

Effect of Heat Stress on Female Reproduction in Dairy Animals

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<https://doi.org/10.5281/zenodo.7998773>

Abstract

Heat stress impairs fertility and reproductive function in livestock by impairing the physiology of the reproductive tract, resulting in hormonal imbalances, lower oocyte quality, and decreased embryo development and survival. Heat stress reduces luteinizing hormone and estradiol release, resulting in shorter and weaker estrus expression, an increased incidence of anoestrus, and silent heat in agricultural animals. Thermally stressed oocytes lose their ability to fertilise and grow into the blastocyst stage, resulting in lower fertility due to the creation of poor-quality oocytes and embryos. Low progesterone secretion also inhibits endometrial processes and, as a result, embryo development. Furthermore, increased release of endometrial prostaglandin F₂ alpha under heat stress jeopardises pregnancy maintenance. Heat stress in animals can be reduced by implementing scientifically sound measures that include environmental change and nutritional management. Furthermore, advanced reproductive technologies such as hormone therapies, timed artificial insemination, and embryo transfer may be used to combat summer infertility in farm animals, potentially increasing the chances of establishing pregnancy.

Keywords: Heat stress, dairy animals

Introduction

The female reproductive process is hampered by high environmental temperatures at various stages during pubertal development, conception, and embryonic death. Stress decreases livestock species' reproductive function by activating the hypothalamic-pituitary-adrenal (HPA) axis, which then activates the pituitary gland, causing it to release adrenocorticotrophic hormone (ACTH). The ACTH hormone induces the release of glucocorticoids and catecholamines, which help to relieve

stress. The reproductive axis, on the other hand, is inhibited by ACTH-stimulated glucocorticoid production.

In the antral and pre-antral follicles, heat stress lowers the length and intensity of estrus, changes follicular development, and raises the rate of apoptosis. Furthermore, heat stress during follicular recruitment inhibits subsequent follicular growth and development leading to delayed ovulation. Changes in follicular development obstruct the oocytes' ability to proceed and function. Chronic ACTH release, such as that caused by heat stress, reduces ovulation and follicular development by affecting the efficiency of follicular selection and dominance, and glucocorticoids are important mediators of this reproductive inhibition. Furthermore, a high level of glucocorticoids during heat stress directly slows oocyte meiotic maturation, and corticotropic releasing hormone (CRH) suppresses ovarian steroidogenesis, which is caused by a decrease in luteinizing hormone release (LH). As a result of the drop in estradiol, the length and intensity of estrus expression is reduced.

Effect of heat stress on reproductive hormones in female livestock

Reproductive hormones play an important part in the female reproductive system, since they govern various stages of growth and function. The hypothalamic-hypophyseal-ovarian axis may be influenced by high ambient temperature and solar radiation as a result of climate change. Several studies have also discovered a substantial negative relationship between environmental temperature and reproductive hormone concentration, which results in reduced reproductive efficiency in farm animals. The gonadotropin-releasing hormone (GnRH) from the hypothalamus and the gonadotropins (FSH and LH) from the anterior hypophysis are the two most essential components that govern ovarian activity. GnRH-induced FSH secretion is found to be significantly reduced in cattle after a 16-hour exposure to a higher temperature (40°C) on day 12 of the estrous cycle, although tonic FSH secretion was raised, likely due to reduced inhibition of negative feedback from smaller follicles. Low tonic LH levels also affect luteal development by reducing follicular growth, whereas heat stress reduces LH pulse amplitude and frequency in cattle with low estradiol, compromising the maturation and ovulation of dominant follicles. Furthermore, during heat stress, a decrease in pre-ovulatory LH secretion reduced the expression of estrus behaviour and delayed ovulation.



Effect of heat stress on follicular growth and development

When the core body temperature rises above 40°C, it causes harm to the developing follicles. Heat stress interrupts oocyte growth, lowers the growth of dominant follicles, and increases the growth of subordinate follicles by lowering steroid hormone release, which disrupts oocyte growth. Furthermore, heat stress is also linked to reduction in follicular dominance by causing the formation of multiple big follicles with diameters greater than 10 mm, as well as a prolonged dominance of ovulatory follicles. As a result, high tonic follicular stimulating hormone (FSH) availability may disrupt the selection and dominance of normal follicles. The formation of small dominant follicles under higher temperatures results in ovulation of the infertile oocyte or sub-functional corpora lutea, as prolonged follicular dominance impairs normal oocyte maturation and lowers developmental competence. The premature dominant follicle's regression before it reaches the greater size results in a significant fall in ovulation percentage.

Effects of heat stress on estrus incidences

The photoperiod controls the seasonal cycle of reproduction in female animals, and it has been discovered to be greatly influenced by climate change. Heat stress has been shown to have a deleterious impact on estrus incidence and duration, and thus on estrus detection in several investigations. The duration and severity of estrus are inversely related to ambient temperatures, with greater temperatures causing an increase in anestrus and silent heat in farm animals. When compared to winter (23.4 days), the inter-estrous interval in Japanese black cattle was found to be significantly shorter during the summer (21.5 days).

Embryonic growth and development

Another key factor affecting cattle fertility is embryonic loss, and bovine embryos are susceptible to maternal heat stress during the first two weeks following breeding. The negative effects of increased body temperatures on developing zygotes and embryos may be a primary source for a loss in embryonic survival caused by heat stress. During egg maturation and ovulation, as well as the first 3–7 days of pregnancy, high ambient temperatures affected embryonic viability and development. Although increased temperatures have an effect on embryos in the pre-attachment stage, the effect diminishes as the embryo develops. Heat stress induces embryonic death through



interfering with protein synthesis, oxidative cell damage, a decrease in effective pregnancy detection, and the production of apoptosis-related stress genes.

