, these findings identify the transition and dry periods as critical windows during which HS has lasting consequences for subsequent productivity, fertility, and health.

The consequences of HS may extend well beyond a single production cycle. In a cohort of 7331 Holstein cows, animals born in autumn after prenatal exposure to summer HS had a significantly shorter lifespan ( $1303 \pm 16$  vs.  $1385 \pm 17$  days;  $p < 0.01$ ), lower lifetime milk yield, and fewer completed lactations [76]. However, because season of birth is only a proxy for prenatal HS and may also reflect seasonal differences in nutrition, disease pressure, and management, these findings demonstrate a strong long-term association rather than definitive causality.

These observations indicate that the consequences of maternal HS may extend well beyond pregnancy and birth, affecting offspring development and subsequent productive performance. Evidence indicates that intrauterine HS is associated with reduced birth weight, impaired passive immune transfer, slower postnatal growth, delayed puberty,

shortened productive life, lower lifetime milk yield, and an increased risk of premature culling [76,77]. Although the long interval between prenatal exposure and postnatal outcomes complicates causal inference, the consistency of these associations strongly suggests that fetal development is the principal biological pathway through which the long-term consequences of maternal HS are transmitted across generations.

Mechanistic studies attribute these long-term effects to impaired placental development and reduced uteroplacental nutrient and oxygen transport, leading to persistent alterations in immune, endocrine, and metabolic development [78]. Supporting this concept, prenatal HS has been associated with structural remodelling of the adrenal gland together with transcriptomic changes affecting immune function, inflammation, prolactin signalling, calcium transport, and cellular regulation, indicating long-term programming of the hypothalamic–pituitary–adrenal axis and adaptive capacity [79]. Collectively, these findings identify placental dysfunction and fetal developmental programming as key biological mechanisms linking maternal HS with long-term alterations in offspring phenotype.

Overall, current evidence supports the concept of a biological memory of HS. Cohort studies consistently associate prenatal and periparturient HS with reduced health, fertility, productivity, longevity, and offspring performance, while mechanistic studies identify placental dysfunction and endocrine programming as the biological basis of these long-term effects. Although additional longitudinal studies are required to strengthen causal inference, the available evidence indicates that the consequences of HS may persist throughout the productive life of the offspring.

### 3.5. Early-Life Effects of HS and Programming of Future Productivity

The biological memory established by prenatal HS originates during specific developmental windows, when environmental conditions exert disproportionate effects on organ development and physiological plasticity. Fetal life, neonatal adaptation, and the pre-weaning period represent these critical windows, during which thermal exposure may induce persistent changes in physiological, metabolic, and productive phenotypes. This process, commonly described as developmental programming, establishes the biological limits of future productive performance [77].

Among the mechanisms underlying developmental programming, placental function occupies a central position because it represents the primary biological interface through which maternal HS is translated into long-term alterations in the offspring. During late gestation, hyperthermia alters placental morphology and vascularisation, impairs oxygen and nutrient transport, and modifies both gene expression and epigenetic regulation. Consequently, fetal development occurs within an altered metabolic and endocrine environment that influences organ growth, tissue differentiation, and the establishment of functional reserves [78].

Evidence from organ-specific studies demonstrates that placental dysfunction is accompanied by persistent developmental remodelling of endocrine organs. In calves born to cows exposed to HS during the final  $54 \pm 5$  days of gestation ( $\text{THI} > 68$ ), adrenal glands exhibited greater relative weight ( $0.08$  vs.  $0.06$  g/kg;  $p = 0.04$ ), increased size, and cortical thickening after weaning. Transcriptomic analysis further identified approximately 30 differentially regulated signalling pathways associated with inflammation, immune function, prolactin signalling, and calcium transport. Notably, circulating cortisol concentrations remained unchanged, indicating that conventional endocrine biomarkers may fail to detect substantial structural and molecular reprogramming of the stress-response system [79].

These developmental alterations extend beyond endocrine function to include thermoregulation and energy metabolism. Offspring born to dry cows with elevated  $\beta$ -hydroxybutyrate concentrations ( $0.64 \pm 0.02$  vs.  $0.21 \pm 0.005$  mmol/L) exhibited reduced

brown adipose tissue mass, lower mitochondrial density, suppressed expression of UCP1, CREBP4, and CPT1B, reduced abundance of UCP1 and PGC-1 $\alpha$  proteins, and impaired glucose tolerance. Complementary in vitro experiments demonstrated that  $\beta$ -hydroxybutyrate directly inhibited brown adipocyte differentiation and mitochondrial biogenesis, suggesting a mechanistic link between the maternal metabolic environment and prenatal programming of thermogenesis and energy homeostasis [80]. Considered alongside the endocrine findings, these results indicate that the long-term adaptive capacity of the offspring is shaped through coordinated remodelling of stress responsiveness, thermogenic capacity, and metabolic regulation [79,80].

The persistence of these effects after maternal heat exposure has ceased indicates that they cannot be explained solely by transient alterations in placental or maternal metabolism. Instead, increasing evidence points to epigenetic programming as a mechanism of biological memory, whereby stable modifications of gene regulation occur without changes in DNA sequence. Among these mechanisms, DNA methylation appears particularly important because of its potential influence on immune development, endocrine function, energy metabolism, and adaptive responsiveness.

Direct evidence for this mechanism has recently been reported in calves prenatally exposed to HS (THI > 68) during the final  $54 \pm 5$  days of gestation. At birth, 682,898 differentially methylated cytosines were identified, including 55,304 located within genes expressed in blood cells. Although many methylation differences diminished before weaning, reflecting postnatal epigenetic plasticity, 97,289 methylation sites (14.2% of the original profile) remained stable throughout early life, while the 500 most informative loci clearly distinguished calves according to prenatal HS exposure. These persistent changes were primarily associated with pathways regulating immune development, inflammation, metabolism, and cellular signalling, indicating that the epigenetic response comprises both dynamic and stable components. The latter may provide a foundation for future biomarkers of prenatal HS exposure and its long-term biological consequences [81].

Epigenetic modifications should therefore be viewed as one component of an integrated developmental response rather than as an isolated mechanism. Placental dysfunction, together with alterations in the prenatal endocrine environment, may reshape the regulatory networks governing growth, metabolism, stress responsiveness, and immune development. Likewise, the presence of stable DNA methylation patterns alone does not establish causality, as their biological significance ultimately depends on corresponding changes in gene expression, tissue development, physiological function, and productive performance.

Taken together, these findings support a multilevel model linking prenatal HS with persistent developmental alterations. Placental dysfunction establishes an adverse fetal environment, endocrine and metabolic remodelling influences organ development, and epigenetic modifications provide a potential mechanism through which these effects are retained after birth. Establishing definitive causal relationships, however, will require integrated longitudinal studies combining molecular, physiological, and lifetime productivity data [78,79,81].

After birth, the biological consequences of prenatal HS become evident through alterations in thermoregulation, energy metabolism, and metabolic adaptation. During the neonatal period, the functional reserves established in utero determine the newborn's ability to maintain thermal homeostasis, transition to enteral nutrition, and adjust its metabolism to extrauterine life. Consequently, even subtle impairments in functional maturity may reduce neonatal viability and influence subsequent developmental trajectories.

One of the earliest manifestations of developmental programming is the efficiency of neonatal thermoregulation, which is critical for survival during the first days of life. Ther-

mal homeostasis depends on the coordinated action of peripheral vasoconstriction, shivering, non-shivering thermogenesis mediated by brown adipose tissue, timely colostrum intake, and behavioural responses. Impaired maturation of these mechanisms increases the risk of perinatal mortality, which in buffalo calves, largely owing to hypothermia, may reach 3.0–17.9% [82]. Consistent with this concept, calves with greater birth weight (>46 kg), indicative of more favourable prenatal development, maintained significantly higher body surface temperatures throughout the first six days of life ( $p < 0.05$ ). These findings demonstrate a close relationship between prenatal development and postnatal thermoregulatory competence while highlighting infrared thermography as a valuable tool for assessing neonatal functional maturity [83].

Together, these observations indicate that the earliest consequences of developmental programming are expressed not through isolated physiological abnormalities but through the newborn's overall capacity to maintain thermal and metabolic homeostasis. Importantly, the final phenotype remains strongly influenced by the postnatal environment, which may either exacerbate or partially compensate for prenatal programming, emphasising that the biological consequences of maternal HS are modifiable rather than fully predetermined [82,83].

The biological consequences of prenatal HS persist well beyond neonatal adaptation, influencing postnatal growth, organ development, and ultimately future productivity. The mammary gland provides one of the clearest examples of such long-term programming because its morphogenesis begins long before the onset of lactation. Disturbances during early tissue development may therefore remain functionally silent until the animal enters productive life.

Structural evidence of this process is detectable soon after birth. Heifer calves born to cows exposed to HS during the final  $54 \pm 5$  days of gestation (mean THI = 79) exhibited reduced mammary gland, fat pad, and parenchymal mass, fewer and smaller mammary ducts, and approximately 35% lower parenchymal cell proliferation at birth and 27% lower proliferation after weaning ( $p < 0.05$ ), indicating that prenatal HS limits mammary secretory potential before lactation has even commenced [84].

However, developmental programming is not confined to the mammary gland. Maternal HS during the final trimester of gestation (THI 70.15–74.28) shortened gestation by approximately 1.4 days, increased the incidence of neonatal diarrhoea more than 2.6-fold during the first week of life, and altered endocrine (ACTH, GnRH, insulin), cellular stress (HSP70), oxidative status (SOD, LPO), and immune (TNF- $\alpha$ , IFN- $\gamma$ , IgG) biomarkers in the offspring. These findings indicate that prenatal HS induces coordinated remodelling of endocrine, metabolic, and immune function rather than isolated changes in individual organs [85].

Evidence from longitudinal studies indicates that these developmental alterations persist throughout growth. At 11–14 months of age, heifers prenatally exposed to maternal HS during the dry period exhibited lower body weight, reduced wither height, and approximately 24% less mammary parenchymal mass, accompanied by decreased proliferation of secretory tissue cells ( $p = 0.01$ ). Thus, changes initiated before birth remain detectable through puberty and may constrain mammary secretory capacity long before the first lactation [86].

Collectively, these observations identify postnatal growth as a critical intermediate stage linking prenatal programming with adult productive performance. They also emphasise the value of longitudinal studies that follow animals from fetal exposure to productive maturity, as only such designs can establish whether early developmental disturbances are subsequently expressed in the adult phenotype.

Recent research has substantially expanded the concept of developmental programming by incorporating the gastrointestinal microbiome and rumen development. Whereas earlier work primarily attributed the long-term consequences of maternal HS to altered growth, endocrine regulation, or organ development, increasing attention is now directed towards the establishment of microbial communities, maturation of the rumen epithelium, and their interactions with host metabolism.

