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REVIEW ARTICLE

The Effects of Heat Stress on Reproduction in Cattle

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Bülent BÜLBÜL
DVM, Ph.D.
Dokuz Eylül University Faculty of Veterinary Medicine

https://orcid.org/0000-0002-7130-8796

Corresponding author: bulent.bulbul@deu.edu.tr

Abstract

Along with global warming, the increasing heat stress is one of the most significant environmental factors seriously threatening animal welfare, production/yield, and reproductive performance in dairy and beef cattle farming. This stress condition, which becomes evident when the Temperature-Humidity Index (THI) exceeds the 68–72 threshold, disrupts the hypothalamic-pituitary-gonadal axis, thereby suppressing the release of critical reproductive hormones such as luteinizing hormone (LH) and estradiol in cows. These hormonal imbalances lead to weakened estrus behavior (subestrus), impaired follicle development, and reduced oocyte quality due to increased oxidative stress at the cellular level. Deteriorated oocyte quality and an adversely affected uterine environment increase early embryonic deaths, resulting in a sharp decline in pregnancy rates during the summer months. Heat stress negatively affects not only females but also bulls; increased testicular temperature due to impaired scrotal thermoregulation disrupts the spermatogenesis process. While sperm volume and motility decrease in bulls, sperm DNA damage caused by the accumulation of reactive oxygen species (ROS) reduces fertilization capacity. Consequently, to minimize these negative effects of heat stress on reproduction, the use of environmental cooling systems such as shade and fans in housing, the addition of antioxidants like selenium to rations, and the utilization of supportive reproductive biotechnologies such as timed artificial insemination and embryo transfer are of great importance for sustainability.

Introduction

Heat stress is a condition that arises when increases in ambient temperature and humidity exceed an animal's ability to dissipate heat, thereby disrupting physiological balance (5). The thermoneutral zone, where optimal welfare and thermal balance are maintained for dairy cows, is generally considered to be within the range of 0 to 20°C (7). Along with global warming and climate change, the increasingly extreme temperatures during summer months pose one of the greatest threats to the profitability and sustainability of dairy cattle farming (6). The most widely used and reliable measure for determining the onset and severity of heat stress in cattle is the Temperature-Humidity Index (THI) (Figure 1) (2). The heat stress threshold in cattle begins when the THI value exceeds the 68-72 range, and a significant decline in pregnancy rates is observed in cows after this level (14). Feed intake in dairy cows decreases slightly at temperatures of 25-26°C but drops sharply above 30°C, and decreases by as much as 40% when the ambient temperature reaches 40°C. This decrease leads to ketosis, negative energy balance, and a series of subsequent adverse effects (15). Heat stress not only reduces milk production and feed intake in animals but also severely disrupts the reproductive physiology of both female and male cattle through the cellular damage it causes (8). Due to rising ambient temperatures, a significant decrease of 20-30% in the conception rates of cows is observed worldwide during the summer months (10). The effects of heat stress on reproduction in cattle are summarized in Figure 2.

The purpose of this review is to evaluate the effects of heat stress on reproductive physiology in cows and bulls from hormonal, cellular, and practical management perspectives, and to summarize current mitigation strategies.

Ambient Temperature (°C)	Relative Humidity (%)									
	10	20	30	40	50	60	70	80	90	100
22	65	66	66	67	68	69	69	70	71	72
24	67	68	69	70	70	71	72	73	74	75
26	69	70	71	72	73	74	75	77	78	79
28	71	72	73	74	76	77	78	80	81	82
30	72	74	75	77	78	80	81	83	84	86
32	74	76	77	79	81	83	84	86	88	90
34	76	78	80	82	84	85	87	89	91	93
36	78	80	82	84	86	88	90	93	95	97
38	79	82	84	86	89	91	93	96	98	100
40	81	84	86	89	91	94	96	99	101	104
42	83	86	89	92	94	97	100	103	105	108
44	85	88	91	94	97	100	102	105	108	111
46	87	90	93	96	99	102	105	109	112	115
48	88	92	95	98	102	105	108	112	115	118
50	90	94	97	101	104	108	111	115	118	122

THI	Stress Level
≤71	Light
72-79	Moderate
80-89	Severe
90-99	Very severe
≥100	Deadly

Figure 1: Temperature-Humidity Index (THI): (32+1.8x°C)-[0.55-0.55(Relative humidity/100)]x[(32+1.8x°C)-58] (2).

