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Impact of heat stress on bull fertility: A comprehensive physiological and managerial review

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Abstract

Global climate change represents a formidable challenge to the sustainability of the livestock sector. With global surface temperatures rising by 1.1 °C in the last decade and projected to increase further, the reproductive efficiency of breeding bulls is under threat. This review elucidates the physiological maladaptations caused by heat stress, specifically the failure of testicular thermoregulation and the subsequent endocrine and molecular disruptions. It details the biphasic biological response to stress, the cascade of oxidative damage and apoptosis in germ cells, and the "lag effect" where thermal insults manifest in ejaculate quality weeks post-exposure. Furthermore, it evaluates evidence-based mitigation strategies, ranging from environmental modifications like multiple showering to genetic selection for thermos-tolerance, to sustain semen production in a warming world.

Keywords: Global climate change, warming world, bull fertility, comprehensive physiological and manage mental review

1. Introduction

The global climate has undergone significant shifts in recent decades, creating a hostile environment for agriculture and animal husbandry. According to the Intergovernmental Panel on Climate Change (IPCC, 2023) ^[14], global surface temperatures increased by 1.1 °C between 2011 and 2020 compared to historical averages from 1850-1900. Projections indicate a further increase of up to 1.5 °C between 2030 and 2052 due to anthropogenic activities and greenhouse gas emissions.

India, a nation heavily reliant on its dairy sector, is already experiencing these rising temperatures. Gupta *et al.* (2024) ^[10] define heat stress as the cumulative result of internal and external forces acting on an animal that raises body temperature and elicits a physiological response. While all livestock are affected, ruminants are uniquely vulnerable. Cheng *et al.* (2016) ^[6] highlight that ruminants sweat at only 10% of the rate of humans, rendering them inefficient at dissipating excess body heat via evaporation.

For breeding bulls, this inefficiency is catastrophic. As noted by Hansen (2009) ^[11], when environmental heat overwhelms the animal's thermoregulatory capacity, the resulting hyperthermia disrupts the delicate microclimate required for spermatogenesis. Given the National Dairy Development Board's (NDDB, 2024) data showing over 4,600 bulls under collection producing 135 million frozen semen doses annually, any decline in fertility due to heat stress represents a massive economic loss for the country.

2. Physiological Responses to Heat Stress

2.1 The Thermoneutral Zone (TNZ) and Biological Rhythms

Livestock operate optimally within a Thermoneutral Zone (TNZ), a range of ambient temperatures where the animal maintains core body temperature without expending additional energy for thermoregulation. Capela *et al.* (2022) ^[4] and Krishnan *et al.* (2017) ^[16] identify the TNZ for Zebu cattle (*Bos indicus*) as ranging between 10 °C and 36 °C. For exotic and crossbred cattle, the Upper Critical Temperature (UCT) is significantly lower, between 24 °C and 26 °C. Buffaloes generally face thermal stress when temperatures exceed 33 °C.