After weaning, calves born to heat-stressed cows exhibited marked remodelling of the rumen microbiome, characterised by increased bacterial diversity and shifts in the relative abundance of major microbial taxa. These changes were accompanied by differential expression of ruminal epithelial genes involved in nutrient transport, cell proliferation, barrier integrity, and volatile fatty acid metabolism, indicating that maternal HS simultaneously influences the microbial ecosystem and functional maturation of the rumen epithelium [87]. Transcriptomic analyses further revealed differential regulation of genes associated with cell-cycle control, intercellular communication, immune function, tissue remodelling, and metabolite transport, providing molecular evidence that microbial restructuring is closely coupled with epithelial development [88].

Importantly, these molecular alterations have also been linked to productive performance. Associations between specific bacterial taxa, ruminal epithelial gene expression, and growth traits indicate that the rumen microbiome is not merely a passive consequence of postnatal nutrition or management but an active component of long-term physiological adaptation established before birth [89]. These findings identify the rumen as a major target of fetal programming through which maternal HS may influence nutrient utilisation, metabolic efficiency, and lifetime productive potential [87–89].

Overall, the available evidence indicates that prenatal HS should no longer be regarded as a transient environmental challenge affecting isolated physiological traits. Instead, it represents a trigger for multilevel biological programming encompassing organ development, endocrine and immune regulation, metabolic adaptation, establishment of the microbiome, and the lifelong expression of productive potential. Consequently, offspring health, welfare, and productivity are shaped to a considerable extent by biological processes initiated during fetal development.

These advances also have important practical implications. Effective mitigation strategies should extend beyond the protection of lactating cows to include cooling during the dry period, optimisation of the maternal thermal environment throughout critical stages of gestation, and appropriate management during early postnatal life, thereby supporting neonatal viability, tissue development, growth, and future productivity [90,91]. Assessment of the biological impact of HS should therefore encompass the entire life course, from fetal development to adult productive performance. Protecting cows from HS during gestation should be regarded not only as a welfare intervention but also as a fundamental strategy for safeguarding the health, productivity, and adaptive capacity of future generations [92].

### 3.6. Biomarkers and Monitoring of HS: From Detection to Prediction

Although THI remains the most widely used measure of environmental heat load, no single climatic index consistently predicts the biological response to HS. Diagnostic performance varies according to the physiological endpoint considered. The Comprehensive Climate Index, which incorporates wind speed and solar radiation, showed the strongest associations with skin ( $r = 0.845$ ), rectal ( $r = 0.672$ ) and ocular temperatures ( $r = 0.617$ ), whereas the Equivalent Temperature Index for Dairy Cattle was more closely related to respiratory rate ( $r = 0.850$ ) [93]. Among THI-derived metrics, THI\_PST best predicted tympanic temperature ( $r = 0.75$ ;  $R^2 = 53\%$ ), compared with 44% of the variation explained by conventional THI, while associations with respiratory rate remained weaker across all in-

dices evaluated ( $r = 0.27\text{--}0.34$ ) [94]. Climatic indices therefore capture different components of thermal exposure, and their value depends on the biological outcome being assessed.

Recognition of this limitation has encouraged a shift from static environmental measurements towards metrics that account for the duration and temporal pattern of exposure. A composite five-day THI measure improved daily milk-yield prediction by 21% ( $R^2 = 0.73$ ; Lin's concordance coefficient = 0.84), confirming that cumulative heat load may be more informative than single-day conditions [95]. The Grazing Heat Load Index applies a similar principle to pasture-based systems by combining air temperature, solar radiation and wind speed. However, its performance also declines during extreme weather, illustrating the inherent limitations of environment-based assessment alone [96]. Climatic indices are therefore most useful as measures of external challenge rather than direct indicators of the animal's biological response.

Physiological variables provide a more immediate indication of thermoregulatory strain. Respiratory rate and peripheral body temperature generally respond before sustained hyperthermia because they reflect the initial activation of heat-dissipation mechanisms. In cows with low immune responsiveness, respiratory rate exceeded 50 breaths/min at  $\text{THI} \geq 75\text{--}77$ , whereas highly responsive cows reached this threshold only at approximately  $\text{THI} 80$ . Rectal temperature remained within  $38.5\text{--}39.3^\circ\text{C}$  and rumination changed little over the same range, indicating that respiratory rate is more sensitive to moderate thermal challenge, although its response varies with individual adaptive capacity [97].

The sequence in which physiological variables change also provides information on the progression of thermal strain. Forehead skin temperature responded at ambient temperatures of  $24.1\text{--}24.2^\circ\text{C}$ , followed by respiratory rate at  $24.4^\circ\text{C}$  and ocular temperature at  $24.6^\circ\text{C}$ , whereas vaginal and muzzle temperatures increased later, at approximately  $25.3$  and  $25.4^\circ\text{C}$ , respectively [98]. Surface temperature and respiratory rate therefore characterise the compensatory phase of thermoregulation, while increases in core temperature indicate that heat dissipation is becoming insufficient. Their diagnostic value lies in this temporal complementarity rather than in the selection of a single optimal physiological measure.

Laboratory biomarkers extend HS assessment beyond observable thermoregulatory responses by characterising systemic metabolic and cellular disturbances. NEFA, BHB, glucose, indices of protein metabolism, liver enzymes, oxidative status and haematological variables remain useful for evaluating changes in energy balance, hepatic function and immune homeostasis [56–58]. However, because many of these measures respond to several metabolic and pathological conditions, they are generally more informative when interpreted as part of a broader biomarker profile.

HSP70 is among the most sensitive molecular indicators of cellular heat strain. At  $\text{THI} > 80$ , its concentration nearly doubled, while cortisol increased more than fivefold, despite comparatively limited changes in conventional metabolic and haematological variables [99]. In high-producing cows, increased HSP70 expression was accompanied by higher cortisol and IL-6 concentrations, lower total antioxidant capacity and poorer reproductive performance [100]. HSP70 was positively associated with THI and the number of inseminations per pregnancy, whereas elevated cortisol was related to reduced pregnancy rates. These markers represent distinct dimensions of the response to HS and consequently have greater diagnostic value when evaluated jointly rather than in isolation.

Despite its high sensitivity to acute HS, HSP70 expression is tissue-specific and primarily reflects the early cellular response to thermal challenge. Consequently, it may not accurately represent prolonged or chronic HS and should be interpreted together with complementary physiological, endocrine, inflammatory and oxidative biomarkers.

Non-invasive matrices offer an additional means of characterising prolonged exposure. Unlike blood, saliva or milk, which predominantly represent the current physiological state, hair, earwax and other tissues may accumulate glucocorticoids over several weeks or months, enabling retrospective assessment of sustained hypothalamic–pituitary–adrenal axis activity. Their interpretation nevertheless requires consideration of tissue-specific hormone deposition and metabolism.

No cumulative matrix appears universally applicable across species or production conditions. In pigs, hair cortisone and the cortisone-to-cortisol ratio were more informative than cortisol concentration alone, indicating that local glucocorticoid metabolism influences the assessment of chronic heat load [101]. Milk HSP70 provides a different non-invasive opportunity because it can be incorporated into routine sampling without additional animal handling [102]. Redox profiling may further improve diagnostic resolution when biomarkers of oxidative damage, including isoprostanes, AOPP, 8-Oxo-dG and MDA, are considered together with TAC and antioxidant enzyme activity. By contrast, TBARS or MDA alone lack sufficient specificity for robust assessment of oxidative stress [103]. Combining molecular indicators, redox profiles and cumulative matrices therefore permits simultaneous evaluation of cellular strain, metabolic consequences and exposure duration [104].

Behavioural responses add a functional dimension because they represent the combined outcome of thermoregulatory, metabolic and motivational adjustments. Their diagnostic value is not confined to the total duration of feeding, rumination, lying or standing, but also includes changes in behavioural sequence and spatial organisation. Alteration of the characteristic lying posture with the head turned backwards preceded reductions in total lying time and the prolonged standing behaviour commonly associated with HS [105]. Specific postural patterns may therefore reveal subtle thermal discomfort before conventional time-budget measures change substantially.

PLF technologies have made continuous and objective behavioural assessment feasible under commercial conditions. Automated rumination detection using nasal pressure sensors and accelerometers achieved sensitivities of 99.1–100% and specificities of 87.8–95.0% relative to visual observation [106]. In postpartum cows exposed to HS, the combination of reduced rumination, lower activity and increased rumen temperature distinguished animals that subsequently developed postpartum disorders before overt clinical signs appeared [107]. Behavioural data can therefore support both welfare surveillance and the identification of animals at increased risk of functional or clinical impairment.

Digital assessment has subsequently progressed from recording individual variables to combining climatic, behavioural, physiological and production data within decision-support frameworks. A five-level HS assessment system incorporating THI, dry matter intake, milk yield, milk composition, mastitis and lameness demonstrated that different exposure metrics correspond to different biological outcomes. Daily maximum THI was most strongly associated with changes in milk production and composition, whereas cumulative heat load and elevated night-time THI were more informative for mastitis and lameness [108]. This distinction emphasises the need to match the temporal scale of exposure with the outcome being predicted. Collar-based systems apply the same principle at the animal level by simultaneously recording rumination, feed intake, activity and respiratory rate [109].

Infrared thermography has expanded the possibilities for rapid, non-contact assessment of body surface temperature. The ocular region, muzzle, udder and tail base show the most consistent associations with rectal temperature, respiratory rate and related physiological measures [110]. However, solar radiation, air movement, camera characteristics, recording angle and distance can materially affect temperature estimates. Thermography

should therefore be regarded as a complementary measurement requiring standardised acquisition rather than as a direct substitute for core-temperature assessment.

The integration of sensors, computer vision and AI has enabled the development of more complex prediction models. Body surface temperature has been associated with rumen temperature, panting and posture, supporting its use within multimodal models of thermal strain [111]. Rumen bolus data analysed using machine-learning algorithms increased prediction sensitivity to 79%, compared with 34–68% for conventional climate-based models [112]. Nevertheless, the diagnostic value of a sensor depends on the biological relevance of the variable recorded. Rumen temperature showed little association with THI in one study, whereas rumination duration was substantially more responsive to increasing heat load [113]. Sensor selection should therefore be guided by biological validity rather than technological availability alone.

Machine-learning models can further identify non-linear relationships among climatic, physiological, behavioural and production variables. Models combining rumination, rumen pH and milk-quality data achieved high classification performance, with Random Forest reaching an AUC of 0.99 and an overall accuracy of 98.3%, outperforming SVM, KNN and ANN approaches [114]. Computer-vision systems have also advanced non-contact phenotyping. A modified YOLOv8 model integrating infrared and RGB images recognised anatomical regions with an accuracy exceeding 93% and estimated body temperature with a mean absolute error of 0.40 °C [115]. These results demonstrate strong technical potential for automated herd-level surveillance.

