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Impact of heat stress on female reproduction in farm animals: challenges and possible remedies

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ABSTRACT

Global warming poses significant challenges to agriculture, particularly within the livestock industry. Heat stress in animals refers to the physiological and metabolic distress experienced when environmental heat causes an animal's body temperature to exceed its thermoneutral zone. Heat stress has a significant detrimental impact on reproductive outcomes, posing a direct threat to the profitability of livestock operations. Heat stress disrupts the normal hormonal balance essential for reproduction in female livestock, adversely affecting events of folliculogenesis and embryogenesis. As a result, the developmental competence of oocytes is compromised, which is crucial for successful fertilization and reproduction. Consequently, heat stress can lead to issues such as immature oocytes, atypical or sluggish embryonic development, silent estrus, and increased pregnancy loss causing a decline in reproductive rates and livestock owners face substantial financial losses. Despite using various cooling strategies, such as water sprinkler systems, cooling shades, and cooling devices, these measures are often insufficient to restore normal reproductive rates. Therefore, it is crucial to implement and explore novel strategies to enhance reproductive efficiency during periods of heat stress. Potential mitigation techniques include estrus synchronization, artificial insemination, hormonal alterations, embryo transfer, genetic engineering, sophisticated breeding strategies, the use of nutraceuticals, and changes in management practices. This review aims to consolidate recent findings on the adverse effects of heat stress on female reproduction, especially in tropical environments, and to evaluate potential strategies for improving summer fertility of livestock.

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Introduction

The climate is influenced by various factors such as day length, direct solar radiation, relative humidity, latitude, altitude, the presence of water reservoirs like ponds, rivers, and oceans, as well as winds and precipitation. These elements differentiate one climatic zone from another and affect the living conditions for humans, livestock, and other organisms. Livestock species maintain a body temperature of 35-39°C (Prosser et al., 1991), achieved through metabolic heat generation to stay above environmental temperatures. This balance between heat generation and heat loss is crucial for maintaining a stable body temperature. However, increasing global temperature is severely affecting reproductive efficiency in livestock. Since the early 20th century, global warming has caused an approximate 0.7°C increase in surface temperatures, with projections suggesting a rise of 1.8°C-4.0°C by 2100 (IPCC, 2014). Developing countries, where economies are heavily dependent on climate-sensitive sectors like agriculture, are particularly vulnerable to extreme climate events (Dash et

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al., 2016). High-producing farm animals, often developed in temperate regions, are specialized in converting feed into valuable products. This process generates significant internal heat. In arid and semi-arid regions, high solar radiation and humidity impair the animals' ability to dissipate this internal metabolic heat (Nardone et al., 2010; Mader et al., 2006). Consequently, these animals experience heat stress, which reduces feed intake, hampers production and reproduction, and increases susceptibility to diseases. In ruminants, reproductive functions are particularly sensitive to environmental stress (Singh et al., 2011) since extreme temperatures and high humidity interfere with reproductive tissues and organs, leading to infertility and reproductive inefficiency (Dash et al., 2016). The effects of heat stress on reproductive capabilities include disruptions in reproductive tract function, hormonal imbalances, decreased oocyte quality, and reduced embryonic development and survival (Wolfenson et al., 2002; Gendelman et al., 2012a; Gendelman et al., 2012b). In hot arid regions, compromised oocyte and embryo quality leads to lower conception rates and extended calving intervals, resulting in significant economic losses for the dairy industry (Collier et al., 2006). Moreover, heat stress also negatively affects female fertility by disrupting estrus occurrence, reducing conception rates, and impairing embryo survival (Hansen, 2009). It compromises immune function in newborns, increasing disease susceptibility as well. Furthermore, heat stress impacts various cellular functions and metabolic processes, leading to altered blood flow distribution and reduced feed intake, ultimately affecting production performance (Belhadj-Slimen et al., 2016; Johnson and Wiersma, 2009; Chen et al., 2024).

Despite advances in understanding animal responses to environmental conditions, managing livestock to mitigate climate change effects remains a significant challenge. Addressing heat stress in animals is crucial not only for improving the livelihoods of marginal farmers but also for enhancing the overall economy of the livestock industry.

This review aims to state various impacts of heat stress on the reproductive capabilities of bovine animals and summarize mitigation techniques that can enhance overall reproductive efficiency (Figure 1).

Effect of heat stress on reproduction of female

Female livestock is particularly susceptible to heat stress as it disrupts the endocrine balance necessary for maintaining normal physiology, production, and reproduction (Takahashi, 2011; Danso et al., 2024). This environmental stress impairs crucial reproductive events, including oocyte maturation, estrus behavior, early embryonic development, fetal growth, and lactation.

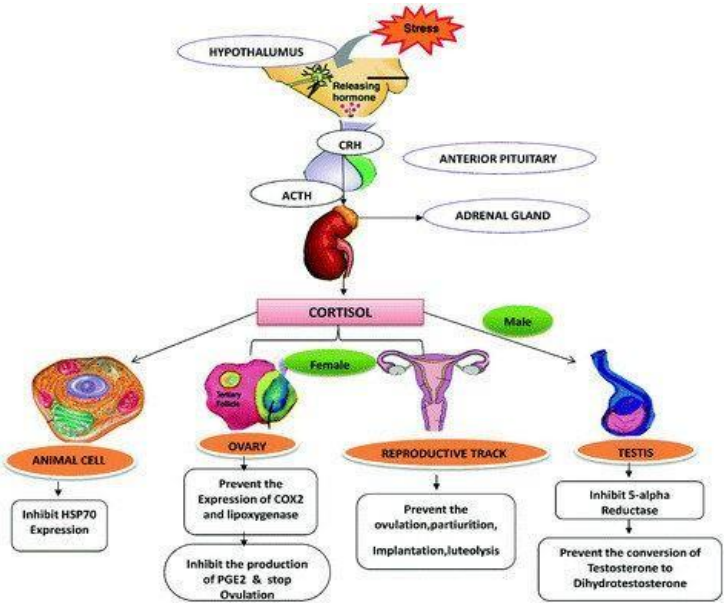

Figure 1: Different mechanisms with which stress affects reproduction in livestock. Corticotrophin releasing hormone (CRH); Adrenocorticotropic hormone (ACTH), Cyclooxygenase 2 (COX2); Prostaglandin E2 (PGE2) (Brown and Green, 2022)

Heat stress leads to hyperthermia, which triggers physiological responses aimed at regulating body temperature (Hansen, 2009). This stress can adversely affect various reproductive tissues and processes, both immediately and over time. The impact of heat stress on female reproduction is

complex, affecting several stages of reproduction (Table 1). High environmental temperatures interfere with pubertal maturation, ovulation, zygote formation, implantation, and embryo survivability (Wolfenson et al., 2000; Hansen, 2007). Severe heat stress can lead to delayed puberty, reduced estrus length and intensity, impaired follicular development, and an increased proportion of apoptotic follicles (Wolfenson et al., 2000; De Rensis and Scaramuzzi, 2003; Roth and Hansen, 2004; Badinga et al., 1993). The initiation of disturbance in normal physiology occurs when stress activates the hypothalamus pituitary adrenal gland. This activation causes the pituitary gland to release adrenocorticotropic hormone (ACTH), which stimulates the release of glucocorticoids and catecholamines. While these hormones help relieve stress, glucocorticoids also inhibit the reproductive axis (Sapolsky et al., 2000; Charmandari et al., 2005; Rivier and Rivest, 1991). Furthermore, heat stress particularly affects follicular recruitment during each follicular wave, hindering the normal growth and development stages of follicles needed for ovulation (Ozawa et al., 2005). This disruption in follicular growth impairs the progress and function of oocytes (Roth et al., 2000; Hasen et al., 2009). Continuous ACTH secretion alters follicular selection efficiency and dominance, further inhibiting ovulation and follicular development. Elevated glucocorticoid levels during the summer months directly inhibit the meiotic phase of oocyte maturation (Al-Katanani et al., 2002). Additionally, decreased luteinizing hormone and estradiol release, due to increased corticotropin-releasing hormone, results in reduced estrus length and intensity as ovarian steroidogenesis is inhibited (Khodaei-Motlagh et al., 2011).