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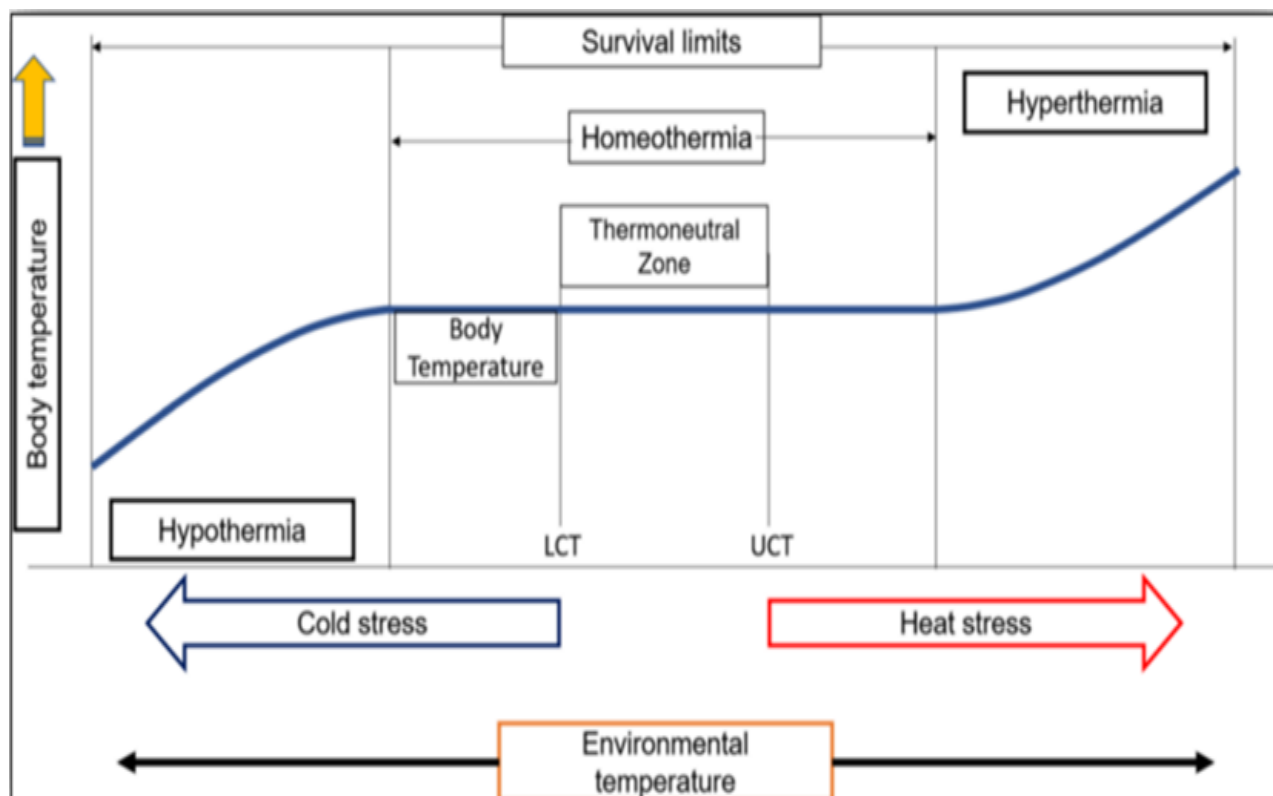


Fig 1: Homeothermy chart (LCT, lower critical temperature; UCT, upper critical temperature)

A graphical representation (Figure 1) showing the relationship between environmental temperature and body temperature. It illustrates the zone of Homeothermy (between Lower Critical Temperature and Upper Critical Temperature) and the zones of Hypothermia and Hyperthermia outside these limits.

2.2 The Temperature-Humidity Index (THI)

To accurately assess the thermal load, Hansen (2009) ^[11] advocates for the Temperature-Humidity Index (THI), which combines air temperature and relative humidity. Celeghini *et al.* (2024) ^[5] and Kendall *et al.* (2008) ^[15] classify stress levels as follows:

- **THI < 74:** Normal / Thermal Comfort.
- **THI 74-79:** Alert Status.
- **THI 79-84:** Danger Level
- **THI ≥ 84:** Emergency Status. Research indicates that bull fertility begins to decline significantly once the THI exceeds 72.

2.3 Biphasic Biological Response

Collier *et al.* (2017) describe the biological response to heat stress as biphasic:

- **Acute Phase:** Activated by the nervous system (hypothalamus) and lasts minutes to days. It involves immediate reactions such as vasodilation, increased respiration (> 80 breaths/min), panting, and behavioral changes like shade-seeking and increased water intake (up to 30% increase).
- **Chronic Phase:** Driven by the endocrine system (homeorhesis) and lasts days to weeks. This involves long-term hormonal reprogramming to ensure survival, often sacrificing reproductive function. This state of "acclimation" is a reversible phenotypic response.

3. Mechanisms of Testicular Thermoregulation

The testes require a strictly regulated microclimate to function. Rizzoto *et al.* (2020) ^[22] emphasize that for physiological spermatogenesis and steroidogenesis, mammalian testes must be maintained at 2 °C to 6 °C below core body temperature.

3.1 Anatomical Adaptations

Barros *et al.* (2015) ^[2] and Morrell (2020) ^[18] detail the anatomical structures responsible for this cooling:

- **Pampiniform Plexus:** A vascular network acting as a countercurrent heat exchanger. It cools arterial blood entering the testes by transferring heat to the cooler venous blood leaving the testes.
- **Tunica Dartos Muscle:** This smooth muscle relaxes in warm conditions to lower the testes away from the body warmth and contracts in cold conditions to pull them closer.
- **Cremaster Muscle:** Assists in raising and lowering the testes.
- **Scrotal Skin:** Thin, relatively hairless, and devoid of subcutaneous fat to maximize evaporative cooling via sweat glands.