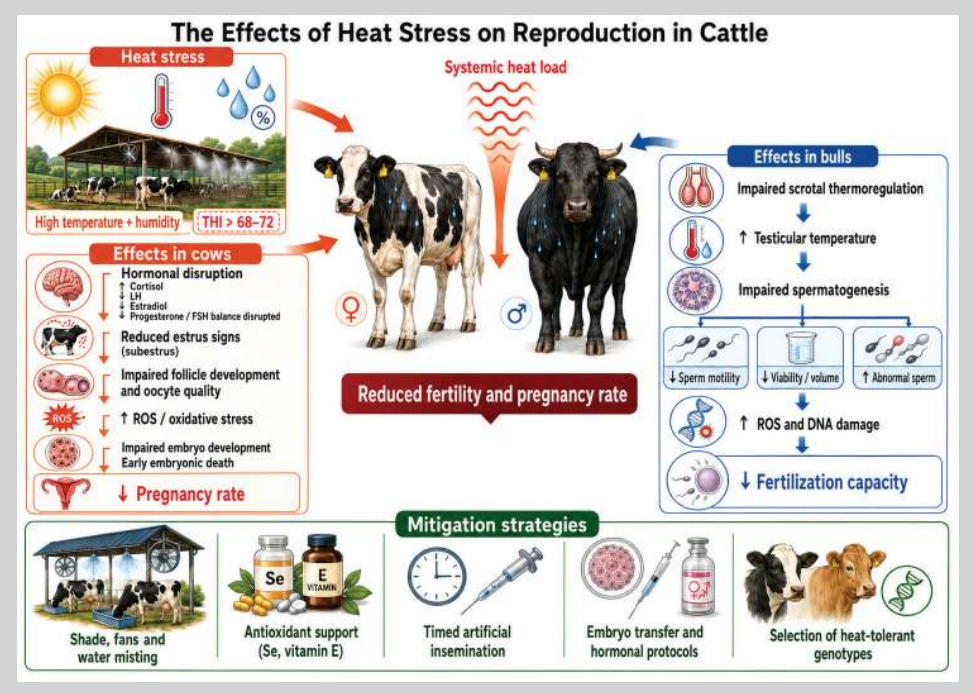

Figure 2: The effects of heat stress on reproduction in cattle (Conceptual diagram was generated using artificial intelligence from the authors' scientific outline and reviewed for scientific accuracy).

The EFFECTS of HEAT STRESS on REPRODUCTION in COWS

Hormonal Changes and Neuroendocrine Disorders

Heat stress disrupts the hypothalamic-pituitary-gonadal axis in cattle, significantly impairing the normal release dynamics of hormones essential for reproduction (11). In these animals, the adrenal glands become activated, and levels of cortisol—the stress hormone in the bloodstream—increase significantly (12). The high levels of cortisol accumulating in the blood suppress the preovulatory release of luteinizing hormone (LH), which directly inhibits the follicle's maturation and ovulation process (18). Conversely, while LH secretion is suppressed, an increase in follicle-stimulating hormone (FSH) levels or the loss of balance between these two hormones prevents the healthy development of the dominant follicle in the ovaries (11). At the same time, the gene expression of specific proteins responsible for hormone production (steroidogenesis) in granulosa cells—such as StAR, LHR, and aromatase—also decreases due to heat stress. As a result of these enzymatic disruptions, the estradiol concentration in the follicular fluid rapidly decreases, and the estrus cycle is disrupted (12). Furthermore, due to the underdevelopment of the corpus luteum caused by heat stress, progesterone levels decrease, putting the maintenance of a potential pregnancy at risk (10).

The Estrus Cycle and Behavior

Disruptions in neuroendocrine mechanisms and hormonal deficiencies shorten the estrus duration and reduce its intensity in cows (5). Due to low blood estradiol levels, cows do not exhibit normal estrus behavior, which increases the rate of subestrus in herds (10). Particularly during hot summer months, cows' reduced activity—such as standing, displaying estrus signs, and eating—makes it difficult to accurately detect estrus, which is essential for successful artificial insemination (13). In their study analyzing 9,972 estrus records, Bülbül and Ataman (2) found that the incidence of estrus in cows decreased when the THI exceeded 72. When estrus detection becomes difficult or insemination is performed at the wrong time, pregnancy and fertility rates during the summer inevitably decline (11).