Table 1: The effects of heat stress on reproduction of female livestock

Aspect	Effects of Heat Stress	References
General Impact	Disrupts endocrine balance, affecting physiology, production, and reproduction.	Takahashi, 2011; Danso et al., 2024
Reproductive Events	Impaired oocyte maturation, oestrus behavior, early embryonic development, fetal growth, and lactation.	Hansen, 2009
Immediate and Long-term Effects	Adverse effects on reproductive tissues and processes.	Hansen, 2009
Reproductive Process Disruption	Hampered at stages of pubertal maturation, ovulation, zygote formation, implantation, and embryo survivability.	Wolfenson et al., 2000; Hansen, 2007
Physiological Stress Response	Activation of the hypothalamus-pituitary-adrenal (HPA) axis, release of ACTH, glucocorticoids, and catecholamines to relieve stress effects.	Sapolsky et al., 2000; Charmandari et al., 2005
Reproductive Axis Inhibition	Inhibitory effect of glucocorticoids on the reproductive axis.	Rivier and Rivest, 1991
Puberty and Estrus	Delayed puberty, decreased estrus length and intensity.	Wolfenson et al., 2000; De Rensis and Scaramuzzi, 2003
Follicular Development	Deferred follicular development, a high proportion of apoptotic follicles.	Roth and Hansen, 2004; Badinga et al., 1993
Follicular Recruitment	Hampered during each follicular wave, subdues the growth and development of potential follicles to ovulate.	Ozawa et al., 2005
Oocyte Function	Altered follicular growth hampers oocyte progress and function.	Roth et al., 2000; Hansen et al., 2009
ACTH and Follicular Selection	Continuous ACTH secretion alters follicular selection efficiency and dominance, inhibiting ovulation and follicular development.	Al-Katanani et al., 2002
Oocyte Maturation	Elevated glucocorticoids during summer inhibit the meiotic phase of oocyte maturation.	Al-Katanani et al., 2002
Estrus Expression	Reduced estrus length and intensity due to decreased release of luteinizing hormone and estradiol.	Khodaei-Motlagh et al., 2011
Ovarian Steroidogenesis	Inhibited by increased corticotropic releasing hormone.	Khodaei-Motlagh et al., 2011

Heat stress and its disruptive effects on hormonal control of reproduction in female

Heat stress leads to hormonal imbalances in animals, reducing reproductive efficiency and interfering with fertility (Khan et al., 2023). This occurs primarily through the disruption of the hypothalamo-hypophyseal–gonadal axis, which governs the endocrine function of the female reproductive system (De-Rensis and Scaramuzzi, 2003). Key hormones such as Gonadotropin-

Releasing Hormone (GnRH), Follicle-Stimulating Hormone (FSH), and Luteinizing Hormone (LH) are the prime hormones in regulating the ovarian cycle. The pituitary gland releases both FSH and LH into circulation, where they play crucial roles in female reproduction. FSH is essential for puberty onset and ovarian health. It promotes the growth of ovarian follicles in the ovary, leading to the production of estrogen. This process prepares the oocyte for release during ovulation (Stocco, 2008). Similarly, LH is a gonadotropic hormone secreted by anterior pituitary gland cells. It stimulates the ovarian follicles to produce estradiol, the main female sex hormone, during the early stages of the cycle. Around day 14, an LH surge triggers ovulation, leading to the release of a mature oocyte. Additionally, LH supports the formation of the corpus luteum, which produces progesterone to sustain early pregnancy (Miller and Auchus, 2011; Niswender et al., 2000) . Heat stress has a strong inverse relationship with reproductive hormone concentrations, leading to low reproductive efficiency in farm animals (Naqvi et al., 2012; Sejian et al., 2016). Exposure to severe temperatures (40°C) during the estrous cycle causes a substantial reduction in GnRH-induced FSH production, as evidenced in cattle (Gilad et al., 1993). Furthermore, heat stress reduces estradiol levels, which in turn lowers the amplitude and frequency of LH pulses. This disruption compromises the maturation and ovulation of the dominant follicle (Wolfenson et al., 2000). Low tonic LH levels, exacerbated by heat stress, inhibit follicular growth and depress luteal development (Wolfenson et al., 2000). Additionally, decreased pre-ovulatory LH levels during the summer lead to reduced estrus expression and delayed ovulation. In buffalo, heat stress significantly alters the endocrine profile, often resulting in anestrus and reduced ovarian activity (Das and Khan, 2010). Estradiol and progesterone are critical hormones in female reproduction. The rise in estradiol during the estrous cycle triggers the LH surge, leading to oocyte maturation and ovulation. Estradiol also thickens the uterine endometrial lining. However, heat stress suppresses estradiol secretion by reducing the production of androstenedione by theca cells and limiting granulosa cells' aromatase activity and viability (Wolfenson et al., 2000; Roth et al., 2001). This reduction in estradiol leads to lower estrus expression in farm animals.

Several reports have suggested the detrimental effects of heat stress on endocrine function on female reproduction (Table 2). Similarly, heat stress decreases plasma progesterone concentrations, shrinking ovulatory follicles and reducing LH and estrogen levels (Wakayo et al., 2015). The diminished LH surge further hampers ovulation and corpus luteum (CL) formation, leading to reduced progesterone production (Khan et al., 2023; Wolfenson et al., 2018). This hormonal imbalance involving progesterone, inhibin, estradiol, and LH impairs fertility (Wolfenson et al., 2000). Additionally, heat stress increases circulating prolactin levels, which may contribute to the suspension of the estrus cycle and infertility in animals (Singh et al., 2013; Alamer, 2011; Bridges et al., 2005). Alterations in LH, FSH, and steroid hormones such as progesterone and estrogen at both the hypophyseal and ovarian levels further exacerbate fertility issues during heat stress (Roth et al., 2000; Ronchi et al., 2001).

Table 2: Heat stress and its effects on hormones associated with reproduction in female livestock

Aspect	Details	References
Hormonal Imbalances	Heat stress causes hormonal imbalances that lower reproductive performance and interfere with fertility.	Khan et al., 2023
Hypothalamo-Hypophyseal-Gonadal Axis	Heat stress affects the endocrine function of the female through this axis.	De-Rensis and Scaramuzzi, 2003
GnRH, FSH, and LH	These hormones control the ovarian cycle. The pituitary gland releases them into circulation.	Stocco, 2008; Miller and Auchus, 2011
Oestradiol Production	Oestradiol is produced by ovarian follicles when LH stimulates these during the first two weeks of the cycle.	Stocco, 2008
Progesterone Production	To support early pregnancy, progesterone is produced by the corpus luteum in response to LH stimulation.	Miller and Auchus, 2011; Niswender et al., 2000
Heat Stress and Hormone Levels	High ambient temperature has an inverse relationship with reproductive hormone concentrations, leading to low reproductive efficiency.	Naqvi et al., 2012; Sejian et al., 2016
Theca Cells and Androstenedione Production	Heat stress reduces androstenedione production in theca cells, leading to depressed oestradiol secretion.	Wolfenson et al., 2000