3.2 The Temperature Gradient

A healthy bull exhibits a specific thermal topography. Kastelic *et al.* (1995) ^[14] established that there is a temperature gradient of 1.6 °C from the top (warmer) to the bottom (Cooler) of the scrotal surface. This gradient is essential for maintaining uniform intratesticular temperatures (-0.2 °C gradient). During heat stress, this gradient is disrupted, leading to testicular hyperthermia

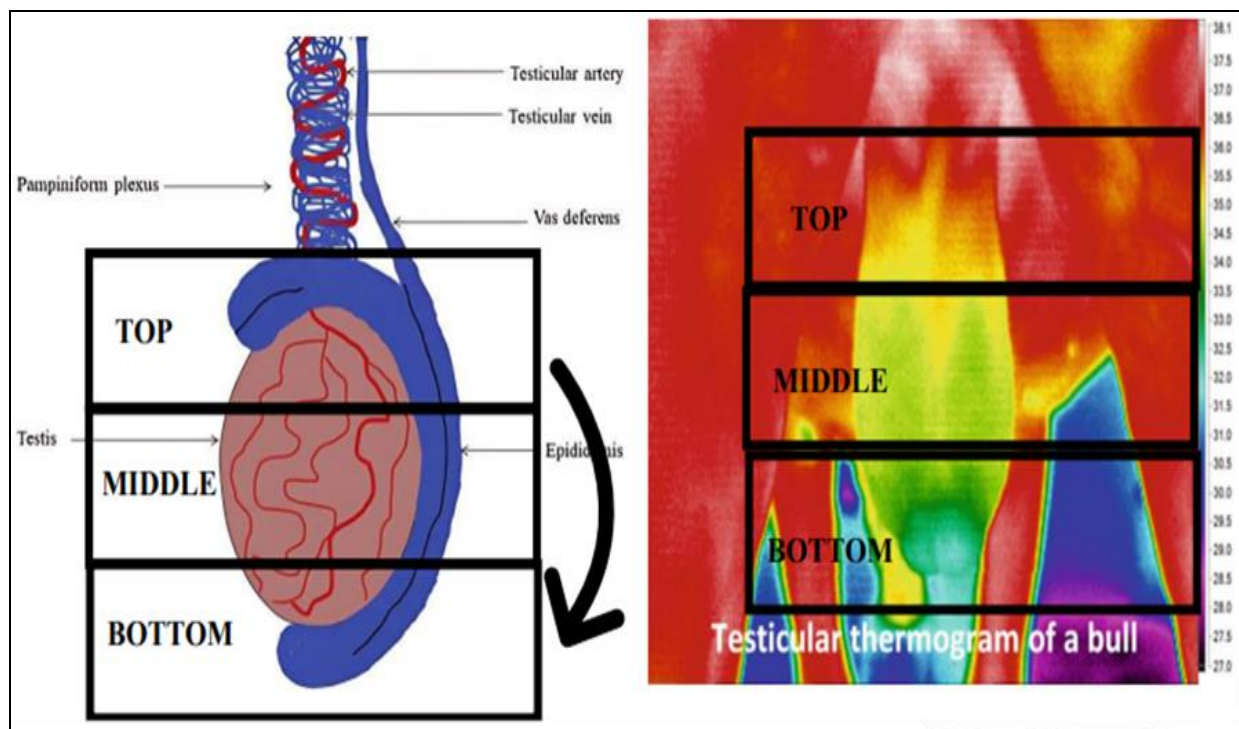


Fig 2: Representation of temperature gradient of testes and testicular thermogram

A visual representation (Figure 2) showing the temperature gradient. The top of the scrotum is warmer due to proximity to the body, while the bottom is cooler. Infrared thermography is used to visualize this gradient.

4. Pathophysiology: Endocrine and molecular mechanisms

The damage caused by heat stress operates on multiple levels: endocrine suppression, oxidative stress, and apoptosis (cell death).