Pre-partum period and days open

The dry period is a crucial time for mammary gland involution, rapid foetal growth, and lactation induction, as well as subsequent mammary development. Heat stress in cows inhibits the release of placental hormones, which can have a deleterious impact on intrauterine foetal growth and milk output. Heat stress during pregnancy can alter endocrine responses, which can lead to an increase in foetal abortions, a shorter gestation period, a lower calf birth weight, and a reduction in follicular and oocyte maturation during postpartum estrous cycles. Pre-partum heat stress may lower thyroid hormones and placental oestrogen levels while raising non-esterified fatty acid levels in the blood, affecting udder and placenta growth, placental angiogenesis, food supply to the unborn calf, and milk production.

Mitigation strategies to ameliorate the impact of heat stress

The consequences of heat stress on cattle result in significant economic losses for farmers, but there are few opportunities to recoup some of these losses by using appropriate heat stress mitigation methods. Physical adaptations of the environment, nutritional management, and genetic creation of breeds that are less sensitive to heat stress are three important critical components for maintaining the productivity of animals in hot environments. These tactics can be used singly or in combination to improve performance by providing the best possible environment for farm animals to work in. In addition, modern reproductive technologies such as gonadotropins, timed artificial insemination, and embryo transfer may be used to address summer infertility.

In general, livestock environmental management is a growing field in animal science that is receiving more attention in the face of climate change. It aims to provide an appropriate microclimate for optimum productivity by minimising negative environmental impacts on animal production systems. The primary methods of modifying the environment can be classified into two categories: (i) providing shade and (ii) using evaporative cooling techniques. During heat stress, environmental adjustments like as shade and cooling systems are crucial to maintaining milk production, milk component levels, reproductive function, and animal welfare in arid and semi-arid zones. The principles of providing shade may be traced back to efforts to reduce heat load in animals caused by



direct solar irradiation. Natural or artificial shade structures could be used. Because they protect from the sun and collect sunlight through evaporation of humidity in the leaves, trees are said to be the most cost-effective technique of providing shade. The cooling systems reduce the heat burden on livestock by combining water misting and forced ventilation through the use of spray and fans, and are commonly used within free-stall barns or under shade in open area. The adoption of an evaporative cooling system improves milk output and reproductive function in dairy cattle.

Amid order to maximise livestock output in changing climatic conditions, it is critical to ensure optimal nutritional levels for the animals. The need of providing adequate nutrition to enable optimum reproduction in animals should not be underestimated, as their energy balance is intimately linked to their fertility. Environmental temperatures are highest in arid and semi-arid locations, where available feed resources are both low in quality and quantity, negatively impacting livestock species' reproductive success. Combating the metabolic effects of heat stress is consequently critical, as animals suffering from mild to severe heat stress require an additional 7–25% maintenance requirement. Feeding feed additives promotes energy use while also stabilising the rumen environment. In addition, lipid content in the diet has a positive impact on cholesterol, progesterone, PGF₂ synthesis and metabolism, follicle growth, and pregnancy rates in dairy herds. Vitamin, trace element, and mineral supplements can also help to reduce the negative effects of heat stress. Injections of vitamin E and selenium lower rectal temperature and body weight loss in sheep during the summer. Inorganic chromium supplementation in the feed of buffalo calves reared in hot environments increased heat tolerance and immunological status without influencing nutritional intake or growth performance.

Hormonal treatments have the ability to reduce the consequences of heat stress in animals. The administration of GnRH during the early phases of estrus corresponds with the release of endogenous LH, resulting in a successful increase in the conception rate. On day 5 of the estrous cycle, a GnRH agonist or hCG injection causes ovulation or luteinization of the first wave dominant follicle, as well as the formation of an auxiliary corpus luteum (CL) that boosts plasma progesterone levels to compensate for persistent heat stress. When combined with a GnRH injection to promote a controlled recruitment of the ovulatory follicle, the timed artificial insemination (AI) programme increases summer fertility. This approach should be followed by a PGF₂ injection 7 days later to regress the CL and allow the ovulatory follicles to mature fully. Furthermore, 48 hours after PGF₂,



a second dose of GnRH may induce ovulation and cow insemination at 16 hours, ensuring successful pregnancy. When paired with timed AI, the Ovsynch procedure successfully synchronised ovulation in buffaloes and boosted conception rate. Embryo transfer (ET) improves pregnancy rates in the summer because embryos are transferred after they are more susceptible to heat stress. Transfer of either frozen or unfrozen embryos produced by superovulation has enhanced pregnancy rates in cows exposed to heat stress when compared to AI.

Conclusion

Increased temperature and relative humidity, as a result of climate change, will undoubtedly cause heat stress in all livestock species, affecting their reproductive potential. Fortunately, there are tried-and-true methods for reducing the effects of heat stress on animal reproduction. These include maintaining animals in climate-controlled environments, using scheduled AI algorithms to overcome poor estrus detection, and implementing embryo transfer programmes to avoid heat-related harm to the oocyte and early embryo. To help livestock cope with severe weather, management options such as strategic use of shade, wind protection, sprinklers, and ventilation in the heat should be explored. There are also some interesting research pathways that could lead to novel ways to improve reproduction during heat stress. Antioxidants are given and the growth axis is manipulated, among other things.