Aspect	Details	References
Granulosa Cells Aromatase Activity	Limited aromatase activity and viability in granulosa cells under heat stress also contribute to low oestradiol secretion.	Roth et al., 2001
Steroidogenic Hormones	Theca and granulosa cells produce steroidogenic hormones, which are suppressed during heat stress.	Para et al., 2018
Progesterone and Corpus Luteum	Heat stress decreases plasma progesterone concentration, affecting ovulatory follicle size and LH/estrogen production.	Wakayo et al., 2015
LH Levels and Ovulation	Reduced LH tonic levels and a lesser LH surge during heat stress hinder ovulation and functional corpus luteum emergence, reducing progesterone levels.	Khan et al., 2023; Wolfenson et al., 2018
Prolactin Levels	Circulating prolactin tends to increase under heat stress, suspending the estrus cycle and causing infertility.	Singh et al., 2013; Alamer, 2011; Bridges et al., 2005

Effects of heat stress on follicular growth, development, and oocyte competence

Heat stress significantly alters follicular growth and development, compromising ovarian follicular biomechanics across the estrus cycle which subsequently compromises the reproductive ability of farm animals (Badinga et al., 1993; Wolfenson et al., 1995; Wilson et al., 1998a). Mietkiewska et al. (2002) demonstrated heat stress induced impairment of oocyte development at different stages of ovarian follicles (Figure 2). Ovarian functions are negatively influenced by heat stress, which is associated with the inhibition of follicular growth and oocyte quality (Roth, 2017). A developing follicle turns into non-viable and experiences irreversible damage when it is exposed to 40°C (Roth et al., 1997). Heat stress can affect reproduction either immediately or over time (Roth et al., 1997, 2000; Torres-Júnior et al., 2008).

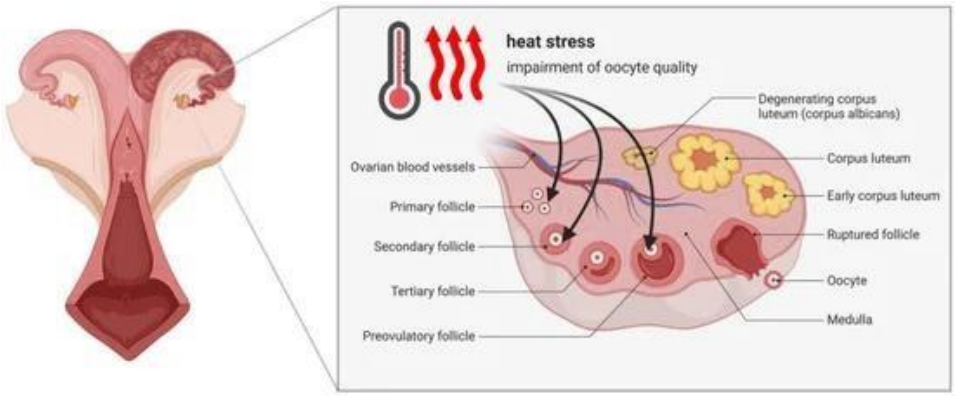

Figure 2: Impact of elevated environmental temperature (heat stress) on the impairment of oocytes at different stages of follicular development (Miętkiewska et al., 2022)

Animals under extreme heat stress have core body temperatures above 40°C and this elevated temperature is seriously harmful to the developing follicles (Roth et al., 2000). The development competence of oocytes is decreased by heat stress because of increased oxidative damage and apoptotic cell death, which consequently cause irreversible alterations to the cytoskeleton and meiotic spindle (Hansen, 2009). The synthesis of necessary proteins and formation transcripts needed for embryonic development may be harshly affected by heat stress (Edwards and Hansen, 1997). A decrease in mRNA and pre-embryonic storage protein along with changes in membrane integrity interfere with signal transduction and transport of protein. A lower steroid hormone secretion due to heat stress deflects the follicular development by upsetting the growth of oocytes which in turn halts the growth of dominant follicles (Badinga et al., 1993). The promotion of numerous large follicles over a diameter of 10 mm resulted in decreased follicular dominance and prolonged ovulatory follicle dominance (Hansen, 2009). Thus, the selection and dominance of normal follicles may be compromised in the presence of a substantial tonic FSH level (Wolfenson et al., 2000).

When gonadotropin is stimulated, follicles obtained from heat-stressed cows release lesser amounts of androstenedione and estradiol (Bridges et al., 2005). This is most likely because the expression of luteinizing hormone (LH) receptors is downregulated and the follicular response to LH is impaired (Ozawa et al., 2005). The lack of a sufficient level of LH combined with negative energy balance during summer interferes with the development process of the dominant follicle which leads to improper maturation and subsequent ovulation (Wakayo et al., 2015). Extended follicular dominance reduces the oocytes' aptitude for growth and impedes their proper maturation (Sakaguchi et al., 2004). If follicular dominance is maintained for an extended period, premature meiosis will occur and the oocyte will be aged and have poor development prospects. Ovulation of an aged follicle which consists of oocytes with improper competence could be the result of incomplete dominance (Mihm et al., 1994). As a result, ovulation of the infertile oocyte and subsequently small-size sub functional Corpus luteum form due to the development of an undersized dominant follicle during heat stress. During warm months, Heat-stressed Holstein cows exhibited a lower percentage of oocytes (Al-Katanani et al.,2002). Consequently, a robust fall in the ovulation percentage occurs due to the recession of the premature dominant follicle before reaching optimum size (Ozawa et al., 2005; Wakayo et al., 2015; Al-Katanani et al., 2002). Moreover, summer heat stress has a deleterious effect on the mRNA expression levels of several genes (MOS, GDF9, and POUF51) linked to oocyte growth and competence at different cell stages (Gendelman and Roth, 2012). Compromised-quality oocytes not only influence the genes that are related to transcription but also affect the development of the embryo, which results in apoptosis and reduced embryo quality (Roth and Hansen, 2005). Due to the detrimental effects of heat stress on the cytoplasmic and nuclear maturation of bovine oocytes, oocytes harvested in the summer are of lesser quality than those harvested in the winter (Roth, 2018). Heat stress affects different aspects of oocyte and follicular development in mammals (Table 3)

During oocyte maturation, heat stress not only disrupts the spindle fibers and microtubules essential for development but also diminishes the size of the meiotic spindle, ultimately impairing oocyte maturation (Ju et al., 2005). During heat stress high levels of glucocorticoids directly inhibit the meiotic maturation of oocytes (Khodaei-Motlagh et al., 2011). Oocytes under heat stress undergo decreased protein synthesis, changed microfilament and microtubule architecture, a damaged meiotic spindle, and an increased risk of triggering cell death due to apoptosis, according to in vitro studies (Roth et al., 2011). Reduced sperm penetration is caused by changes in the oocytes' cytoplasm and pellucid ozone layer as a result of increased free radical production and inadequate protein (Wolfenson et al., 2000). It has been found that metabolic markers like blood glucose level affect the follicular process of farm animals encountering heat stress (Takahashi, 2012). During the cool season, the glucose content of bovine follicular fluid is around 85% of the plasma glucose level whereas the follicular glucose level considerably drops along with a corresponding drop in blood glucose levels in the summer (Shehab-El-Deen et al., 2010). This suggests that factors such as blood nutrition or biochemical substances that change during the summer have an impact on the follicle formation process (Sakatani et al., 2012). Many species, including humans, pigs, cattle, and rabbits, often maintain their ovarian temperatures 1 to 1.5°C lower than their rectal temperatures (Grøndahl et al., 1996; Hunter et al., 2000; Grinsted et al., 1980a and Grinsted et al., 1985b). Regardless of pregnancy or nursing state, heat-stressed rabbits showed a 20–30% decrease in ovarian, cervical, and oviductal blood flows and a 40% increase in vulvar blood flow (Lublin et al., 1996) which indicates that local blood flow and body temperature may be related to each other, as well as the distribution of nutrients to follicles for oocyte development.