High internal accuracy does not, however, guarantee practical applicability. Many current algorithms have been developed and tested within a single herd, breed or production environment, increasing the risk that performance will decline when they are transferred to different populations or climatic conditions. External validation, calibration across management systems and transparent identification of the variables driving predictions are therefore essential. The value of AI-based systems will ultimately depend on robustness, reproducibility and biological interpretability rather than on classification accuracy alone.

A further development is the analysis of temporal patterns rather than isolated observations. Combining continuous rumination data with cumulative indicators such as hair cortisol and blood fructosamine improved the characterisation of chronic stress [116]. Individual 24 h behavioural profiles allow each animal to serve as its own reference, increasing sensitivity to deviations that may remain undetected when fixed population thresholds are applied. The related concept of a herd signature identifies departures from established group-level daily rhythms and may reveal emerging disturbances before clinical manifestations become apparent [117]. In this context, the trajectory of change is often more informative than the absolute value of an individual measurement.

Overall, HS assessment is moving from isolated environmental thresholds and biological measurements towards systems that combine data across different temporal and functional scales. Multidimensional models incorporating routine milk records, welfare indicators, sensor outputs and biochemical measures can identify distinct health, metabolic and adaptive profiles at both animal and herd levels [118]. The principal contribution of digital technologies is therefore not the replacement of conventional indicators, but their integration into predictive frameworks that characterise individual resilience, anticipate adverse outcomes and support precision management under increasing thermal challenge.

### 3.7. Nutritional Strategies for Improving Resilience to HS

HS profoundly disrupts energy metabolism, nutrient utilisation and gastrointestinal function, reducing dry matter intake (DMI), impairing nutrient digestibility, and promoting oxidative stress, systemic inflammation and negative energy balance. Nutritional interven-

tions have therefore become an integral component of comprehensive HS management, complementing environmental modification and genetic improvement rather than merely compensating for nutrient deficits [119].

Reduced DMI, typically by 10–30%, is among the earliest consequences of HS and accounts for only 35–50% of the associated decline in milk yield. The remaining losses arise from altered energy partitioning and increased maintenance requirements. Current feeding programmes therefore focus on increasing dietary energy density, optimising rumen fermentation and incorporating functional feed additives to maximise nutrient-use efficiency rather than simply stimulating feed intake [120].

Feed efficiency during HS also has a substantial genetic component. Analysis of 6333 Holstein cows comprising nearly 390,000 daily feed-intake records revealed moderate heritability of feed intake ( $h^2 = 0.16\text{--}0.33$ ) together with negative genetic correlations between thermoneutral and HS conditions (up to  $-0.48$ ), indicating that highly productive cows are particularly susceptible to disruption of energy balance. These findings support integrating nutritional and genetic approaches to enhance thermotolerance [121].

Increasing dietary energy density through rumen-protected fat remains one of the most effective nutritional strategies. Supplementation with 0.70 kg/day increased 4% fat-corrected milk without affecting DMI or physiological variables. Subsequent studies demonstrated that production responses depend primarily on fatty acid composition rather than total fat content. Palmitic acid (16:0) most effectively increased milk fat and energy-corrected milk yield, oleic acid (18:1) enhanced fatty acid digestibility, whereas stearic acid (18:0) reduced digestibility and limited productive responses. Combining palmitic and oleic acids therefore provides the greatest production benefit [122,123].

Beyond increasing dietary energy supply, lipid supplementation also modifies rumen function. Palmitic acid, either alone or combined with stearic and oleic acids, enhanced fibre degradation, increased short-chain fatty acid production and shifted the rumen microbiome towards greater abundance of *Fibrobacter* and *Prevotella*, accompanied by reduced *Ruminococcus* and *Butyrivibrio*. These findings demonstrate that dietary lipids improve nutrient utilisation not only by increasing energy availability but also by optimising rumen fermentation and microbial ecology during HS [124]. However, productive responses remain context-dependent, being influenced by diet composition, stage of lactation, production level and the severity of HS. Therefore, the optimal fatty acid strategy should be selected according to specific production conditions.

Current concepts of protein nutrition have shifted from correcting crude protein deficiencies towards targeted supplementation with functional amino acids. Because reduced DMI coincides with increased metabolic demands associated with thermoregulation and immune activation, dietary efficacy depends increasingly on amino acids that support cellular adaptation in addition to protein synthesis. Methionine, lysine, arginine, glutamine and tryptophan participate in pathways regulating antioxidant defence, intestinal integrity, stress responses and cellular metabolism, making them promising targets for nutritional intervention during HS [125].

Controlled feeding trials strongly support this concept. Supplementation with rumen-protected methionine nearly doubled plasma methionine concentrations (59 vs. 30  $\mu\text{M}$ ;  $p < 0.001$ ) and preserved immunometabolic homeostasis despite HS-induced increases in serum amyloid A, haptoglobin, lipopolysaccharide-binding protein and IL-1 $\beta$  [126]. Likewise, rumen-protected L-tryptophan increased milk yield by 11.3%, reduced rectal temperature and cortisol concentrations, and enhanced HSP70 and HSP90 expression, whereas zinc methionine increased milk yield by 4.9% while lowering malondialdehyde concentrations by 24% [127,128].

Despite their differing modes of action, these supplements converge on common physiological outcomes, including enhanced antioxidant capacity, attenuation of inflammatory responses and more efficient nutrient utilisation. Functional amino acid supplementation is therefore increasingly recognised as a strategy for promoting metabolic adaptation and thermotolerance rather than simply correcting protein deficiencies [125–128].

Oxidative stress is a central component of the systemic response to HS, making antioxidant support an essential element of nutritional intervention. Under severe HS (THI = 88.2), small ruminants exhibited increased malondialdehyde concentrations, reduced total antioxidant capacity and impaired metabolic homeostasis, highlighting that effective interventions must preserve not only redox balance but also energy metabolism, inflammatory regulation and cellular adaptation [129].

L-theanine exemplifies this multifunctional approach. Supplementation improved nutrient digestibility, increasing acid detergent fibre digestibility by 10.4% and milk yield by approximately 1.8 kg/day while reducing malondialdehyde concentrations and enhancing antioxidant enzyme activity. Likewise, combined supplementation with silymarin and selenium yeast proved more effective than either component alone, whereas betaine enhanced antioxidant defence, improved productivity and maintained osmotic homeostasis. Collectively, these findings demonstrate that interventions targeting multiple physiological pathways provide greater adaptive benefits than strategies based solely on antioxidant activity [130–132].

Similar multifunctional properties have been reported for other nutraceuticals. Ferulic acid improved growth performance and energy metabolism while reducing rectal temperature and respiratory rate in growing sheep, demonstrating concurrent effects on thermoregulation, metabolism and oxidative status. Their efficacy depends less on chemical origin than on the biological pathways they modulate, including antioxidant defence, inflammatory regulation, cellular metabolism and osmotic balance. Consequently, nutritional programmes tailored to the predominant mechanisms of HS are likely to provide the greatest practical benefit [133].

The gastrointestinal microbiome is increasingly recognised as a central mediator of systemic adaptation to HS rather than merely a component of digestive function. Heat stress disrupts rumen fermentation, alters microbial composition, impairs intestinal barrier integrity and promotes systemic inflammation, driving the development of dietary strategies that modulate the gut and rumen microbiota while preserving digestive efficiency, immune competence and metabolic stability.

Probiotic supplementation represents one of the most effective approaches to achieving this objective. In lactating Holstein cows, *Saccharomyces cerevisiae* increased DMI by 1.3 kg/day and milk yield by 1.8 kg/day while increasing IgA, IgG and IgM concentrations, reducing TNF- $\alpha$  and IL-1 $\beta$ , and improving antioxidant status. Comparable benefits have been reported across livestock species. In poultry, probiotics increased body weight gain by 5–12% and reduced mortality by 20–40%, accompanied by enhanced antioxidant defence and reduced lipid peroxidation [134,135].

Collectively, these findings establish the gut microbiome as a major biological target of nutritional intervention. By enhancing nutrient utilisation, limiting inflammation and maintaining metabolic stability, microbiome-directed strategies substantially strengthen thermotolerance under HS [134,135].

Phytogenic feed additives have emerged as promising nutritional interventions because of their ability to influence multiple mechanisms underlying HS. Their biological effects extend beyond antioxidant activity to include modulation of inflammatory responses, intestinal barrier integrity, heat shock protein expression and the gut microbiome. However, considerable variation in plant composition, dosage and supplementation protocols contin-

ues to limit the development of universally applicable recommendations, underscoring the need for standardised formulations and long-term production studies [136].

Additional evidence from feeding trials supports this concept. In lactating Holstein cows, a phytogenic blend reduced cholesterol concentrations in milk by approximately 26% and in plasma by 15%, while increasing lactose and selected unsaturated fatty acids without affecting milk yield. Together with earlier evidence on ferulic acid, these findings indicate that individual phytogenic compounds preferentially influence metabolic status, thermoregulation or product quality according to their bioactive composition [133,137].

Rather than replacing antioxidants or probiotics, phytogenic compounds are best regarded as complementary components of integrated nutritional programmes. Their capacity to simultaneously regulate oxidative balance, inflammatory activity, metabolism and the gut microbiome underpins their considerable adaptive potential, although wider commercial implementation will depend on greater standardisation and validation under commercial production conditions [136,137].

Beyond supporting physiological adaptation to HS, modern nutritional strategies are increasingly recognised as tools for improving the environmental sustainability of dairy production. Feed interventions that reduce enteric methane emissions while maintaining productive performance contribute not only to animal resilience but also to improved nutrient-use efficiency and a lower carbon footprint [138]. Meta-analyses consistently identify *Asparagopsis* spp. and 3-nitrooxypropanol as the most effective methane-mitigating additives, whereas lipid supplementation, nitrates, condensed tannins, and polyunsaturated fatty acids generally provide moderate but biologically meaningful reductions without compromising milk production [139,140]. Their environmental benefits can be further quantified through integrated modelling approaches combining rumen fermentation models with life cycle assessment, enabling evaluation of carbon mitigation at both farm and agri-food system levels [141,142].

Collectively, these findings indicate that no single nutritional intervention provides universal protection against HS. Instead, the greatest benefits are achieved through integrated, context-specific nutritional strategies that simultaneously improve metabolic homeostasis, oxidative stress, immune regulation, rumen function, and environmental efficiency.