4.1 Endocrine Disruption

The hypothalamus-pituitary-gonadal axis is highly sensitive to thermal stress. Rizzoto *et al.* (2020) [22] and Takahashi (2012) report that:

- **LH Suppression:** Heat stress suppresses the secretion of Luteinizing Hormone (LH) from the pituitary gland.
- **Testosterone Decline:** Reduced LH stimulation and the downregulation of steroidogenic enzymes (StAR, CYP17) in Leydig cells lead to significantly reduced testosterone concentrations.
- **Cortisol Spike:** Simultaneously, the adrenal cortex releases cortisol, a stress hormone that further antagonizes reproductive function, leading to decreased libido and spermatogenesis.

4.2 Oxidative Stress (ROS Pathway): Durairajanayagam *et al.* (2015) [9] describe the cellular damage caused by the overproduction of Reactive Oxygen Species (ROS) such as hydrogen peroxide (H_2O_2).

- **Lipid Peroxidation:** Sperm membranes are rich in polyunsaturated fatty acids. ROS attack these lipids, compromising membrane integrity.
- **DNA Damage:** Heat reduces the availability of DNA repair enzymes (DNA ligase III, PARP). Consequently,

DNA fragmentation increases, posing a risk of subfertility even if fertilization occurs.

4.3 Molecular pathways of apoptosis

Heat stress triggers apoptosis in germ cells via two primary pathways, as detailed by Durairajanayagam *et al.* (2015) [9]:

- **Intrinsic Pathway (Mitochondrial):** Triggered by internal oxidative stress. Heat stress causes the translocation of the pro-apoptotic protein Bax to the mitochondria, leading to the release of Cytochrome C. This activates Caspase-9 and subsequent executioner Caspases (3, 6, 7), resulting in cell death.
- **Extrinsic Pathway (Death Receptor):** Triggered by external signals. Stress activates Fas/FasL death receptors on the cell surface. This recruits FADD, which activates Caspase-8/10, eventually leading to the same executioner caspases and apoptosis.

Additionally, the protein p53 acts as a transcription factor that upregulates pro-apoptotic genes during thermal stress.

5. Impact on sperm quality: The lag effect

A critical concept in bull fertility is the "lag effect." Because spermatogenesis takes approximately 60 days, the sperm ejaculated today reflects the environmental conditions from two months prior.

5.1 Thermotolerance of Germ Cells

Cheng and Mruk (2012) and Setchell (1998) note that not all cells are equally vulnerable. They observed that spermatocytes (meiosis) and spermatids (differentiation) cells are most sensitive. Whereas, mature sperm in the cauda epididymis and undifferentiated spermatogonia cells are least sensitive to the heat.