Table 3: Effect of heat stress on follicular growth, development, and oocyte competence

Aspect	Details	References
Oxidative Damage and Apoptosis	Increased oxidative damage and apoptotic cell death cause irreversible alterations to the cytoskeleton and meiotic spindle.	Hansen, 2009
Oocyte Development	Heat stress affects oocyte growth, protein synthesis, and transcript formation; Decreases mRNA and pre-embryonic storage protein.	Edwards and Hansen, 1997; Badinga et al., 1993
Steroid Hormone Secretion	Lower secretion due to heat stress disrupts follicular development and growth of oocytes, halting dominant follicle growth.	Badinga et al., 1993; Hansen, 2009; Wolfenson et al., 2000

Aspect	Details	References
Follicular Dominance	Promotion of large follicles decreases dominance; extended dominance reduces oocyte growth and maturation.	Hansen, 2009; Sakaguchi et al., 2004; Mihm et al., 1994
Hormonal Influence	Downregulated LH receptors and lack of sufficient LH impair follicular response; negative energy balance interferes with dominant follicle development.	Bridges et al., 2005; Ozawa et al., 2005; Wakayo et al., 2015
Gene Expression	Summer heat stress affects mRNA levels of MOS, GDF9, and POUF51; Compromised oocytes affect genes related to transcription and embryo development.	Gendelman and Roth, 2012; Roth and Hansen, 2005
Seasonal Variations	Lesser quality oocytes are harvested in summer; heat stress affects the cytoplasmic and nuclear maturation of oocytes.	Roth, 2018; Al-Katanani et al., 2002
Meiotic Spindle	Heat stress reduces meiotic spindle size; high glucocorticoids inhibit meiotic maturation during heat stress.	Ju et al., 2005; Khodaei-Motlagh et al., 2011; Roth et al., 2011
Sperm Penetration	Changes in the cytoplasm and pellucid zone layer reduce sperm penetration due to increased free radicals and inadequate protein.	Wolfenson et al., 2000
Blood Glucose	Blood glucose levels affect the follicular process; follicular glucose levels drop in summer.	Takahashi, 2012; Shehab-El-Deen et al., 2010
Ovarian Temperature and Blood Flow	Ovarian temperatures are 1-1.5°C lower than rectal temperatures; heat-stressed rabbits show decreased ovarian, cervical, and oviductal blood flows.	Grøndahl et al., 1996; Hunter et al., 2000; Grinsted et al., 1980a, 1985b; Lublin et al., 1996
Nutrient Distribution	Local blood flow and body temperature are related to nutrient distribution to follicles.	Lublin et al., 1996; Sakatani et al., 2008

Changes in estrus incidences and behavior under heat-stressed conditions

In farm animals, day length primarily regulates the seasonal cycle of reproduction. Estrus incidences, duration, intensity, and behavior have been demonstrated to be negatively affected by heat stress (Dash et al., 2016). Increased incidences of anestrus and silent heat were observed in farm animals during encountering higher temperatures (Singh et al., 2013; Kadokawa et al., 2012). When the ambient temperature crosses above 20.5°C, estrus occurrence in cattle goes down (Bulbul and Ataman, 2009). Extreme temperatures combined with high humidity and solar radiation also negatively influence the reproductive rhythm expression in cattle and buffaloes (Upadhyay et al., 2009). Hence in dairy cattle and buffaloes, summer estrus detection rates were found to be lower than those of spring and winter. The majority of Indian river buffaloes have been seen to demonstrate a diurnal pattern of estrus behavior, with estrus occurring during the cooler part of the day (Vale, 2007). Low temperatures in the morning and evening are associated with the highest levels of sexual activity, whereas afternoons are associated with the lowest levels (Chawicha and Mummed, 2022). The lowest percentage of estrous incidents occurred around midday, with 12% of cases beginning between 9.00 and 15.00 hours. Estrus indications were most noticeable in the early morning and evening (15.00–21.00 hours) (El-Wardani and El-Asheeri, 2000).

In summer, when the ambient temperature drops at night hours, cows exhibit estrus (West, 2002). Similar to cattle, poor estrus expression is often seen in Indian buffalo due to the low level of estradiol on the day of estrus during the summer months which obstructs estrus detection in buffaloes in summer (Upadhyay et al., 2009). Anestrus is caused by an increase in the animal's ACTH-cortisol ratio under heat stress, which alters endocrine secretion (Collier et al., 2006). Numerous factors, such as the time of year, the environment, the photoperiod, temperature, and nutrition, significantly affect the length of the estrus cycle and the degree of estrus expression in cows (El-Wardani and El-Asheeri, 2000). As a result, silent ovulation and delayed ovarian activity were observed in cattle during heat stress (Collier et al., 2006). Under heat stress, reduced blood hormone levels play a key role in suppressing estrus expression in cows. Similarly, decreased duration of estrus and delayed estrus onset had been observed in heat-stressed ewes due to abnormal LH pulsatility, inadequate synthesis of estradiol from granulosa and theca cells of oocytes during heat stress (Maurya et al., 2005). Shortened Estrus cycle length was also observed due to heat stress (Torres- Júnior et al., 2008).

In female farm animals, estrus or sexual behavior is a key determinant of reproductive

soundness which has been demonstrated to be adversely affected by extreme temperature (Alejandro et al., 2014). Mounting to the other animals, restlessness, mucus discharge from the vagina, frequent urination, and bellowing are the typical estrusbehavior in farm animals. Sexual behavior in farm animals is controlled by the estradiol concentration during estrus. In dairy cattle the duration, and intensity of estrus as well as estrous cycle length reduced by heat stress which increases anestrus and silent ovulation rate (Younas et al., 1993; Gwazdauskas et al., 1981; Trout et al., 1998; Para et al., 2018). The number of mounts is also reduced in hot weather creating difficulty in detecting estrus, as a result, the success of artificial insemination and pregnancy rate are reduced (Pennington et al., 1985). Heat stress inhibits follicular growth during the pre-ovulatory period of proestrus, lowering estradiol levels and, thus, limiting the intensity of estrus signs (Wilson et al., 1998). Endocrine activities in females are shown to be affected by heat stress. High ambient temperature hampers estradiol (E2) secretion in goats (Ozawa et al., 2005). In sheep, heat stress also lowers progesterone levels and results in a decrease in LH surge (Hill et al., 1981). Heat stress lowers the levels of estradiol, follicular estradiol concentration, aromatase activity, and the amount of LH receptor linked to delayed ovulation in goats (Ozawa et al., 2005). Reduced estrus expression could also be explained by heat-related physical inactivity. During estrus, cows are less active and less prone to ride other cows. cows' decreased motor activity, which is an attempt to diminish endogenous heat generation, also lowers estrus activity (Samal, 2013). The luteal phase in cows under thermostress is lengthier than in cows under thermoneutral stress. Because of reduced synthesis of estradiol and/or because heat disrupts Prostaglandin F2alpha (PGF2α) release from endometrial cells, the uterus secretes less PGF2α. (Chawicha and Mummed, 2022). The uterine endometrium needs to be primed by estradiol to create the necessary amounts of prostaglandin for luteolysis (Chawicha and Mummed, 2022). An increase in prolactin circulation during heat stress is linked to the cessation of estrous cycles and infertility (Bridges et al., 2005; Alamer, 2011; Singh et al., 2013). Buffaloes may become acyclic or infertile in the summer due to a noticeably higher mean plasma prolactin concentration than in the winter (Roy and Prakash, 2007).