Taken together, the evidence presented in Sections 3.2–3.7 demonstrates that HS should be regarded as a systemic biological condition involving coordinated responses across multiple levels of biological organisation, from thermoregulation and cellular homeostasis to metabolism, immune function, reproduction and long-term adaptive capacity. These interconnected mechanisms underpin the broad spectrum of productive, health and welfare consequences observed in dairy cattle and provide the biological foundation for the development of effective adaptation strategies. The principal biological domains, their underlying mechanisms, major consequences and key supporting evidence are synthesised in Table 1.

**Table 1.** Biological mechanisms and major consequences of heat stress in dairy cattle: From disrupted homeostasis to lifetime and intergenerational effects.

| Biological Domain                     | Key Mechanisms                                                                                                                                                                                                                                                                                         | Major Consequences                                                                                                                                                                                                                        | Key References    |
|---------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------|
| Thermoregulation and heat dissipation | Hypothalamic integration of thermal signals; increased respiratory rate and peripheral vasodilation; redistribution of blood flow towards the skin; increased water intake; reduced activity; behavioural avoidance of heat; progressive loss of heat dissipation efficiency during prolonged exposure | Increased body temperature and respiratory effort; reduced lying and feeding activity; cumulative heat storage; failure of nocturnal recovery; transition from compensated thermal strain to hyperthermia and thermoregulatory exhaustion | [19–21,93–98,108] |

Table 1. Cont.

| Biological Domain                                           | Key Mechanisms                                                                                                                                                                                                                                                                                             | Major Consequences                                                                                                                                                                                                                                              | Key References                     |
|-------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------|
| Neuroendocrine adaptation                                   | Activation of the hypothalamic–pituitary–adrenal axis; increased CRH, ACTH and cortisol secretion; altered prolactin signalling; modification of water, electrolyte and energy homeostasis; endocrine plasticity dependent on physiological status and individual thermotolerance                          | Redistribution of resources towards survival; altered stress responsiveness; impaired lactation and reproductive signalling; reduced conception; persistent endocrine dysregulation after heat exposure                                                         | [22,23,65–69,79,99,100]            |
| Oxidative stress and cellular homeostasis                   | Excessive ROS production; activation of NF- $\kappa$ B and suppression of Nrf2-dependent antioxidant defence; mitochondrial dysfunction; impaired ATP synthesis and calcium homeostasis; endoplasmic reticulum stress; autophagy, apoptosis and heat shock protein responses                               | Oxidative damage to lipids, proteins and DNA; chronic inflammation; impaired tissue function; reduced cellular resilience; dysfunction of the liver, mammary gland, skeletal muscle, intestinal epithelium and reproductive tissues                             | [24,25,68,99,100,103,104,125–129]  |
| Metabolic and nutrient partitioning                         | Reduced dry matter intake; increased maintenance requirements; altered insulin signalling; preferential use of glucose and amino acids for thermoregulation, immune activation and tissue repair; impaired lipid mobilisation; negative energy balance; suppression of anabolic pathways                   | Lower feed efficiency; reduced availability of substrates for milk synthesis and reproduction; increased susceptibility to ketosis and other transition disorders; progressive decline in productive efficiency beyond the effects of reduced feed intake alone | [25–28,36,37,45,56–58,119–124]     |
| Mammary gland function and milk synthesis                   | Reduced mammary perfusion, oxygen supply and glucose uptake; inhibition of mTOR signalling; decreased expression of amino acid transporters and milk protein genes; altered lipid metabolism; transcriptomic reprogramming of mammary epithelial cells                                                     | Reduced milk yield; earlier declines in milk fat and protein than in milk volume; altered fatty acid composition; poorer technological and hygienic milk quality; increased somatic cell count; persistent production losses after heat exposure                | [26–28,36–49,59,60,84,86]          |
| Gastrointestinal function, barrier integrity and microbiome | Heat-induced remodelling of the ruminal microbiome; reduced abundance of fibrolytic bacteria; altered carbohydrate and nitrogen metabolism; changes in short-chain fatty acid production; impaired epithelial barrier integrity; increased intestinal permeability and translocation of microbial products | Reduced fibre digestion and energy harvest; altered nutrient availability; amplification of systemic inflammation; impaired immune competence; variation in feed efficiency, milk composition and individual thermotolerance                                    | [25,29–31,87–89,113,124]           |
| Immune remodelling and disease susceptibility               | NF- $\kappa$ B-mediated inflammatory activation; altered PBMC, macrophage, monocyte and neutrophil function; concurrent pro-inflammatory and regulatory responses; disruption of immunometabolic signalling; reduced mammary and uterine immune defence                                                    | Increased susceptibility to mastitis, metritis, retained placenta and postpartum disorders; delayed disease manifestation after heat exposure; impaired passive immune transfer; prolonged recovery and increased culling risk                                  | [25,32–35,56–61,74,75,100,107,118] |

Table 1. *Cont.*

| Biological Domain                                  | Key Mechanisms                                                                                                                                                                                                                                                                                                               | Major Consequences                                                                                                                                                                                                                                                         | Key References      |
|----------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------|
| Productivity and product quality                   | Systemic diversion of energy and nutrients away from production; impaired mammary synthetic activity; reduced feed intake and nutrient digestibility; cumulative effects of heatwave duration and insufficient recovery; interaction with production level and lactation stage                                               | Lower milk yield and energy-corrected milk; reduced fat, protein and solids-not-fat content; increased somatic cell count and bacterial contamination; reduced economic performance; persistent production losses for 7–12 days or longer after thermal conditions improve | [36–49,95,108]      |
| Reproductive function and pregnancy maintenance    | Impaired oestrus expression and detection; altered gonadotrophin and ovarian signalling; reduced follicular development and oocyte competence; oxidative damage to gametes; altered uterine perfusion and microenvironment; impaired maternal–embryonic communication                                                        | Lower conception and pregnancy rates; increased embryonic loss; prolonged days open and service period; more inseminations per pregnancy; slower reproductive recovery than recovery of milk yield                                                                         | [65–70,74,75,100]   |
| Placental function and developmental programming   | Reduced uteroplacental perfusion and nutrient–oxygen exchange; altered placental vascularisation and gene expression; endocrine and metabolic reprogramming; persistent DNA methylation changes; altered adrenal, brown adipose tissue, mammary gland and ruminal development                                                | Shorter gestation; lower birth weight; impaired neonatal thermoregulation and passive immunity; slower growth; reduced mammary development; altered microbiome establishment; lower fertility, lifetime milk yield, longevity and adaptive capacity                        | [71–92]             |
| Behaviour, locomotion and animal welfare           | Reduced feeding and rumination; increased standing; shorter lying periods; altered posture and daily activity rhythms; competition for water, shade and cooling; prolonged mechanical loading of claws; pain-associated neuroendocrine responses following lameness                                                          | Thermal discomfort; impaired behavioural expression and rest; increased risk of sole ulcers and lameness; chronic pain and stress; reduced mobility and feeding activity; deterioration in physical health and affective state                                             | [62–64,105–110,117] |
| Integrated resilience and biological heterogeneity | Interaction among thermoregulatory capacity, endocrine responsiveness, antioxidant defence, immune plasticity, microbiome composition and genetic background; coordinated genomic, transcriptomic, epigenomic, metabolomic and phenotypic networks; dynamic, multilevel adaptation rather than reliance on individual traits | Marked individual and breed differences in heat tolerance; variable recovery trajectories; identification of resilient and susceptible phenotypes; transition from descriptive heat stress assessment towards predictive, systems-level selection and management           | [97,116–118,121]    |

Note: Key references were selected based on their direct relevance to the biological mechanisms summarised within each domain.

### 3.8. Management and Technological Adaptation of Production Systems to HS

Active cooling remains one of the most effective approaches for mitigating HS, although its benefits do not increase proportionally with cooling intensity or duration. Under moderate thermal conditions, cooling systems generally maintain body temperature within the physiological range. During prolonged heat waves, however, even intensive

cooling often fails to prevent hyperthermia, indicating that technological interventions have physiological limits once environmental heat gain exceeds the animal's capacity for heat dissipation [143].

Cooling performance is governed primarily by the mechanism of heat transfer rather than cooling intensity alone. Local delivery of cooled air to the head reduced respiratory rate, maintained feed intake and improved feeding behaviour without significantly affecting body temperature or milk yield [144]. In contrast, automated sprinkler systems simultaneously lowered body temperature and respiratory rate, increased feed intake and lying time, and improved milk yield by approximately 1.5 kg/cow/day [145]. These contrasting responses indicate that air cooling mainly alleviates respiratory heat load, whereas evaporative cooling provides more efficient whole-body heat dissipation.

Mechanistic modelling supports these physiological observations. Convective cooling remained beneficial only while a favourable temperature gradient existed between the animal and the surrounding air. Increasing air velocity to 2.0 m/s delayed the onset of body temperature elevation from approximately 22 to 26 °C, whereas its cooling capacity declined rapidly above 28 °C. Wetting the body surface introduced an additional evaporative pathway, increasing total heat dissipation by 162% compared with convection alone, while optimisation of sprinkler cycles further enhanced cooling performance by approximately 19% and reduced unnecessary water use. Nevertheless, evaporative cooling became progressively less beneficial under high relative humidity because evaporation was constrained by air saturation [146].

Accordingly, cooling systems should operate adaptively rather than continuously at maximum capacity. The relative contribution of convective and evaporative heat loss varies with ambient temperature, humidity and the physiological state of the animal, making dynamic adjustment of cooling intensity more appropriate than fixed operating schedules.

The benefits of active cooling are closely linked to ventilation performance. Modern ventilation systems therefore focus on optimising airflow distribution within the animal-occupied zone rather than simply increasing air exchange. Similar ventilation rates may produce markedly different cooling responses depending on building geometry, air-distribution systems and airflow patterns.

Experimental studies and computational fluid dynamics (CFD) modelling consistently demonstrate that ventilation performance is determined by airflow distribution rather than airflow volume alone. In perforated air-distribution systems, a discharge angle of 45° generated the most uniform airflow (1.8–2.0 m/s) within the occupied zone, whereas smaller angles directed airflow away from the animals and larger angles caused premature upward deflection. CFD simulations further showed that realistic representation of building geometry, animal heat production and ventilation characteristics is essential for predicting local airflow patterns and temperature gradients, highlighting the importance of optimising air distribution instead of simply increasing fan capacity [147,148].

Accurate evaluation of ventilation likewise requires detailed assessment of spatial airflow patterns. In naturally ventilated dairy barns, vertical ultrasonic anemometer profiles combined with trapezoidal integration reduced discrepancies between inlet and outlet airflow estimates to −5–11%, whereas conventional central-point measurements produced deviations of 20–36%. Bidirectional airflow occurred in approximately 38% of observations when wind direction was nearly parallel to side-wall openings, indicating that measurement accuracy depends more on sensor placement than on sensor number [149].