Ovarian Function, Follicle Development, and Oocyte Quality

One of the most damaging effects of heat stress on the ovarian microenvironment is severe oxidative stress, characterized by the intracellular accumulation of reactive oxygen species (ROS) (16). Although the production of antioxidant enzymes such as SOD1 and SOD2 increases in granulosa cells as a defense mechanism against rising ROS accumulation, this proves insufficient to protect the cells from apoptosis (programmed cell death). Oxidative stress triggers cell death in granulosa cells of the ovary by increasing the levels of apoptosis markers such as caspase-3 (12). In this adverse environment, the cytoskeletal integrity of developing oocytes is disrupted, and nuclear maturation processes fail (16). Levels of oocyte-derived growth factors such as GDF9 and BMP15, which are critical for oocyte development, also decrease significantly as a result of heat stress

(12). The transcription and translation of maternal mRNAs stored within the oocyte are also impaired by heat stress, thereby limiting the capacity for early embryonic development following fertilization (16). Additionally, a significant increase in the expression levels of Heat Shock Proteins (particularly HSP70) is observed as a cellular response of oocytes to environmental stress factors (12).

Embryonic Development and Pregnancy Losses

Embryos derived from oocytes twhose quality has deteriorated under heat stress are significantly less capable of implanting in the uterus, continuing their development, and surviving (10). In particular, embryos in the early preimplantation stage prior to zygotic genome activation (e.g., the 2-8-cell stages) are extremely sensitive to increased temperature and oxidative stress (16). Heat stress accelerates embryonic cell death (apoptosis) by causing DNA damage and functional impairments in these developing early-stage embryos (10). Because both low progesterone levels and genetic and epigenetic damage in the embryo make it difficult for the embryo to implant in the uterus, early embryonic deaths increase during the summer months, leading to significant pregnancy losses (6). Cavestany et al. (4) reported that pregnancy rates, which range from 40-60% during the cooler season, drop to 10-20% during the hot season.

The EFFECTS of HEAT STRESS on REPRODUCTION in BULLS

Heat stress has extremely detrimental effects on reproductive potential and fertility not only in female but also in male cattle (3). For healthy and high-quality spermatogenesis to occur in bulls, testicular temperature must be approximately 2-8°C lower than body temperature (17). High ambient temperatures bypass the bulls' scrotal thermoregulatory systems, leading to an increase in testicular temperature (10). Increased testicular temperature causes significant declines in sperm motility and overall viability, while also increasing the proportion of morphologically abnormal and damaged spermatozoa. ROS accumulated in spermatozoa due to heat stress leads to oxidative stress and lipid peroxidation in spermatozoon cell membranes (3). This oxidative damage reduces fertilization capacity by causing breaks in sperm DNA and increases cellular apoptosis (10). Bhakat et al. (1) reported that sperm quality (motility, concentration, and percentage of abnormal spermatozoa) in bulls is significantly higher during the winter months than during the hot summer months. This severe decline in sperm quality not only leads to fertilization failures during insemination but also negatively affects the viability and quality of the developing embryo even if fertilization occurs (10).

STRATEGIES to MITIGATE the EFFECTS of HEAT STRESS

To prevent these serious physiological impairments and economic losses, it is recommended to implement environmental mitigation strategies in animal housing, such as shade structures, fans, and water misting (evaporative cooling) systems (9). Additionally, adding antioxidants such as selenium or vitamin E, as well as cell-protective dietary supplements, to rations can help neutralize oxidative damage caused by heat stress (20). To minimize

reproductive losses, it is crucial to utilize fixed-time artificial insemination during hot summer days, the transfer of in vitro-produced embryos that are more resistant to the period when heat stress is most severe, and hormonal protocols (e.g., GnRH or progesterone treatments) (5). As a long-term and more sustainable solution, it is necessary to develop cattle genotypes capable of naturally tolerating high ambient temperatures through genomic selection programs utilizing multi-omics technologies (19).

Conclusion

Heat stress poses one of the greatest threats to reproductive performance by severely disrupting cellular functions and hormonal balance in cattle, thereby impairing oocyte quality and embryonic development in cows and sperm quality in bulls (8). Heat stress poses one of the greatest threats to reproductive performance by severely disrupting cellular functions and hormonal balance in cattle, thereby impairing oocyte quality and embryonic development in cows and sperm quality in bulls. Identifying this problem and taking measures to address it are of vital importance for profitability in cattle farming.