Heat stress and fertility

In the dairy industry, infertility induced by heat stress is a global concern. Heat stress may result in economic loss for roughly 60% of the global dairy cattle stock (Wolfenson et al., 2000). In tropical and subtropical areas during the heat season, low-quality oocytes and embryos result in a lower conception rate and longer days open, which leads the dairy industry to encounter enormous financial losses (Collier et al., 2006). Heat stress affects the high-yielding lactating cows more than heifers, as they possess a higher metabolic rate, which generates more internal heat resulting in lower fertility percentage in summer and fall (Ahmed et al., 2015). The conception rate in the first service decreased from 25% to 7% when the temperature rose from 29.7°C in April to 33.9°C in July (Cavestany et al., 1995). When compared to the winter season, the conception rate can decrease by roughly 20–30% in the summer (Para et al., 2018). A dramatic reduction in the fertilization rate occurs when the uterine temperature rises 0.5°C above normal temperature (Alejandro et al., 2014). In the summertime, a massive decline in animal fertility may be attributed to several factors, including increased embryonic mortality, decreased progesterone production, ovulatory failure risks, and poor oocyte quality or embryonic development (Wolfenson et al., 2000). Heat stress-induced hyperthermia can disrupt cellular function across various tissues of the female reproductive tract (Para et al., 2018). It has also been suggested that hyperthermia-induced fertility disruptions are related to oxidative stress (David Wolfenson and Zvi Roth, 2019). Decreased fertility has been seen in cattle and sheep exposed to heat stress before insemination (Ahmed et al., 2015). Conception rates are inversely correlated with elevated ambient temperature on the day of insemination (Nabenishi et al., 2011; Schuller et al., 2014; Greer et al., 1983). According to a different study, heat stress was found to negatively impact conception throughout the breeding phase, 42 days before and 40 days following insemination (Schuller et al., 2014).

Conception rates in summer were significantly lower than in winter in dairy cows chosen for high milk production (Lopez-Gatius, 2003). There are two primary developmental stages of the estrus cycle in farm animals. The stage of development known as the follicular phase occurs when the oocyte matures on the ovary surface as a follicle. The size of the follicle grows as the oocyte ages, reaching its maximum size when the antral follicle bursts, freeing the oocyte for fertilization. Reduction in follicle selection and increase in the size of pre-ovulatory follicles are the results of heat stress. It has been demonstrated that larger follicles and delayed ovulation lower the likelihood of conception (Roth et al., 2000). Heat stress also has an impact on oocyte fertilization. A study found that when cows experience heat stress, the rate of fertilization decreases from

83% in the non-stressed cows to just 37% in the stressed animals (Hackbart et al., 2002). This indicates that a rise in the mother's body temperature probably modifies the oocyte, which reduces the rate of fertilization. Doe and Smith (2023) revealed that heat stress affects overall fertility in mammals (Figure 3). Heat stress can impede the early growth and development of an embryo (Putney et al., 1989). Effects were detected as early as the two and four-cell stages. Heat-stressed embryos exhibit delayed development, often failing to reach critical milestones by day 17, a pivotal point in embryonic growth (Hansen, 2009). The hormone that lowers PGF2α secretion, interferon-tau (INFT), is not produced in sufficient amounts in underdeveloped embryos. PGF2α release will result in regression of Corpus Luteum (CL) in the absence of interferon-tau or insufficient production, which

will cause early embryonic mortality and the animal to go back intoestrus (Robinson et al., 2008). In high-yielding cows of Israel, the conception rate was found 45% in winter which declined to 20% in summer due to the negative effect of heat stress (Wolfenson et al., 2000; Roman-Ponce et al., 1978 and Amundson et al., 2006). Following a threshold Temperature Humidity Index (THI) of 75, the adverse result of heat stress on fertility is observed in buffaloes (Dash et al., 2016). Furthermore, for each unit increase in THI beyond 70 a reduction of 4.6% in conception rate was seen in dairy cows (West, 2007 and Paula-Lopes et al., 2012).

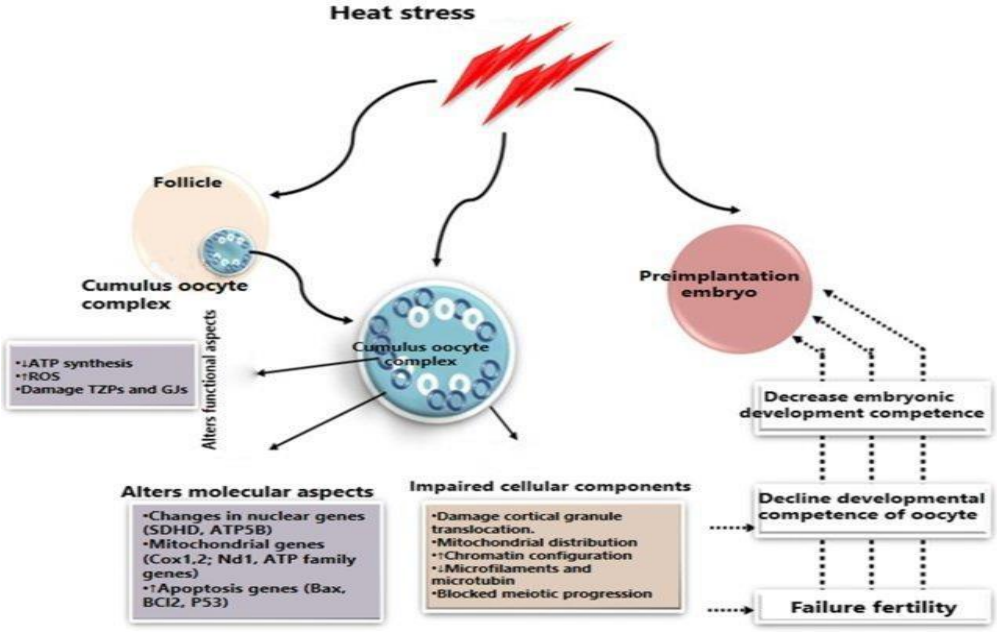

Figure 3: Effects of heat stress on fertility (Doe and Smith, 2023)

Effects of heat stress on embryonic growth and development

Numerous in vitro and in vivo studies have investigated how heat stress impacts the development and growth of embryos (Vendrell-Flotats et al., 2017; Hansen, 2007; Roth et al., 2001) . After fertilization, cows exposed to heat stress produce fewer and lower-quality embryos (Kastelic et al., 2001; Setchell, 1998). Various studies have shown that post-fertilization heat stress reduces embryo quantity and quality in cows after superovulation, while also hindering fetal development in pigs, mice, and beef cattle (Wettemann et al., 1988; Putney et al., 1988; Ealy et al., 1993; Ozawa et al., 2002; Aroyo et al., 2007; Baumgartner et al., 1988; Roth et al., 2008; Ozawa et al., 2004; Biggers et al., 1987). The surrounding environment significantly affects the growth and development of pre-implantation embryos following fertilization. Studies in mice (Arechiga et al., 1998) and cows (Ealy et al., 1995; Sakatani et al., 2004) have demonstrated that heat stress during the pre-implantation stage impedes embryo growth and reduces the total number of blastocyst cells. Maternal heat stress during the first 15 days after breeding is especially critical for the survivability of the mobile embryo (Wakayo et al., 2015; Ahmed et al., 2015). Among the early stages of development, two-cell embryos are more vulnerable to heat stress compared to four- and eight-cell stages, whereas later stages such as morula and blastocysts are more resilient (Wolfenson and Roth, 2019; Hansen, 2007). Both in vivo and in vitro investigations have shown that heat exposure significantly slows the development of cow embryos, particularly at 48–72 hours following fertilization, or the 8–16 cell stage (Sakatani et al., 2004). Beyond this stage, heat stress has less impact on the rate of development and cell proliferation. While heat

stress slows blastocyst growth, it does not affect the cleavage rate (de S Torres-Júnior et al., 2008). In lactating cows, heat stress after day one of estrus reduces the likelihood of embryo development to the blastocyst stage by day 8 (Ealy et al., 1993). Exposure of embryos to high temperatures up to the blastocyst stage, both in vitro and in vivo, leads to lower pregnancy rates up to day 30 and increased embryo loss by day 42 of gestation (Demetrio et al., 2007). Embryos, particularly those at the zygotic genome activation (ZGA) stage, are highly sensitive to heat stress. In cows, ZGA occurs at the 4- to 8-cell stage (Telford et al., 1990; De Sousa et al., 1998). Heat stress during this period alters chromatin structure, which can disrupt gene expression during and after ZGA (Edwards et al., 1998).