The influence of external aerodynamics has also been demonstrated under commercial naturally ventilated housing systems. Although these observations were obtained in pig facilities, the underlying aerodynamic principles are broadly applicable to livestock buildings with large side-wall openings. Wind direction substantially altered airflow

patterns, while the optimal measurement strategy varied with airflow configuration. Grid-based sampling reduced measurement error to below 5%, although bidirectional airflow under specific wind conditions required considerably denser spatial sampling [150].

Rather than maximising air exchange, contemporary ventilation systems aim to deliver air precisely where it is most needed. Consequently, successful environmental control relies on optimised airflow distribution, reliable environmental monitoring and adaptive ventilation strategies that maximise heat dissipation while avoiding unnecessary energy consumption.

Unlike active cooling, which enhances heat dissipation after heat load has accumulated, passive strategies reduce heat gain before physiological strain develops. Shading therefore represents the first line of defence against HS and an essential component of climate-resilient dairy production systems.

The benefits of shade depend not only on its presence but also on the availability of sufficient protected space. Under severe HS (THI > 80), increasing shaded area to 9.6 m<sup>2</sup> per animal reduced rectal temperature by approximately 0.3 °C, decreased respiratory rate by 15–20 breaths/min and improved average daily gain by 8–10%. However, differences between 2.4 and 9.6 m<sup>2</sup> per animal were considerably smaller than those between shaded and unshaded conditions, indicating that ensuring simultaneous access to shade is more important than continually expanding shaded space beyond a practical threshold [151].

Shade can be provided by either artificial structures or natural vegetation. Silvopastoral systems reduced afternoon air temperature by approximately 2 °C, lowered body surface temperature by 1.5–3.0 °C and decreased respiratory rate from 55–60 to around 40 breaths/min compared with open pastures. Unlike artificial shelters, trees simultaneously modify solar radiation, airflow, humidity and ground temperature, creating a more favourable microclimate without additional energy inputs [152].

Across production systems, the physiological benefits of shading are generally more consistent than improvements in productivity. In temperate regions, shading enhanced respiratory comfort and animal welfare without significantly affecting growth performance [153]. Under subtropical conditions (THI > 84), by contrast, it reduced rectal temperature by 0.4–0.6 °C, lowered respiratory rate by 20–30 breaths/min and increased average daily gain by 10–12%, particularly when solid-roof shelters were used [154]. These observations suggest that productive responses become increasingly apparent as thermal challenge intensifies.

The complementary roles of passive and active cooling are clearly illustrated by integrated environmental management. Combining shade, mechanical ventilation and evaporative cooling reduced rectal temperature by 0.3–0.8 °C, decreased respiratory rate by 15–35 breaths/min, increased dry matter intake by 3–8% and improved milk yield by approximately 1–3 kg/day, whereas individual interventions produced substantially smaller responses. These synergistic effects reflect the distinct physiological functions of each technology: shading limits radiant heat gain, ventilation promotes convective heat exchange, and evaporative cooling enhances latent heat loss [155].

This integrated approach is fully consistent with recent recommendations from the European Food Safety Authority (EFSA), which identifies HS as one of the major welfare risks affecting dairy cattle. Thermoregulatory disturbances may begin at a THI of approximately 68, whereas severe HS becomes increasingly likely above a THI of 78. Current guidance therefore recommends combining shade, adequate ventilation and evaporative cooling with environmental, physiological and behavioural monitoring to enable timely intervention [156].

Housing management represents another key component of thermal resilience. In compost-bedded pack barns, maintaining optimal bedding moisture, regular tilling, adequate resting space and appropriate ventilation supports composting, reduces moisture accumulation and improves both udder health and thermal comfort [157]. Likewise, natu-

rally ventilated loose-housing systems remain vulnerable during periods of elevated THI, when natural airflow alone is often insufficient to dissipate excess heat. Supplementing natural ventilation with mechanical airflow and sprinkler or evaporative cooling substantially improves physiological responses, although overall performance continues to depend on housing design, resting area management and microclimatic conditions [158].

Collectively, these studies demonstrate that the success of cooling programmes depends less on individual technologies than on their coordinated implementation within the housing environment. The greatest improvements in thermal comfort, welfare and productivity are achieved when passive cooling, active cooling, ventilation and housing management operate as complementary elements of an integrated environmental control strategy.

Although engineering interventions remain fundamental for mitigating HS, their success increasingly depends on timely implementation. Consequently, conventional reactive cooling strategies are being replaced by precision livestock farming (PLF), where continuous monitoring and artificial intelligence enable intervention before severe physiological strain develops.

The transition towards PLF began with automated environmental monitoring. Wireless sensor networks continuously measure temperature, relative humidity, and concentrations of CO<sub>2</sub> and NH<sub>3</sub> with high accuracy ( $R^2 > 0.99$ ; error  $< 0.3$  °C and approximately 2%), allowing spatial variation within livestock buildings to be detected while supporting automated control of ventilation and cooling systems. Integration of these environmental measurements with wearable biosensors and computer vision has further enabled continuous assessment of heart rate, respiratory rate, activity and other physiological responses. Automated video-based estimation of respiratory rate, for example, achieves high accuracy ( $R^2 = 0.97$ ; MAE = 1.81; RMSE = 2.32 breaths/min) in less than 2 s per recording, providing a reliable basis for early detection of HS and individualised management [159–161].

The most significant advance of recent years, however, has been the transition from data collection to intelligent decision support. Machine-learning algorithms integrate information on milk yield, milk conductivity, rumination, activity, feed intake and environmental conditions into predictive models capable of identifying mastitis and metabolic disorders several days before clinical signs appear. Ensemble algorithms such as Random Forest and XGBoost achieve accuracies exceeding 90% (AUC  $> 0.90$ ) up to 3–7 days before disease onset, while personalised models incorporating individual circadian patterns further improve predictive performance (Accuracy  $> 95\%$ ; AUC  $\approx 0.97$ ) and predict heat-related stress 24–48 h before clinical manifestation [162,163].

This technological evolution reflects a broader shift from threshold-based management towards predictive, animal-centred decision support. Instead of responding to predefined environmental limits, PLF increasingly detects deviations from each animal's normal physiological profile, allowing management decisions to be tailored to individual responses rather than herd-wide averages.

Nevertheless, digital technologies cannot compensate for inadequate environmental control. Their greatest value lies in supporting adaptive management, where real-time sensing, artificial intelligence and engineering systems operate together to optimise cooling strategies while improving animal welfare and production efficiency.

Environmental sustainability has become an equally important consideration when evaluating HS mitigation strategies. Increasing ventilation effectively reduces heat load but may increase NH<sub>3</sub> emissions by approximately 10–20% owing to enhanced mass transfer from manure surfaces. In contrast, sprinkler systems provide efficient cooling without substantially increasing NH<sub>3</sub> emissions, whereas the performance of evaporative cooling remains strongly influenced by ambient temperature and relative humidity. As global

warming increases the demand for cooling, balancing animal welfare, resource use and atmospheric emissions will become an increasingly important challenge [164].

Achieving this balance requires coordinated housing design and manure management. Because 50–80% of  $\text{NH}_3$  emissions originate from barn floors and manure accumulation areas, automated manure removal, improved manure channels, low-emission flooring and optimised airflow can reduce emissions by 20–60% without compromising thermal comfort [165]. Emission mitigation must also extend beyond the livestock building. According to the ALFAM2 model, most  $\text{NH}_3$  losses occur within the first 24 h after slurry application, whereas immediate soil incorporation, slurry injection or trailing-hose spreading systems, together with weather-adapted application strategies, reduce emissions by 30–80% [166]. Effective climate adaptation therefore requires coordinated management of cooling, ventilation and manure handling throughout the entire manure management chain.

Environmental performance should likewise be assessed beyond  $\text{NH}_3$  emissions alone, because dairy production also contributes to  $\text{CO}_2$  and  $\text{CH}_4$  emissions. Improving ventilation without addressing emission sources may simply transfer pollutants from indoor to outdoor environments, whereas integrating low-emission housing design, efficient manure management, optimised ventilation and environmental monitoring provides substantially greater environmental benefits [167].

Overall, the evidence reviewed in this section demonstrates that HS mitigation is evolving from isolated cooling technologies towards integrated climate-resilient production systems. The effectiveness of modern adaptation strategies depends on coordinated interactions among housing design, ventilation, cooling technologies, digital monitoring and manure management rather than on any single intervention. Future dairy production will therefore rely on adaptive environmental control that integrates engineering solutions, precision livestock farming and resource-efficient management to simultaneously improve animal welfare, sustain productivity and reduce environmental impacts.

### 3.9. Genetic and Breed-Related Mechanisms of Thermotolerance and Resilience

Pronounced breed differences provide compelling evidence for a substantial genetic contribution to heat tolerance. Under identical feeding and management conditions, Holstein cows maintained higher milk yield, mounted a stronger immune response and resolved the acute stress phase more rapidly, whereas Brown Swiss cows showed more persistent oxidative and metabolic adjustments. These included increased concentrations of reactive oxygen metabolites, ceruloplasmin, bilirubin and  $\beta$ -hydroxybutyrate, greater liver enzyme activity and reduced paraoxonase activity, without a proportional loss of productivity. Thus, highly productive and more heat-adapted breeds appear to rely on distinct physiological strategies when coping with HS [168].

A similar contrast was observed between Holstein and Jersey cattle exposed to high ambient temperature and direct solar radiation. Jerseys maintained dry matter intake relative to body weight more effectively and exhibited greater heat tolerance, whereas Holsteins showed a larger decline in feed intake. The absence of marked biochemical changes following active cooling suggests that these breed-specific differences primarily reflect behavioural and physiological adjustments rather than extensive metabolic remodelling [169]. Breed effects therefore extend beyond production capacity to encompass distinct adaptive phenotypes.

Substantial variation also occurs within breeds, creating opportunities for selective improvement. An integrated TOPSIS assessment showed that approximately 22% of cows maintained both a lower body temperature and greater milk production under comparable heat-load conditions. This degree of within-population diversity indicates

meaningful scope for selecting animals that combine productive performance with superior thermal tolerance [170].

Inherited morphological characteristics provide an additional phenotypic expression of heat adaptation. Coat structure and pigmentation influence heat transfer and the absorption of solar radiation, making them potentially useful indicators of thermal sensitivity. Hair cortisol, an integrated marker of chronic HS, was consistently higher in dark- than in light-pigmented hair [171,172]. In Holstein cows, the proportion of black pigmentation was also associated with skin temperature, core body temperature and heat tolerance index ( $p < 0.05$ – $0.01$ ) [173]. Similarly, sheep with shorter and less dense fleece displayed greater phenotypic stability across climatic conditions ( $r^2 = 0.22$  vs.  $0.38$ ) [174]. Readily measurable coat traits may therefore complement physiological phenotyping and facilitate the identification of animals with favourable adaptive characteristics.