References

1. Bhakat M, Mohanty TK, Gupta AK, Abdullah M. Effect of season on semen quality of crossbred (Karan Fries) bulls. Adv. Anim. Vet. Sci. 2014; 2 (11), 632-637.
2. Bülbül B, Ataman MB. The effect of some seasonal conditions on oestrus occurrence in cows. Arch. Anim. Breed. 2009; 52 (5), 459-465.
3. Capela L, Leites I, Romão, R, Lopes-da-Costa L, Pereira RMLN. Impact of heat stress on bovine sperm quality and competence. Animals 2022; 12 (8), 975.
4. Cavestany D, El-Whishy AB, Foote RH. Effect of season and high environmental temperature on fertility of Holstein cattle. J. Dairy Sci. 1985; 68, 1471-1478.
5. Dash S, Chakravarty AK, Singh A, Upadhyay A, Singh M, Yousuf S. Effect of heat stress on reproductive performances of dairy cattle and buffaloes: A review. Veterinary World 2016; 9 (3), 235-244.
6. Dovolou E, Giannoulis T, Nanas I, Amiridis GS. Heat stress: A serious disruptor of the reproductive physiology of dairy cows. Animals 2023; 13 (11), 1846.
7. Felini R, Cavallini D, Buonaiuto G, Bordin T. Assessing the impact of thermoregulatory mineral supplementation on thermal comfort in lactating Holstein cows. Vet. Anim. Sci. 2024; 24, 100363.
8. Gupta M, Vaidya M, Kumar S, Singh G, Osei-Amponsah R, Chauhan SS. Heat stress: A major threat to ruminant reproduction and mitigating strategies. Int. J Biometeorol. 2025; 69, 209-224.
9. Jongbo AO, de Borba LP, Pereira RMM, Bello QO, Gregoratto LL, de Souza DP, Adeyeye OA, Vieira FMC. Heat stress in livestock under tropical climates: Impacts and mitigation strategies. Trop. Anim. Health Prod. 2026; 58, 65.
10. Khan I, Mesalam A, Heo YS, Lee SH, Nabi G, Kong IK. Heat stress as a barrier to successful reproduction and potential alleviation strategies in cattle. Animals 2023; 13(14), 2359.
11. Khodaei-Motlagh M, Zare Shahneh A, Masoumi R, Derensis F. Alterations in reproductive hormones during heat stress in dairy cattle. Afr. J Biotech. 2011; 10, 5552-5558.
12. Kim TG, Kim SH, Kim SJ, Lee WJ. Heat stress disrupts the ovarian microenvironment in cattle: An in vivo analysis of hormonal alterations, oxidative stress-induced apoptosis, and reduced pregnancy outcomes. Anim. Reprod. Sci. 2026; 287, 108111.
13. Leliveld LMC, Lovarelli D, Riva E, Provolo G. Dairy cow behaviour and physical activity as indicators of heat stress. Italian J Anim. Sci. 2025; 24 (1), 772-783.
14. Ojo TO, Vandenplas J, Mulder HA, van Pelt ML, Calus MPL. Genetic analysis of the impact of heat stress on fertility traits in dairy cows in the Netherlands. J Dairy Sci. 2025; 108 (2), 1699-1713.
15. Rhoads RP, Baumgard LH, Suagee JK, Sanders SR. Nutritional interventions to alleviate the negative consequences of heat stress. Adv. Nutr. 2013; 4 (3), 267-276.

References

16. Roth Z. Effect of heat stress on reproduction in dairy cows: Insights into the cellular and molecular responses of the oocyte. Ann. Rev. Anim. Biosci. 2017; 5, 151-170.
17. Takahashi M. Heat stress on reproductive function and fertility in mammals. Reprod. Med. Biol. 2012; 11, 37-47.
18. Wolfenson D, Roth Z. Impact of heat stress on cow reproduction and fertility. Anim. Front. 2018; 9 (1), 32-38.
19. Xiong Z, Li L, Ouyang K, Qu M, Qiu Q. Deciphering heat stress mechanisms and developing mitigation strategies in dairy cattle: A multi-omics perspective. Agriculture 2025; 15, 1477.
20. Zheng Y, Xie T, Li S, Wang W, Wang Y, Cao Z, Yang H. Effects of selenium as a dietary source on performance, inflammation, cell damage, and reproduction of livestock induced by heat stress: A review. Front. Immunol. 2022; 12, 820853.