Heat stress-induced embryonic loss is a significant factor affecting the fertility of farm animals (Figure 4). The detrimental effects of heat stress on embryonic development before implantation are the results of both direct heat stress and harmful alterations to the reproductive tract. Elevated uterine temperatures induced by heat stress can adversely affect developing zygotes and embryos, leading to embryonic loss. Possible mechanisms for heat stress-induced embryonic death include reduced protein synthesis, oxidative damage, failure in pregnancy recognition, and apoptosis (Krishnan et al., 2017; Chawicha and Mummed, 2022; Hansen, 2009). Progesterone, often referred to as the pregnancy maintenance hormone, plays a crucial role in embryonic development and is secreted by the corpus luteum (CL) of pregnant females. In dairy and beef cattle, summer heat inhibits luteal function, leading to reductions in progesterone levels in theca cells, plasma, and luteinized granulosa cells (Howell et al., 1994; Burke et al., 2001; Wolfenson et al., 2002). The luteal dysfunction during heat stress further contributes to embryonic loss. PGF2α, primarily secreted by the uterus, is one of the key factors regulating luteal function. Heat stress increases PGF2α production in the endometrial tissues of both pregnant and non-pregnant bovine uteri (Putney et al., 1988). Additionally, maternal heat stress raises the levels of PGFM and PGF2α in the placenta (Nakamura et al., 2004). Elevated PGF2α levels can disrupt embryo development, prevent implantation, and cause luteal regression (Stocco et al., 1998). Researchers have observed a negative correlation between higher uterine luminal concentrations of PGF2α and embryo quality and pregnancy rates (Schrick et al., 1993). Thermal stress may also compromise embryonic and fetal development by altering uterine blood flow and the uterine environment (Yadav et al., 2022) and is associated with fetal malnutrition and other stress conditions that may lead to post-implantation embryonic death (Kadokawa et al., 2012).

Heat stress may also influence the sex ratio of offspring, although this phenomenon has not been conclusively demonstrated in livestock species. In mice, when females were mated with males exposed to heat stress on the day of mating, the sex ratio of the embryos shifted in favor of females (Perez-Crespo et al., 2008).

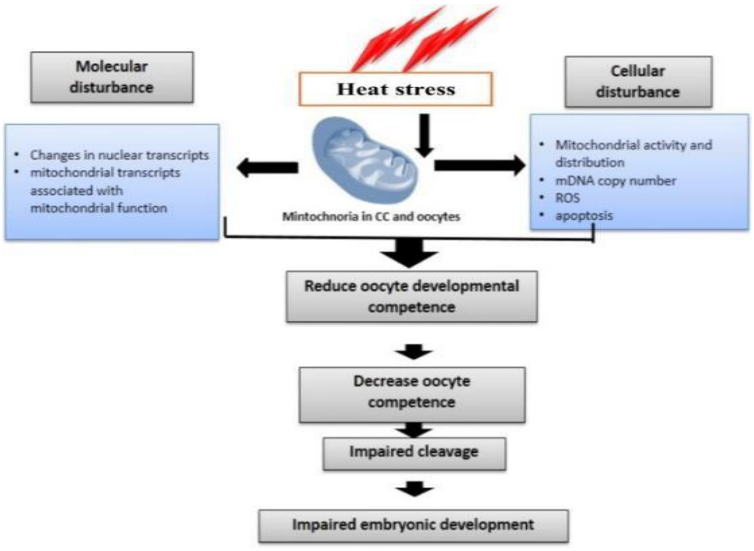

Figure 4: Effect of heat stress on embryonic development (Doe and Smith, 2023)

Effects of heat stress on pregnancy

The two most interesting aspects that have a direct impact on the profitability of the livestock industry are reproduction and production. The reproductive ability of an animal is adversely affected by heat stress in a variety of ways including reduced size corpus luteum (CL) formation, altered estrus events and conception, hampered follicular development, and incomplete uterine endometrium preparation for retaining the fetus (Dash et al., 2016).

It has been seen that the pregnancy rate decreases up to 50% if the rectal temperature of the animal goes higher up to 38.5 to 40 °C at 3 days after insemination (Ryan et al., 1993). Another study showed that in Bos Taurus cattle, the conception rate decreased by 3.2% for every unit rise in (temperature humidity index) THI above 70 and by 3.5% for every degree increase in ambient temperature above 23.4°C (Amundson et al., 2005). Furthermore, heat stress during pregnancy severely impairs fetal growth because it reduces the blood flow, depriving the conceptus of the nutrients and hormones it needs to promote normal growth and development (Amundson et al., 2006; Savasani et al., 2015). An embryo that develops slowly may fail to signal the dam's uterus effectively at the critical time, leading to increased endometrial secretion of PGF2alpha during heat stress, which can compromise pregnancy maintenance by inducing luteolysis (Upadhyay et al., 2009).

Most embryonic loss due to heat stress occurs during the early pregnancy of 8 to 17 days (Savasani et al., 2015; Inskeep and Dailey, 2009) during the peri-implantation period of pregnancy. Heat stress also stunts the embryo's growth up until day 17, which is usually considered the critical period for the production of IFNT from cattle embryos (Krishnan et al., 2017). In cattle, Interferon Tau (INFT) acts as the pregnancy recognition signal from the conceptus, inhibiting the release of luteolytic PGF pulses from the uterus. This allows for the maintenance of functional corpora lutea (CL), essential for producing the progesterone needed to initiate and sustain pregnancy.

Heat Stress Management Approaches

Heat stress poses a significant threat to livestock production, animal welfare, and financial sustainability. As climate change intensifies, the frequency and severity of heat stress events are expected to rise, putting even greater pressure on livestock producers to implement effective mitigation strategies. Minimizing heat stress in livestock is essential not only for maintaining animal health and well-being but also for ensuring sustainable production. In this part, several potential approaches to mitigate heat stress in livestock have been stated, ranging from environmental modifications and management practices to genetic selection and technological interventions. By adopting these strategies, livestock producers can better safeguard the welfare and productivity of their animals while enhancing the resilience of their operations in the face of escalating climatic challenges.

Adaptation of Physical Techniques

Provision of shade

Providing adequate shade is a key management practice for mitigating the harmful effects of heat stress in livestock. Shade not only protects animals from direct sunlight but also helps lower the surrounding temperature, reducing heat discomfort. The practice of offering shade primarily aims to lessen the heat load caused by direct solar radiation. Shading structures can be either natural, such as trees, or man-made. Trees are considered the most cost-effective source of shade, as they not only block sunlight but also cool the environment through moisture evaporation from their leaves. In addition, artificial shading can be optimized by painting the tops of shade structures white and adding a 2.5 cm layer of insulating material, which can significantly reduce solar radiation (Buffington et al., 1983). To maximize the reduction in solar radiation, shade structures in corrals should be 3.6 to 4.2 meters high. Studies have shown that shading can reduce the radiant heat load by at least 30%. Furthermore, shading feed and water sources has been demonstrated to improve the productivity of cattle breeds originating from Britain and Europe (Slimen et al., 2014).