Modern genomic technologies have moved the study of heat tolerance beyond external phenotypes towards its underlying genetic architecture. Genome-wide analysis in Holstein cattle identified more than 13 million SNPs, of which 3554 were significantly associated with thermal tolerance, while functional annotation prioritised 61 candidate genes involved in cell survival, apoptosis and immune regulation. The most informative variants were subsequently incorporated into a molecular marker panel for early prediction of adaptive potential. This extensive distribution of associated loci confirms that the trait is governed by multiple interacting regulatory networks rather than by a small number of major genes [175].

Transcriptomic evidence reveals how this genetic architecture is functionally expressed during HS. A total of 428 differentially expressed genes were identified, including *HSP90AA1*, *HSPA1A*, *DNAJB1*, *VEGFA*, *JUN*, *FOS*, *ATF3* and *DUSP1*, reflecting coordinated regulation of protein homeostasis, oxidative balance, inflammation and cell survival [176]. More broadly, over 100 candidate genes, including *HSP70*, *HSP90*, *HSF1*, *ATP1A1*, *SLICK*, *TLR4*, *TNF*, *IL10*, *IFNG*, *PRL* and *PRLR*, converge on a relatively limited set of conserved pathways, notably MAPK, PI3K-Akt, NF- $\kappa$ B, FoxO and JAK-STAT. Genetic improvement should therefore focus on the collective behaviour of biological networks rather than the effects of isolated loci [177].

Nevertheless, individual functional genes may retain value as practical markers. *HSP70*, one of the most extensively studied examples, protects cellular proteins against thermal damage. In sheep, 11 SNPs within this gene were identified, several of which were associated with rectal temperature, respiratory rate and heat tolerance index. Animals carrying favourable alleles exhibited less pronounced physiological responses to heat exposure [178]. Although derived from another livestock species, these results illustrate how candidate-gene information can complement genome-wide prediction and support the cross-species relevance of conserved heat-shock pathways.

Recognition of the polygenic basis of heat tolerance has enabled its inclusion in contemporary breeding programmes. Heritability estimates for thermotolerance-related production traits are generally low to moderate. In Canadian Holstein cattle, heritability increased from 0.20 to 0.23 for milk yield and from 0.14 to 0.16 for protein yield as THI exceeded the heat-stress threshold, while remaining approximately 0.17 for fat yield, indicating sufficient additive genetic variation for genetic improvement [179]. Despite relatively low heritability, large-scale evaluations indicate that sustained genetic progress is feasible.

The practical utility of such selection has been demonstrated using the Heat Tolerance Australian Breeding Value (HT ABV). At THI 79, cows with higher HT ABV maintained lower body temperature, showed smaller increases in respiratory rate and preserved milk production more effectively than animals with lower values, despite comparable dry matter intake. Their advantage therefore appeared to arise from more efficient thermoregulation rather than reduced nutrient intake, confirming the value of HT ABV for early genetic selection [180].

Predictive accuracy may be further strengthened by combining genomic estimates with physiological phenotypes. HT ABV was significantly related to eye temperature, rectal temperature and respiratory rate, and also showed favourable associations with composite production and health indices. The inclusion of readily measurable physiological traits can therefore improve discrimination between animals with contrasting genetic capacity to tolerate HS [181].

These developments are increasingly reflected in national evaluation systems, where genomic information is combined with THI records, physiological measurements, production data and automated sensor outputs. Breeding is consequently moving beyond the identification of individual markers towards the prediction of animal performance under commercially relevant heat-load conditions [182].

Accounting for genotype-by-environment ( $G \times E$ ) interactions is essential because genetic merit is conditional on the environment in which it is expressed. Analysis of 4.79 million test-day records showed that above approximately THI 72, milk yield heritability declined from 0.17 to 0.13, while genetic correlations between performance under thermoneutral and heat-stress conditions fell to 0.79–0.82. The resulting reranking of Brown Swiss sires indicates that reaction norm models may estimate breeding value more accurately than approaches that assume genetic effects remain constant across climatic conditions [183].

At the molecular level, environmental sensitivity is mediated partly through regulation of gene expression. Multi-omics analyses integrating genomic, transcriptomic and epigenomic data have implicated DNA methylation, histone modifications, microRNAs and long non-coding RNAs in the modulation of cellular responses without alteration of the underlying DNA sequence. These regulatory layers help explain why the same genotype can produce contrasting phenotypes in different environments and suggest that epigenetic information may eventually complement conventional genomic markers [184].

Evidence from other livestock species supports the broader relevance of this principle. Analysis of more than 57,000 body-weight records in quail revealed heat-dependent changes in both heritability estimates and genotype ranking. Failure to model environmental sensitivity may therefore reduce selection accuracy across species, rather than solely in dairy cattle [185].

Methodological advances in genetic evaluation reinforce the broader shift towards functional performance. Single-step genomic evaluation combines phenotypic, pedigree and genomic information to predict production, health and longevity simultaneously. In a dataset exceeding 5.8 million lactation records, the inclusion of correlated functional traits increased the reliability of evaluations for young sires by approximately 5–10% compared with conventional models [186]. Reflecting the same change in breeding priorities, functional traits now contribute 35–60% of national selection indices across 10 European countries, with increasing emphasis on longevity, fertility, udder health, calving performance, feed efficiency and hoof health rather than milk yield alone [187].

Resilience indicators extend this framework by quantifying the ability of animals to maintain function or recover following disturbance. Their heritability estimates range from 0.02 to 0.13, revealing modest but exploitable genetic variation. More resilient cows generally produce slightly less milk but maintain higher fat and protein concentrations, illustrating a trade-off between maximum output and functional robustness [188]. Under sustained heat load, favourable genotypes have also been associated with better fertility, shorter service periods and lower climatic sensitivity, supporting the inclusion of resilience in breeding objectives for heat-prone production systems [189].

Overall, heat tolerance is best understood as a complex phenotype shaped by genomic architecture, physiological regulation and environmental exposure, rather than by individual genes alone. Further progress will require harmonised phenotyping, validation of

genomic predictions across populations and production systems, and explicit evaluation of the balance between climatic resilience and sustained productive performance.

### 3.10. A Challenge to the Economic and Environmental Sustainability of Livestock Production

HS can no longer be regarded solely as a constraint on animal productivity, health or welfare. In a warming climate, it increasingly shapes the economic viability, environmental footprint and social acceptability of dairy production. Responses to thermal stress should therefore be evaluated not only by their capacity to limit biological damage, but also by their effects on resource use, production costs, greenhouse gas emissions, animal welfare and the long-term resilience of farming systems. This broader perspective requires economic, environmental, technological and social outcomes to be considered as interconnected components of sustainability rather than as independent objectives.

The economic burden of HS extends well beyond reduced milk yield. It also encompasses poorer feed efficiency, increased water demand, impaired reproduction, greater disease risk and the additional energy and management costs associated with heat abatement. In South Korean dairy farms exposed to  $\text{THI} \geq 73$ , summer milk production declined by approximately 5–8%, whereas maintaining May-like microclimatic conditions was estimated to increase farm profit by up to KRW 12.25 million while reducing greenhouse gas emissions by almost 30 kg  $\text{CO}_2$ -eq per day [190]. Substantial losses have also been documented under temperate conditions. At  $\text{THI} 74.4$ , daily milk yield declined by 1.25–2.78 kg per cow, corresponding to EUR 0.46–0.55 per cow per day and approximately EUR 219–481 per day in large commercial herds [191]. Moderate heat exposure can therefore generate considerable cumulative losses when it persists across large numbers of animals.

The estimated magnitude of these losses depends strongly on how thermal exposure is quantified. In Chile, an adjusted temperature–humidity index incorporating solar radiation and wind speed predicted milk losses 2.0–6.4 times greater than those estimated using conventional THI. The associated economic burden reached USD 91.5–184.2 per cow and USD 29.0–108.4 million across the national dairy sector [192]. In Tunisia, productivity began to decline at three-day mean THI values of only 65–67, despite prolonged seasonal exposure to  $\text{THI} > 72$ , and cumulative losses amounted to 110–142 kg of milk per cow [193]. Universal thresholds and simplified indices may thus underestimate financial risk where solar load, air movement, herd productivity or acclimatisation differ from the conditions under which conventional THI thresholds were established.

Economic evaluation should consequently move beyond the valuation of avoided milk losses to consider investment costs, operating expenditure, expected returns and the useful life of heat-abatement infrastructure. Evidence from 800 *Bos indicus* × *Bos taurus* bulls showed that appropriately designed shade structures, oriented north–south and providing approximately 2.7 m<sup>2</sup> of shade per animal, improved average daily gain, feed efficiency and hot carcass weight. Capital costs were recovered within the first production cycles [194]. Although derived from beef cattle, these findings illustrate a principle relevant across livestock systems: well-targeted thermal protection can generate returns through several production pathways rather than through output preservation alone.

Taken together, the available evidence shows that economic risk is determined by the interaction of climatic intensity, exposure duration, herd productivity, farm scale, assessment method and management capacity. This heterogeneity limits the transferability of universal cost estimates. Farm-level decisions should instead be informed by locally calibrated heat-load indicators and cost–benefit analyses that account for both direct production losses and the broader consequences of thermal stress for health, reproduction, resource demand and production stability.

The economic consequences of declining performance are closely linked to environmental outcomes. When productivity falls, more feed, water, land and greenhouse gas

emissions are required per unit of saleable product. Environmental performance is therefore more meaningfully assessed through emission and resource-use intensity than through absolute input use alone. Improvements in feeding, genetic selection, reproductive management, veterinary care and animal welfare can increase biological efficiency and reduce emissions per kilogram of milk without requiring a reduction in total output [195]. The relevant question is not simply whether an intervention uses additional resources, but whether it lowers the overall environmental burden of the production system.

Life cycle assessment provides a framework for tracing these effects across the dairy supply chain. Farm-level processes, particularly enteric fermentation, manure management and feed production, account for approximately 75–89% of the carbon footprint of dairy products, identifying primary production as the principal target for mitigation. In a local dairy chain, conservation practices such as soil-protective cultivation and rotational grazing offset approximately 17% of emissions, while improvements in transport and logistics provided further reductions [196]. Although interventions beyond the farm gate remain relevant, the largest mitigation potential lies in measures that improve animal performance and resource use within the farm itself.

Cooling systems illustrate why environmental trade-offs should be evaluated in relation to output rather than inputs alone. Their direct contribution to the total water footprint was only 0.04%, whereas the absence of cooling increased the water footprint per unit of milk by approximately 1.1% because of lower productivity [197]. Optimised cooling also remained more resource-efficient than the no-cooling scenario despite additional electricity demand. Increased use of water or energy does not therefore necessarily imply poorer environmental performance when it prevents larger efficiency losses elsewhere in the system. Nevertheless, the net balance depends on local water availability, the source of electricity, climatic severity and system design, making targeted optimisation preferable to unrestricted resource use.