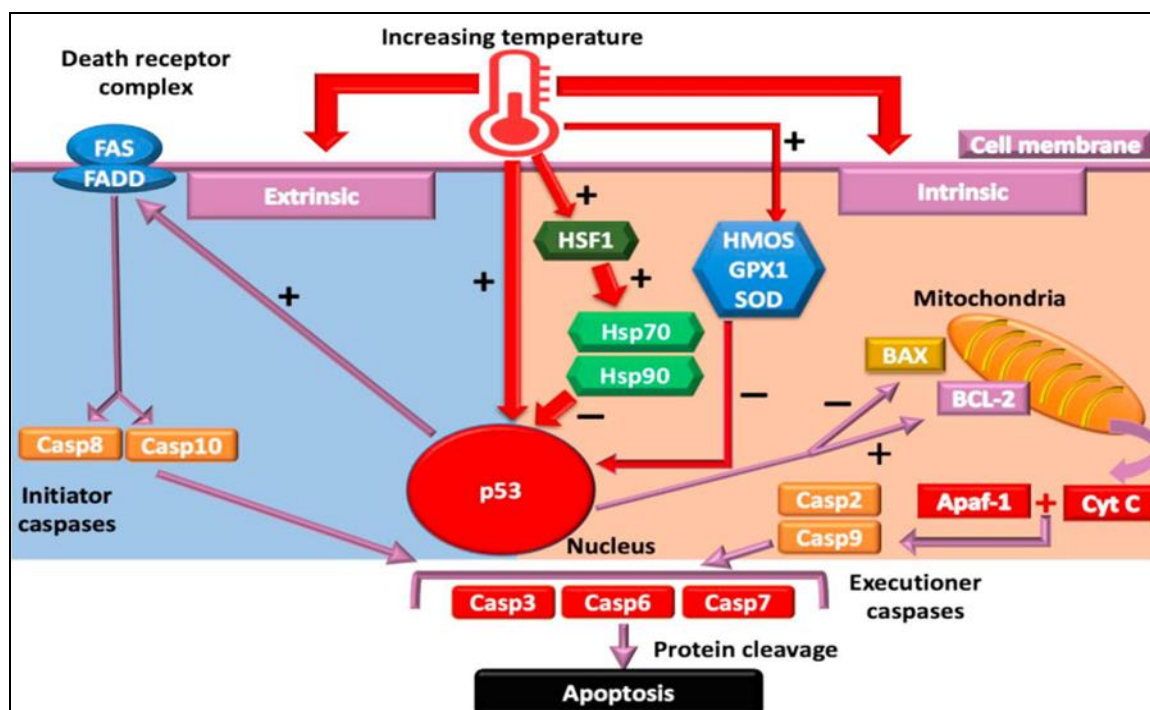


Fig 3: Molecular pathways of apoptosis (Durairajanayagam *et al.*, 2015) [9]

5.2 Chronology of Semen Defects

Celeghini *et al.* (2024) [5] outline the specific timeline of defects appearing in the ejaculate following a heat stress event:

- **Phase 1 (< 7 Days):** Represents damage to epididymal sperm. Clinical findings include decreased acrosomal integrity and minor defects like detached normal heads.
- **Phase 2 (14-42 Days):** The period of peak damage. This corresponds to cells that were undergoing meiosis during the heat event. Ejaculates show severe decreases in motility, high DNA fragmentation, lipid peroxidation,

and major defects like proximal droplets and coiled tails.

- **Phase 3 (49-63 Days):** Affects early spermatogenesis. Continued morphological abnormalities and mitochondrial damage.
- **Phase 4 (> 70 Days):** Recovery phase. Semen characteristics begin to return to normal as new sperm are produced from resistant stem cells.

A timeline (Figure 4 in source) showing the four phases of sperm quality changes post-heat stress, highlighting Phase 2 as the critical window for poor quality