Evaporative cooling techniques

Utilizing evaporative cooling systems is an effective strategy for mitigating heat stress in livestock. These systems leverage the natural cooling effect of water evaporation to provide animals with thermal relief while moderating ambient temperatures. By creating microclimates tailored to meet the animals' physiological needs, farmers can reduce the harmful effects of heat stress and promote optimal conditions for growth, reproduction, and productivity. Evaporative cooling systems typically combine water misting with forced ventilation using fans and sprays to reduce the heat load on livestock (Lourenço et al., 2021). These systems are commonly installed in free-

stall barns or under coverings in open-space corrals. In dairy farms, particularly those located in hot climates, various cooling methods are employed to relieve heat stress in cows. The most common setups include fans, soakers, shades, and water sprinklers. Water is applied to the cows, and fans circulate air over them, facilitating evaporative cooling. Globally, many dairies in hot regions incorporate features such as fans, sprinklers, misters, and foggers to combat heat stress (Chawicha and Mummed, 2022). The adoption of evaporative cooling systems has been shown to enhance the reproductive efficiency and milk production of dairy cattle (Sejian et al., 2012). Additionally, cows cooled with sprinklers tend to consume more feed and use water more efficiently, leading to increased milk yield, fat content, protein levels, and overall productivity (Igono et al., 1987). Fogging and misting systems utilize tiny water droplets that evaporate quickly, cooling the surrounding air. While these cooling methods are crucial in reducing heat stress, they do not fully prevent the negative impacts on reproductive function (Dash et al., 2016; Sejian et al., 2016).

Air movement and conditioning

Increasing air movement is a key strategy for promoting evaporation, which enhances the cooling effect of perspiration in animals. This airflow helps to dissipate heat generated by the animals through radiation, conduction, and convection. Additionally, it aids in removing moisture in the form of vapor, contributing to a cooler environment. By cooling the surrounding areas—such as the dirt, walls, roofs, and fences—the air conditioning effect helps to alleviate heat stress in animals. This approach leads to improvements in growth rates, milk production, and milk composition. However, despite its benefits, the high cost of electrical power makes it an economically challenging solution during hot weather conditions (Marai et al., 2008).

Cool water for drinking of the animals

Animals require water to sustain their physiological processes, and this need becomes even more critical during hot weather. Cold drinking water plays an essential role not only in quenching thirst but also in supporting the body's thermoregulation processes, which help release excess body heat. Providing animals with access to cool water during periods of heat stress can improve feed intake, hydration, and overall well-being. Moreover, in hot climates, increased water intake enhances heat dissipation by promoting evaporative heat loss through breathing, sweating, and conduction. As a result, body temperature is better regulated, minimizing the adverse effects of heat stress (Aboulnaga et al., 1989; Daader et al., 1989; Habeeb et al., 1994; Marai et al., 1997). By ensuring access to cool water, farmers can safeguard the health and productivity of their animals, helping them thrive despite the challenges posed by an ever-warming climate.

Shearing or clipping

By removing extra wool, hair, or feathers from an animal's body, shearing or clipping helps to improve thermal control and heat dissipation. Shearing or clipping improves an animal's capacity to release excess body heat, which lessens the impacts of heat stress and improves overall health by lowering the insulating layer that traps heat near the skin. Studies have shown that shearing leads to significant decreases in respiratory rates, as well as skin and rectal temperatures (Bianca, 1959; Marai et al., 2008). Shorn animals exhibit a higher growth rate when kept in houses and on the range. However, the skin may be damaged if the clipped skins are exposed to direct sunlight. In this situation, portions of the animals' coats can be clipped to provide an appropriate covering for the skin.

Dietary strategies

Dietary strategies play a critical role in managing animals' physiological responses to heat stress. By optimizing dietary composition, nutrient balance, and feeding practices, farmers can enhance animals' heat tolerance, support adaptive mechanisms, and reduce the negative effects of thermal stress. Nutritional interventions offer a holistic approach to addressing the complex relationship between heat stress and animal physiology, from adjusting feed formulas to incorporating additional supplements (West, 2003).

Heat-stressed animals tend to consume less crude protein and dry matter, leading to a negative energy balance due to reduced feed intake. The elevated core body temperature and inefficient heat dissipation increase the animals' energy requirements for maintenance. To counteract this, feeding high-quality concentrates and additional fats can improve the nutrient density of their diets. Feeding bypass fat to ruminants during periods of low feed intake provides an energy-rich diet. Diets rich in fat may help lower the heat load on animals since fat has a higher calorie density and generates less metabolic heat than fiber or carbohydrates (Chawicha and Mummed, 2022). Additionally, dietary fat intake enhances levels of progesterone, cholesterol, PGF2α synthesis and metabolism, follicle growth, and pregnancy rates in dairy herds (Sejian et al., 2015). A balanced

ration during summer months provides sufficient energy to minimize reproductive and health issues caused by lower dry matter intake (DMI) and total digestible nutrient intake (TDN) (Gorniak et al., 2014).

Easily digestible carbohydrates are essential for stressed animals, and increasing feeding frequency and feed concentration helps boost rumen buffering and saliva production. This is particularly important as rumen acidosis is a common issue in heat-stressed ruminants. Chase (2013) recommends incorporating buffering agents, such as magnesium oxide, sodium sesquicarbonate, and sodium bicarbonate, into the diet to maintain a normal rumen environment by reducing acidity. Additionally, raising potassium levels is beneficial, as potassium is the principal regulator of the sweat glands in ruminants (Chawicha and Mummed, 2022).

Increasing salt and mineral intake, as well as adding yeast or yeast cultures to the ration, may also help alleviate heat stress (Chase, 2013). Feeding animals at night can be beneficial, as it reduces the impact of heat stress during the day (Frigeri et al., 2023). Supplementing with vitamin C has also been shown to alleviate heat stress in animals (Marai et al., 2008).

Heat stress can cause oocytes to produce more reactive oxygen species, leading to an imbalance between pro- and antioxidant levels, which negatively affects fertility (Hansen et al., 2019). To counteract this, exogenous antioxidant supplementation—such as vitamins C, A, and E, along with trace elements like zinc, manganese, copper, selenium, and chromium—can reduce the harmful effects of environmental stress (Jan et al., 2011; Para et al., 2018). In buffaloes, antioxidant supplementation during heat stress can improve fertility by reducing oxidative stress (Dash et al., 2016), and antioxidant feeding during summer months has been shown to enhance reproductive function in cows (Arechiga et al., 1998).

Feeding bypass fat to ruminants during periods of low feed intake provides an energy-rich diet. Diets rich in fat may help lower the heat load on animals since fat has a higher calorie density and generates less metabolic heat than fiber or carbohydrates (Chawicha and Mummed, 2022). Additionally, dietary fat intake enhances levels of progesterone, cholesterol, PGF2α synthesis and metabolism, follicle growth, and pregnancy rates in dairy herds (Sejian et al., 2015).

By incorporating these dietary strategies, farmers can help animals better cope with heat stress, thus safeguarding their health, productivity, and reproductive success.

Bio-physiological strategies

Pharmacological and nutraceutical treatments, such as the administration of diaphoretics, diuretics, and goitrogens, may help mitigate the effects of heat stress in animals. Diaphoretic compounds promote increased sweating, enhancing evaporative cooling and thereby helping heat-stressed animals dissipate excess heat (Marai et al., 2008; El Fouly, 1969; Kamal et al., 1972; Marai et al., 1995). Recent research has demonstrated that feeding lactating cows encapsulated niacin can increase sweating rates and lower core body temperatures compared to thermally neutral controls. Niacin induces vasodilation of peripheral blood vessels, improving heat dissipation (Para et al., 2018). However, these treatments may also elevate respiration rates and rectal temperatures (Marai et al., 2008).

Diuretic compounds are used to promote heat loss by increasing water excretion. By excreting urine at body temperature and consuming water cooler than body temperature, heat-stressed animals can reduce their core heat load (Daader et al., 1989; Marai et al., 2008). Goitrogens inhibit the thyroid's absorption of iodine, which suppresses thyroid gland activity. This reduces the secretion of thyroxine (T4), thereby lowering heat production in heat-stressed animals (Marai et al., 2008).

The inclusion of fungal cultures in the diet has been shown to lower body temperatures and respiration rates in animals exposed to hot conditions (Para et al., 2018). Additionally, feed additives that enhance heat dissipation, along with encapsulation strategies to bypass the rumen or modifications to cellular biochemical composition, can improve reproductive function during summer heat stress (Bilby et al., 2008; Para et al., 2018).