The translation of environmental performance into market value is increasingly pursued through sustainability certification and product labelling. More than 129 European labelling schemes currently incorporate sustainability-related criteria, many of which draw on life cycle assessment. However, heterogeneous standards, complex certification procedures, high auditing costs and the absence of harmonised metrics continue to restrict participation, particularly among small and medium-sized farms [198]. More credible and accessible frameworks will need to integrate carbon footprint, water and energy use, animal welfare and economic performance without imposing disproportionate administrative or financial burdens on producers.

Climate adaptation and environmental mitigation should therefore not be treated as inherently competing objectives. Measures that preserve animal health and productive output can reduce emissions and resource use per unit of milk, although their net effects must be assessed across the full production system. Evaluations based solely on the direct consumption of water or electricity risk misrepresenting the environmental consequences of heat-abatement strategies.

These environmental and economic considerations are inseparable from the social dimension of dairy production, which encompasses animal welfare, working conditions, public confidence and farmers' capacity to adopt new practices. Protection against HS has both ethical and financial significance because inadequate thermal management compromises animal welfare while increasing production losses and reputational risks. Investment in housing, cooling and welfare-oriented management may generate returns through improved health and performance, fewer losses and greater consumer valuation of responsibly produced food [199]. Animal welfare should therefore be regarded as a central component of production resilience rather than as an external constraint.

Credible welfare assessment increasingly relies on animal-based measures (ABMs) that directly capture behavioural, physiological and health outcomes instead of inferring welfare solely from housing conditions or resource provision. Protocols developed according to the principles of Welfare Quality<sup>®</sup> provide a structured basis for this approach. One such system comprised four principles, 12 criteria and 36 indicators, most of which were animal-based. Its application on 14 commercial farms enabled the classification of overall welfare status and identified deficiencies in microclimate, lighting, resource provision, bedding quality and lameness control [200,201]. These tools can support corrective action, investment prioritisation and certification, although their widespread application requires trained personnel, suitable infrastructure and consistent monitoring. Such requirements may be particularly difficult to meet on smaller farms with limited financial and technical capacity.

Adoption is shaped not only by awareness of environmental or welfare benefits, but also by farmers' broader socioeconomic circumstances. Among owners of small dairy farms, willingness to introduce greenhouse gas mitigation measures was positively associated with satisfaction with their financial situation ( $\rho = 0.42$ ), improved working conditions ( $\rho = 0.32$ ) and the possibility of spending more time with family ( $\rho = 0.34$ ). The strongest association was observed between having concrete mitigation ideas and willingness to implement them ( $\rho = 0.59$ ), while the perceived need for financial support was also closely related to adoption intentions ( $\rho = 0.54$ ) [202]. Nevertheless, 84% of respondents had never received funding for farm modernisation, only 22% considered emission-reduction technologies feasible, and just 32% expressed readiness to implement them.

These results reveal a persistent gap between technical potential and practical feasibility. Evidence that an intervention is effective does not guarantee its uptake when producers lack capital, labour, advisory support or confidence in the expected return. Policies for climate-resilient dairy production should therefore combine financial incentives with extension services, staff training and decision-support tools tailored to farms with different scales, resources and technological capacities.

PLF may help bridge these economic, environmental and social dimensions by enabling continuous monitoring, earlier intervention and more precise use of labour and resources. Computer-vision systems can identify individual animals, quantify feeding and behavioural patterns, assess physiological and reproductive status and detect early signs of disease in real time. When integrated with wearable sensors, automated milking systems and artificial intelligence, these technologies can support productivity, welfare surveillance and management precision [203].

Their value also extends beyond immediate farm management. Large-scale digital phenotyping can support selection for heat tolerance, resilience, longevity and resource-use efficiency, thereby linking short-term management responses with long-term genetic improvement. Integrating sensor-derived phenotypes with genomic information may also increase the accuracy of breeding-value estimation for traits that are difficult or expensive to measure conventionally [204]. PLF is thus evolving from a collection of monitoring tools into an infrastructure for coordinated management, phenotyping and selection.

Digitalisation, however, is not intrinsically sustainable. Its benefits depend on data quality, interoperability, affordability, accessibility, cybersecurity and the ability to translate automated outputs into effective decisions. Unequal access may widen existing disparities between large commercial enterprises and smaller farms, while poorly validated algorithms can produce misleading recommendations. Standardised data collection, transparent validation and compatibility among sensor, farm-management and genetic-evaluation platforms are therefore prerequisites for the responsible deployment of digital systems at scale.

Current evidence indicates that sustainable dairy production under increasing heat load cannot be secured through isolated interventions. Economic viability, environmen-

tal performance, animal welfare, farmer capacity, technological management and genetic improvement are interdependent elements of the same production system [205]. Cooling may require additional water and energy while reducing emissions and resource use per kilogram of milk; welfare improvements may involve initial expenditure while supporting productivity and public confidence; and digital monitoring may facilitate earlier intervention while remaining inaccessible to farms without sufficient capital or technical expertise.

Future adaptation should therefore be guided by integrated decision frameworks that compare biological effectiveness, investment requirements, operating costs, resource demand, welfare outcomes and environmental consequences under specific regional and production conditions. The most sustainable strategy will not necessarily maximise any single outcome, but should maintain productive performance and animal welfare while limiting environmental burdens and remaining economically and socially feasible.

### 3.11. From Omics Technologies to Climate Resilient Livestock Production

The study of HS in livestock has evolved from describing isolated physiological responses to understanding the complex biological networks that determine adaptive capacity. Rather than representing a single adaptive trait, thermotolerance is now recognised as a systems-level phenotype that emerges from interactions among molecular, cellular, physiological, behavioural and environmental processes. This conceptual transition has been driven by advances in high-throughput molecular technologies, digital phenotyping, bioinformatics and computational biology, making it possible to investigate adaptive responses across multiple levels of biological organisation instead of focusing on individual physiological traits. As a result, HS research has progressed from descriptive physiology towards systems biology, providing the foundation for predicting resilience rather than simply documenting its biological consequences [206].

Advances in omics technologies have greatly expanded understanding of the molecular mechanisms underlying thermal adaptation. Joint analysis of genomic and transcriptomic data enables the simultaneous identification of genetic variants associated with thermotolerance alongside transcriptional responses to HS. Using weighted single-step genome-wide association studies (WssGWAS) combined with RNA sequencing in Holstein cattle, researchers identified multiple quantitative trait loci, thousands of differentially expressed genes and several promising candidate genes, including PMAIP1, TMEM33 and CHORDC1. Despite the relatively low heritability of physiological indicators of heat adaptation ( $h^2 = 0.03\text{--}0.06$ ), these genes represent valuable molecular markers for breeding programmes aimed at improving thermotolerance [206].

Subsequent research has moved beyond individual candidate genes towards interconnected regulatory pathways governing thermoregulation, energy metabolism, immune function and cellular protection. This systems-oriented perspective recognises resilience as the product of coordinated biological networks rather than isolated “heat-resistance genes”, providing a more realistic framework for genomic improvement under future climatic conditions [207].

This concept has been further advanced through multiomics, which integrates genomic, transcriptomic, epigenomic, metabolomic, microbiome and phenotypic information within unified analytical frameworks. Such strategies reveal coordinated molecular, metabolic and microbial responses that cannot be resolved from individual datasets alone [208]. Their potential is illustrated by studies combining RNA sequencing, untargeted metabolomics and 16S rRNA sequencing of the nasopharyngeal microbiome, which identified shared signalling pathways across transcriptomic and metabolomic datasets and reconstructed coordinated biological responses to HS rather than isolated molecular alterations [209].

The biological relevance of multiomics becomes even more apparent when molecular data are linked with phenotypic traits. Joint analyses of the rumen microbiome, LC-MS metabolomics and milk production demonstrated that greater adaptive capacity was associated with coordinated changes in microbial composition and metabolism, accompanied by higher milk protein and lactose concentrations together with lower somatic cell counts ( $p < 0.05$ ). These findings establish a direct connection between molecular regulation and productive performance [210]. More recently, Bayesian network analysis has been integrated with immune profiling, GC-TOF-MS metabolomics and uterine microbiome characterisation to reconstruct causal relationships among metabolic dysfunction, inflammation, microbial dysbiosis and disease development. Although originally developed to investigate metritis, this systems-based framework demonstrates how multiomics can generate mechanistic models readily applicable to HS and other complex adaptive conditions [211].

The translation of multiomics into herd management has accelerated the evolution of PLF from an automated monitoring system into a predictive decision-support framework. Modern PLF integrates wearable and stationary sensors, computer vision, environmental monitoring and farm management records to generate continuous digital phenotypes describing activity, rumination, feed and water intake, body temperature, respiration rate, behaviour and microclimatic conditions. Continuous acquisition of these data enables physiological disturbances to be detected before clinical signs or production losses become evident, supporting earlier intervention under HS conditions [212].

As livestock datasets become increasingly complex, AI has emerged as a central component of modern herd management. Machine learning, deep learning and computer vision can identify subtle behavioural and physiological alterations, predict productive performance and disease risk, and analyse heterogeneous datasets beyond the capability of conventional statistical approaches. By combining sensor-derived information with environmental variables, including temperature, relative humidity and thermal indices, AI can anticipate HS events and optimise cooling strategies, housing management and other preventive interventions [213]. Predictive accuracy alone, however, is insufficient for successful implementation. The concept of farmer-centred AI therefore emphasises transparent decision-support systems that complement human expertise, making successful adoption dependent not only on algorithm performance but also on sensor infrastructure, interoperability, economic feasibility and user confidence [214].

Beyond advances in AI, digital livestock farming as a whole has progressed more rapidly in monitoring technologies than in automated management. Wearable sensors currently dominate applications in dairy cattle and sheep, whereas stationary systems are more widely used in pig and poultry production. Future development will depend on integrating heterogeneous data streams with automated environmental control and intelligent decision-support platforms capable of transforming continuous monitoring into adaptive herd management [215].

This digital transformation is also reshaping livestock breeding through the convergence of PLF, digital phenotyping and genomics. Continuous collection of high-dimensional phenotypic data provides a far more comprehensive characterisation of adaptive capacity than conventional production records, creating new opportunities for genomic prediction of complex resilience traits. Large-scale genetic studies have shown that convolutional neural networks (CNNs) increased the estimated heritability of digital limb conformation traits from 0.21 to 0.60 for forelimbs and from 0.18 to 0.45 for hindlimbs while maintaining strong genetic correlations with conventional assessments ( $>0.90$ ). Similarly, combining automated behavioural monitoring with ResNet50 architectures and ridge regression predicted live body weight with high accuracy ( $r = 0.89$ ). Although these studies were performed in pigs, they highlight the broader potential of digital phenotyping to improve the efficiency, objectivity and scalability of genomic selection across livestock species [216].