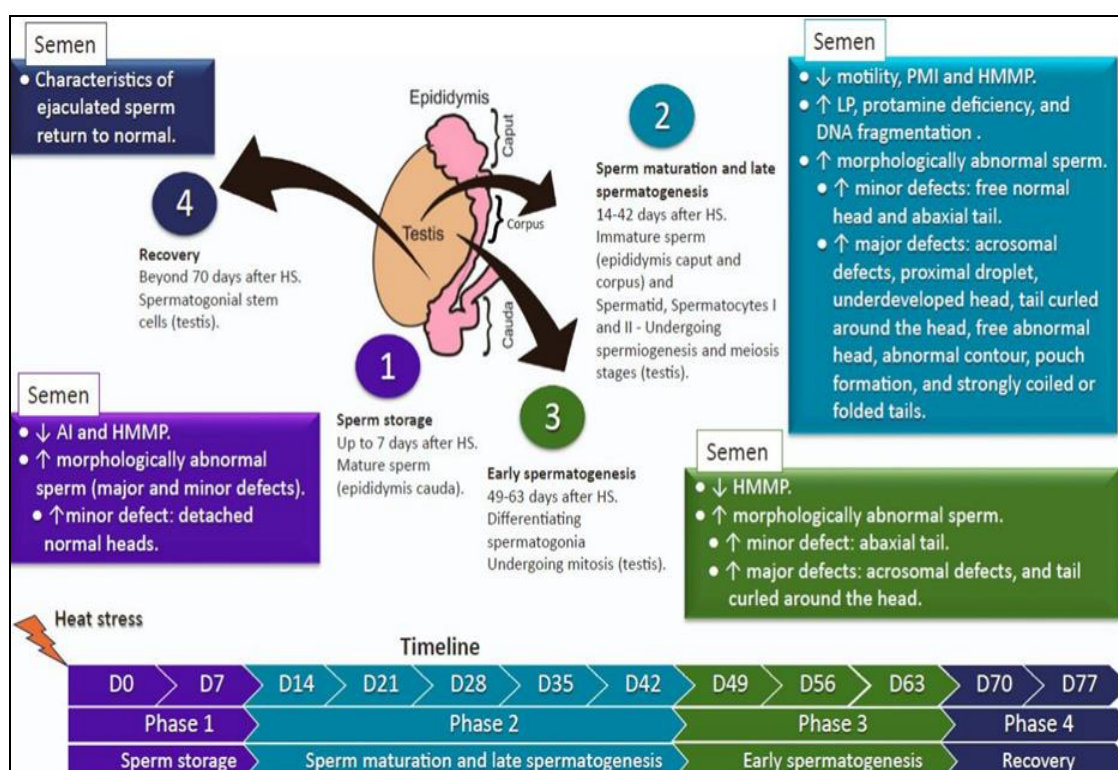


Fig 4: Sperm changes according to the presumed site/stage the ejaculated sperm were during the heat stress insult in bulls. HS: heat stress; AI: acrosome integrity; HMMP: high mitochondrial membrane potential; PMI: plasma membrane integrity; LP: lipid peroxidation (Celeghini *et al.*, 2024) [5]

5.3 Empirical Evidence from Indian Studies

Several studies cited in the seminar validate these mechanisms in indigenous breeds:

- **Parmar *et al.* (2020)** ^[20]: Studied in Jaffarabadi buffalo bulls and significant seasonal differences were observed.
- **Abnormal Sperm**: Increased from 5.00% in winter to 7.12% in summer ($p < 0.05$).
- **Initial Motility**: Decreased from 80.31% in winter to 78.75% in summer.
- **Live Sperm**: Decreased from 86.20% in winter to 82.31% in summer.
- **Dhami *et al.* (2021)** ^[8]: Evaluated oxidative markers in Gir, Murrah, and Surti bulls. While some parameters varied, the study confirmed the presence of heat stress (THI > 72 for 72.6% of the day) and its association with oxidative stress markers like MDA.
- **Parmar *et al.* (2021)** ^[21]: Investigated Heat Shock Proteins. HSP70 levels in post-thaw semen were significantly higher in summer (345.90 ng/mg) compared to Winter (280.90 ng/mg), indicating a cellular stress response to protect proteins from denaturation.

6. Mitigation Strategies

To sustain fertility, a multi-faceted management approach is required.

6.1 Environmental Management

The goal is to reduce the thermal load. Strategies include fans, foggers, and wallowing tanks. A pivotal study by Hoque *et al.* (2018) ^[12] on buffalo bulls demonstrated the efficacy of increasing showering frequency. Where they did comparison of single shower (10:00 AM) vs. four showers (07:00, 10:00, 13:00, 16:00). The multiple-shower group showed significantly higher Ejaculate Volume (2.53 mL vs 2.04 mL) and Live Sperm (90.28% vs 81.38%). Proximal droplets decreased significantly in the treated group.

6.2 Nutritional Interventions

Dietary adjustments can bolster the animal's defense systems. Krishnan *et al.* (2017) ^[16] and Shahat *et al.* (2023) ^[23] recommend supplementing Vitamin E, Selenium, and Zinc as antioxidants. These scavenge ROS, protecting sperm membrane integrity and reducing DNA fragmentation. Because Dry Matter Intake (DMI) drops by 10-30% during heat stress, feeding high-quality, low-fiber, and high-protein diets helps maintain energy balance without generating excessive metabolic heat.

6.3 Medicinal Support

Megahed and Hassan (2023) ^[17] found that injections of Gonadotropin-Releasing Hormone (GnRH) can stimulate the release of LH and testosterone, mitigating hormonal suppression. Melatonin is known for its antioxidant properties, melatonin administration reduces testicular inflammation and oxidative damage.

6.4 Genetic Management

Osei-Amponsah *et al.* (2019) ^[19] advocate for long-term genetic solutions with the help of genetic modifications. Thermotolerance Genes: Selection for the "slick" gene (short hair) and sweating competence. Breed Selection: Utilizing *Bos indicus* breeds, which naturally possess lower metabolic rates and superior heat dissipation compared to exotic breeds.

7. Conclusion and Future Prospects

Heat stress disrupts the reproductive axis at hormonal, cellular, and genetic levels. The failure of testicular thermoregulation triggers a cascade of oxidative damage and apoptosis, the effects of which are often delayed due to the spermatogenic cycle.

As concluded in the seminar, sustaining bull fertility requires an integrated approach. Relying solely on one method is insufficient. The future lies in precision livestock technologies-using real-time monitoring to detect early signs of stress and combining environmental modifications (like multiple showering) with nutritional support and genetic selection for thermotolerance. This holistic strategy is vital for the economic sustainability of the livestock production systems in the face of global warming.

Conflict of Interest

Not available

Financial Support

Not available

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