Several strategies to minimize embryonic loss due to heat stress have also been explored. The development of thermotolerance in embryos appears to be linked to the balance between antioxidant defenses and free radical production (Wolfenson and Roth, 2019). Sakatani et al. (2007) found that administering antioxidants, such as dithiothreitol and anthocyanin, in vitro could protect embryos from heat shock. Furthermore, the use of specific caspase inhibitors to prevent heat-induced apoptosis increased embryonic survival. Insulin-like growth factor 1 (IGF-I) supplementation in vitro has also been shown to enhance embryos' resistance to heat shock (Jousan and Hansen, 2007).

pharmacological and nutraceutical interventions, along with advanced reproductive techniques, can help mitigate the adverse effects of heat stress in animals, improving both health and

reproductive outcomes during periods of extreme heat.

Development of heat-resilient breeds through genetic selection

One important strategy for minimizing the adverse effects of heat stress on livestock seems to be genetic selection. By intentionally selecting and breeding heat-tolerant breeds, we can improve livestock populations' ability to thrive in increasingly harsh temperatures and make them more resilient. Our objective is to maximize livestock productivity while preserving animal welfare by using genetics to alter animals into ones that are more resistant to heat stress through targeted breeding efforts. Production and heat tolerance have an antagonistic connection. Reduced heat tolerance results from production selection that gradually overlooks heat tolerance. Genetically more thermos-tolerant cows also have greater breeding values for fertility (Nguyen et al.,2016). Sadly, they also have a lesser genetic capacity for milk production; therefore, genetic techniques must be developed to enable selection for genes that provide better thermotolerance without sacrificing milk production. Heat tolerance varies significantly between breeds and even among members of the same breed. To sustain high productivity and survivability under heat-stress circumstances, it is helpful to identify and select dairy animals that can withstand the heat. There are two approaches to enhancing animal adaptation to climate stress. One approach involves choosing animals that thrive in hot environments, while another involves using gene editing or crossbreeding to modify a dairy animal's DNA to make it more resistant to heat (Chawicha and Mummed, 2022). Heat shock resistance is a feature that cells inherit from their corresponding genes for coat color and hair length (Hansen et al., 1999).

Important factors that influence the body's non-evaporative heat loss are the thickness and weight of the hair coat per unit area (Dash et al., 2016). Because the slick hair gene is linked to higher sweating rates and lower metabolic rates in animals, it has been determined to provide increased thermal resistance (Dikmen et al., 2008). The slick hair gene, which causes a slick hair coat, enhances heat tolerance ability when introduced into cattle breeds from temperate climates (Berman et al., 2011). Other genes that have been linked to heat resistance and stress adaptation in cattle include the heat shock protein (Heat stress P) gene and the ATP1A1 gene (Dash et al., 2016; Liu et al., 2010; Loredana et al., 2011). The ATP1A1 gene controls the electrochemical gradient of Na+ and K+ across the plasma membrane, which is essential for preserving cellular homeostasis and the balance of body fluids (Dash et al., 2016). The Sahiwal and Tharparkar cattle and buffalo in India were found to have significantly expressed Heat stress P70 family genes throughout the summer, which improves their capacity to adapt to dry, hot, and humid environments and increases their thermotolerance (Kumar et al., 2015). One gene, heat stress PA1L, has mutations that make cells more resistant to heat shock (Hansen, 2019). Breeding strategies for improving both reproductive ability and climate adaptability should focus on identifying major genes linked to thermo-tolerance, which lessens the effects of heat stress in cattle and buffaloes. These genes can then be incorporated into breeding programs through marker-assisted selection. Other genes that are important economically are glycosyltransferase, phosphofructokinase, protein kinase C, fibroblast growth factor, thyroid hormone receptor, interleukins, and ATP1B2 (Pursley et al., 1998; Dash et al., 2016). However, before these genes can be employed as biological markers in marker-assisted selection programs, more in-depth investigation is necessary to determine the expression pattern of these genes across a range of livestock species to develop breeds that are thermotolerant and capable of normal reproduction.

Estrus synchronization and timed artificial insemination (TAI)

In modern farm animal reproductive management systems, protocols for synchronizing estrous cycles and TAI have grown in importance. By synchronizing estrus cycles within a group of animals, producers can prevent themselves from depending too heavily on natural mating cues, which may be compromised by physiological changes brought on by heat. TAI, which enables insemination to occur at predetermined intervals following synchronization, significantly improves this precision. This reduces the probability of missed breeding opportunities while increasing the likelihood of successful fertilization. Combined with a GnRH injection to initiate regulated recruitment of the ovulatory follicle, the timed artificial insemination strategy also increases summer fertility (Santos et al., 2010). To allow for the final maturation of ovulation-causing follicles, this regimen should be followed and PGF2α injected seven days later to regress the CL. To guarantee successful conception, cows or buffaloes may undergo insemination at 16 hours and ovulation at 48 hours following PGF2α (Pasha et al., 2021; Pursley et al., 1998). When paired with TAI, the Ovsynch procedure successfully synchronized buffalo ovulation and enhanced the conception rate (Pasha et al., 2021). Thus, an effective substitute strategy for managing cow reproduction during heat stress is TAI. However

according to a recent study, although the TAI regimen aids in reproductive management, it does not protect the embryo from the detrimental effects of elevated temperatures. (Pursley et al., 1998 and Para et al., 2018).

Hormone Therapy

One possible strategy to minimize the negative effects of heat stress on animal reproduction is the use of hormonal therapies. These treatments provide a potential means of preserving the sustainability and viability of animal husbandry in the face of heat stress brought on by climate change by regulating hormone levels and improving reproductive processes (Hansen, 2019). GnRH, when given at the beginning of estrus, coincides with the natural surge of LH, which effectively boosts the rate of conception. On the fifth day of the estrous cycle, an hCG injection or GnRH agonist induces luteinization of the dominant first-wave follicle, ovulation, and the development of an auxiliary corpus luteum (CL), which increases plasma progesterone levels to compensate for a drop in these levels brought on by prolonged heat stress (Wolfenson et al., 2000). In comparison to untreated (41%) controls, lactating dairy cows given GnRH injections during the summer and autumn at the earliest indications of standing estrus showed higher conception rates (56%) (Para et al., 2018).

Application of reproductive biotechnologies

Several studies have shown that using the embryo transfer procedure can significantly improve the number of pregnancies throughout the summer by preventing embryonic death during the first seven days of development when the embryos are much more vulnerable to heat stress (Para et al., 2018). Given that pre-implantation embryos at early stages are highly sensitive to heat stress, it has been suggested to transfer embryos at day 8, once they have passed the thermosensitive developmental stages, to avoid the negative effects of heat stress (Hansen, 2013). Compared to artificial insemination (AI), the transfer of frozen or unfrozen superovulation-produced embryos has enhanced the rate of conception in heat-stressed cows (Stewart et al., 2011).

Conclusions

Climate change due to global warming significantly impacts animal health, reproduction, and productivity, primarily through heat stress. Heat stress disrupts hormonal balance by affecting the pituitary gland, altering folliculogenesis, estrous events, embryogenesis, and pregnancy leading to reduced reproductive efficiency. Heat stress disrupts the ovarian and uterine environments, impairing follicular development and diminishing steroid production, especially estradiol. This weakens estrus expression, and shortens its duration, resulting in poor oocyte quality, failed fertilization, impaired embryonic development, and higher embryo mortality rates. To mitigate these effects, strategies such as dietary management, environmental modifications, and genetic improvement can be employed. Advanced reproductive techniques, including hormone therapy, timed artificial insemination, and embryo transfer, can also improve fertility and reproductive outcomes in farm animals despite heat stress.

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