Routine use of high-frequency phenotypes also requires computational tools capable of processing datasets at the scale of national breeding programmes. An approximation algorithm implemented within random-regression single-step GBLUP successfully analysed approximately 30 million test-day records, 2.5 million animals and 54,000 genotyped cattle in the Czech national evaluation system, producing results that closely matched full mixed-model inversion ( $r = 0.98$ ; slope = 0.91) while completing the analysis in only 21 min [217]. Together, these developments indicate that future breeding programmes will increasingly combine continuous phenotypic monitoring with genomic information, enabling selection for functional efficiency and climate resilience while maintaining productive performance. In doing so, genomic selection is evolving from a production-oriented methodology into a comprehensive strategy for improving robustness, adaptive capacity and long-term resilience under climate change [218].

Nevertheless, genetic improvement alone cannot deliver climate-resilient dairy production. Sustainable adaptation instead requires coordinated breeding, precision livestock farming, nutritional management, engineering solutions and environmental stewardship to maintain productivity and animal welfare while simultaneously reducing greenhouse gas emissions, improving resource-use efficiency and ensuring long-term economic viability.

Among available mitigation strategies, reducing enteric methane emissions offers the greatest opportunity for decarbonising dairy production. Existing and emerging technologies have the potential to lower methane emissions by approximately 40–50%, while optimised manure storage and processing may provide an additional 5–10% reduction. Nevertheless, widespread implementation remains constrained by economic feasibility, emphasising the need to integrate methane mitigation with genetic improvement, precision nutrition, nitrogen management and whole-herd management rather than relying on individual technologies alone [219].

Environmental sustainability should likewise be assessed using a broader range of indicators than greenhouse gas emissions alone. Although pasture-based systems generally produce lower milk yields, they often achieve reduced overall environmental impacts, more efficient utilisation of locally available feed resources and a more favourable net human-edible protein balance. By contrast, intensive production systems typically exhibit a lower global warming potential per kilogram of fat- and protein-corrected milk (FPCM). Together, these observations demonstrate that meaningful sustainability assessments must balance carbon efficiency with land use, feed-use efficiency, ecosystem services and the broader socio-economic contributions of dairy farming [220].

Ultimately, the convergence of molecular biology, digital agriculture and precision management is transforming HS research from a discipline focused primarily on describing biological responses into one capable of predicting, preventing and mitigating their consequences. By integrating genomics, multiomics, digital phenotyping, AI and sustainable herd management within a unified decision-support framework, future dairy production systems will be better equipped to safeguard animal health and welfare, sustain productive performance and minimise environmental impacts under a changing climate. Realising this vision will depend on harmonised phenotyping protocols, interoperable databases, externally validated predictive models and robust economic evaluations capable of supporting evidence-based implementation across diverse production systems [221].

Taken together, these findings indicate that individual adaptation measures differ substantially in their mechanisms of action, practical applicability, resource requirements, and long-term sustainability. Rather than representing competing alternatives, they should be regarded as complementary components of integrated climate adaptation programmes. Their comparative evaluation is presented in Table 2.

**Table 2.** Comparative evaluation of adaptation strategies for sustainable dairy cattle production under heat stress.

| Adaptation Strategy                                           | Primary Objective                                                        | Expected Benefits                                                                                                                          | Key Limitations                                                                                                                          | Sustainability Implications                                                                                             | Most Appropriate Application                                                   |
|---------------------------------------------------------------|--------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------|
| Environmental cooling (fans, sprinklers, evaporative cooling) | Rapid reduction in body heat load and restoration of thermal homeostasis | Provides the fastest improvement in physiological status, feed intake, milk yield, welfare and reproductive performance during heat events | High installation and operating costs; increased water and electricity demand; reduced efficiency during prolonged or extreme heat waves | Maximises immediate resilience but increases resource consumption and environmental footprint if poorly optimised       | Intensive high-producing dairy herds exposed to frequent or severe heat stress |
| Housing design and microclimate management                    | Improve the thermal environment and reduce cumulative heat exposure      | Long-term reduction in thermal load; improves cow comfort and complements active cooling systems                                           | Infrastructure modifications may require substantial investment; effectiveness depends on climatic conditions and barn design            | Enhances animal welfare while improving production efficiency; energy demand varies according to ventilation strategy   | New or renovated dairy facilities; regions with recurring summer heat          |
| Nutritional interventions                                     | Support energy metabolism, rumen function and antioxidant defence        | Flexible implementation; partially compensates for reduced feed intake; improves metabolic stability and productive performance            | Cannot eliminate heat load; responses depend on diet formulation, physiological stage and heat intensity                                 | May improve feed-use efficiency with relatively moderate resource inputs but requires continuous nutritional management | All production systems, particularly during seasonal heat stress               |
| Genetic improvement for thermotolerance                       | Increase inherent resilience to thermal stress                           | Permanent and cumulative adaptation without continuous external inputs; benefits accumulate across generations                             | Slow genetic progress; possible antagonism with maximum production traits; requires long-term breeding programmes                        | Represents one of the most sustainable long-term adaptation strategies because resilience is genetically embedded       | National breeding programmes and long-term herd improvement strategies         |
| PLF and AI-based monitoring                                   | Early detection of heat stress and individualised management             | Continuous real-time monitoring; enables timely interventions; improves management precision and resource allocation                       | Initial investment, sensor maintenance, data integration and interpretation require technical expertise                                  | Optimises resource use while supporting animal welfare, productivity and precision decision-making                      | Medium- and large-scale farms implementing digital herd management             |

Table 2. Cont.

| Adaptation Strategy                                                                      | Primary Objective                                                                            | Expected Benefits                                                                                                                                                         | Key Limitations                                                                                             | Sustainability Implications                                                                                                                                              | Most Appropriate Application                                          |
|------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------|
| Management interventions (shade, stocking density, feeding schedule, water availability) | Reduce exposure to environmental heat and support behavioural adaptation                     | Relatively simple implementation; immediate welfare benefits; often low investment requirements                                                                           | Limited capacity to prevent severe heat stress when used alone; effectiveness depends on management quality | Cost-effective improvements in welfare and resilience with generally low environmental impact                                                                            | Extensive, pasture-based and resource-limited production systems      |
| Integrated adaptation strategy                                                           | Simultaneously optimise biological adaptation, productivity, welfare and resource efficiency | Combines complementary benefits of environmental, nutritional, genetic and digital approaches; provides the highest overall resilience under variable climatic conditions | Requires coordinated management, multidisciplinary planning and greater initial investment                  | Offers the best balance between productivity, animal welfare, economic viability and environmental sustainability, supporting climate-resilient dairy production systems | Long-term climate adaptation strategies for sustainable dairy farming |

Note: No single adaptation strategy is universally effective under all production conditions. Sustainable heat stress mitigation requires an integrated, tailored implementation of complementary environmental, nutritional, genetic, management, and precision livestock farming approaches.

### 3.12. Future Research Perspectives

Despite substantial progress in understanding the biological mechanisms underlying HS, research is increasingly shifting from describing individual physiological responses towards elucidating the integrated mechanisms that determine thermotolerance and resilience. One of the major scientific challenges is to integrate knowledge generated at the molecular, cellular, physiological, behavioural and production levels into comprehensive models capable of explaining individual variation in responses to thermal load and predicting adaptive capacity across different livestock species and production systems.

A major unresolved question is how to translate the rapidly expanding volume of biological and production data into predictive tools for practical herd management. Although PLF technologies, continuous monitoring systems and AI algorithms have advanced considerably, it remains unclear which combinations of physiological, behavioural, environmental and production indicators provide the earliest and most reliable prediction of HS development. Future studies should therefore prioritise externally validated decision-support systems that transform continuous monitoring into practical, evidence-based management strategies applicable under commercial farming conditions.

Another important priority is the integration of genomics, transcriptomics, proteomics, metabolomics and other omics technologies with digital phenotyping. Although numerous molecular pathways associated with thermal adaptation have already been identified, it remains uncertain which biomarkers are sufficiently robust across breeds, climatic regions and production systems to support reliable assessment of thermotolerance and resilience. Equally important is the integration of molecular information with digital phenotypes and production records to improve breeding programmes capable of enhancing thermal resilience without compromising productive performance.

Further research is also required to clarify the long-term biological consequences of developmental programming. Increasing evidence indicates that HS during critical prenatal and early postnatal periods may influence health, productivity, reproductive performance and adaptive capacity throughout the lifetime of the offspring, yet the underlying mechanisms and opportunities for targeted intervention remain poorly understood. Future studies should likewise focus on developing integrated resilience indices that combine physiological, productive, reproductive, behavioural and clinical traits, providing a more comprehensive assessment of adaptive capacity than can be achieved using individual biomarkers or production traits alone.

Beyond biological research, greater emphasis should be placed on evaluating the long-term economic and environmental performance of adaptation strategies. Most current studies focus primarily on short-term physiological or productive responses to cooling systems, nutritional interventions or digital monitoring, whereas their economic return, water and energy requirements, carbon footprint and scalability across diverse production systems remain insufficiently characterised. Comparative life-cycle assessments integrating biological effectiveness with economic feasibility, environmental trade-offs and practical applicability will therefore be essential for identifying adaptation strategies that remain sustainable under future climatic conditions.

Ultimately, future progress in HS research will depend less on the emergence of individual technologies than on their integration into multidisciplinary frameworks capable of linking climatic, biological, technological, economic and environmental information. Such an integrated systems-based approach provides the foundation for more accurate prediction, prevention and management of HS while supporting the long-term resilience and sustainability of livestock production.

## 4. Conclusions

Climate change is redefining HS from a predominantly seasonal production constraint into one of the principal challenges facing sustainable livestock production. Rather than affecting isolated physiological functions, HS disrupts interconnected thermoregulatory, endocrine, metabolic, oxidative, immune and reproductive processes, resulting in cascading effects on animal health, welfare, productivity and production efficiency.

The evidence synthesised in this review demonstrates that successful adaptation cannot rely on individual mitigation measures alone. Instead, improving resilience will require coordinated nutritional, environmental, technological, management and genetic approaches that address the complex biological and production-related consequences of thermal stress while maintaining resource-use efficiency and economic viability.

Overall, HS should be recognised not only as a biological challenge but also as an economic, environmental and societal issue. Strengthening the resilience of livestock production systems will be essential for safeguarding animal welfare, sustaining efficient food production and supporting the long-term sustainability of animal agriculture under a changing climate. Although this narrative review integrates current evidence from multiple disciplines, differences in study design, livestock species, climatic conditions and production systems should be considered when interpreting the available evidence and translating research findings into practical recommendations.

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