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Mechanisms of cortisol-mediated inhibition of reproductive hormones in heat stressed cows: A mini-review

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Abstract

The reproductive performance of cows is significantly influenced by stress-induced cortisol release, especially in situations of physiological and environmental stress, like heat stress. The zona fasciculata of the adrenal cortex produces cortisol, a glucocorticoid hormone that is a major indicator of stress, and is mostly controlled by the hypothalamic-pituitary-adrenal (HPA) axis. The processes by which high cortisol levels negatively impact bovine reproduction are examined in this review. Increased cortisol concentration in the circulatory system impairs follicular development, ovulation and oestrus behaviour by blocking the secretion of gonadotropin-releasing hormone (GnRH) and gonadotrophins [luteinizing hormone (LH) and follicle stimulating hormone (FSH)], which upset the hypothalamic-pituitary-gonadal (HPG) axis. Cortisol affects steroidogenesis, lowers oocyte quality and modifies folliculogenesis at the ovarian level by interacting with glucocorticoid receptors. Furthermore, the important reproductive hormones like progesterone and oestradiol are suppressed by stress-induced cortisol which results in poor follicular maturation, decreased embryo viability and lower conception rates. Long-term stress especially heat stress, increases embryonic mortality and modifies gene expression linked to embryonic development thereby aggravating these consequences. In general, high cortisol levels negatively affect fertility by disrupting oocyte competence, hormonal balance and gestational processes. Developing efficient management techniques to reduce stress and increase the reproductive performance of cows requires an understanding of these mechanisms.

Keywords: Cattle (cows); Heat stress; Cortisol; Female reproduction; Folliculogenesis; Gonadotropin-releasing hormone.

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Introduction

Cortisol ($C_{21}H_{30}O_5$), which is secreted by the zona fasciculata of the adrenal cortex of the adrenal gland, is the hormone that is mostly produced during stress, and is a helpful biochemical marker of stress in animals (Martinez *et al*., 2016). According to Massey *et al.,* (2014), cortisol is a glucocorticoid hormone that has been shown to be a reliable biological correlate of a number of detrimental health consequences. It is involved in various processes such as metabolism, blood pressure and immune response control.

When exposed to acute heat stress, plasma cortisol levels rise within twenty minutes and plateau in two hours (Christison and Johnson, 1972). Since higher cortisol concentrations may be associated with higher levels of stress and pain, cortisol is the hormone most frequently employed to identify and quantify the extent of the stress and pain response (Molony *et al*., 1993; Fisher *et al*., 1996; Ley *et al*., 1996). However, animals that have lower cortisol concentrations are unable to experience stress (Stilwell, 2012). Cortisol is one of the primary hormones generated during stress reactions in animals because stress can activate the hypothalamic-pituitary-adrenal system, and it is easily activated with increasing cortisol production (Kalin *et al*., 1992).

There is clear evidence that hyperthermia is detrimental to all forms of production from the perspective of animal welfare at both high ambient temperatures and high direct and indirect solar radiation (Silanikove, 2000). In cattle, heat stress results in ovarian inactivity due to hyper-prolactinemia, decreased luteinizing hormone frequency, inadequate follicle maturation and decreased oestradiol synthesis (Wolfenson *et al*., 2000). Adrenocorticotropic hormone (ACTH) may be involved in some symptoms of heat stress. Additionally, high temperatures disrupt the release of some reproductive hormones in cows such as progesterone, follicle-stimulating hormone (FSH) (Howell *et al*., 1994; Burke *et al*., 2001; Wise *et al*., 1988; Gilad *et al*., 1993). Heat stress reduces follicular steroidogenesis, androstenedione and oestradiol levels in the follicle wall, according to an in vitro study from bovine species (Bridges *et al*., 2005).

Mechanism of action of stress-induced cortisol inhibition of reproductive performances via endocrine interactions

Stress triggers the hypothalamic-pituitary-adrenal (HPA) axis (Minton, 1994). The interactions between HPA axis with the hypothalamic-pituitary-gonadal (HPG) axis influence the reproductive system negatively (Collier *et al*., 2017). HPA axis activation causes the secretion of corticotropin releasing hormone (CRH) into the median eminence by neurones in the hypothalamus. Adrenocorticotropic hormone (ACTH) is released from pituitary corticotroph cells when CRH passes through the hypothalamic-pituitary portal system after secretion from the neurons of the hypothalamus. The adrenal gland produces and secretes glucocorticoids (cortisol) in response to adrenocorticotrophic hormone stimulation. Stressors that activate the HPA axis may result in infertility by preventing LH secretion (Breen and Karsch, 2006).

Gonadotropin releasing hormone (GnRH) neurones' cell bodies are located where certain CRH neurones in the hypothalamus end (Wade and Jones, 2004). GnRH release from GnRH neurones may be inhibited when CRH neurones are activated and release CRH. Because GnRH neurones lack the type II glucocorticoid receptor, glucocorticoids have inhibitory effects on the release of GnRH and LH, but these effects are not directly mediated by glucocorticoids. The hypothalamic kisspeptin, neurokinin B and dynorphin (KNDy) cells have a glucocorticoid receptor and can

provide the glucocorticoid signal to the GnRH neurones (Ralph *et al*., 2016; Scott *et al*., 2019).

Long-term stressors, like heat stress, typically trigger the sympatho-adrenal system and the hypothalamo-pituitary-adrenal axis (Tibrook *et al*., 2000). Corticotrophin-releasing factor (CRF) and arginine vasopressin (AVP) neurones in the paraventricular nucleus are activated when the hypothalamo-pituitary-adrenal axis is stimulated and these neuropeptides are secreted into the hypophysial portal system to stimulate the corticotrophs of the anterior pituitary gland (Tibrook *et al*., 2000). Heat stress causes the corticotrophs to secrete melanocyte-stimulating hormone, endorphin and ACTH among other peptides generated from pro-opiomelanocortin (Engler *et al*., 1989). ACTH acts on the cortex of the adrenal glands to boost the synthesis and production of glucocorticoids like cortisol, however the physiological significance of the release of endorphin and melanocyte-stimulating hormone in response to heat stress is unclear. Heat-stressed cows may produce higher concentrations of this catabolic hormone to sustain milk supply (Albiley *et al*., 1975).

The triggering of the hypothalamic-pituitary-adrenal (HPA) axis due to stress reduces livestock species' ability to reproduce, causing infertility (Collier *et al*., 2017). The pituitary gland releases adrenocorticotropic hormone (ATCH) which triggers the release of catecholamines and glucocorticoids or cortisol, both of which significantly act to cushion the effects of stress and have inhibitory effect on reproduction. Heat stress is linked to the persistent release of ACTH which causes the adrenal gland to produce cortisol which affects ovulation and follicular growth by changing the effectiveness of follicular selection and dominance.

Effects of cortisol and glucocorticoid activity on the function of the bovine ovary (cumulus cell) during ovulation

Although the ovaries lack the enzymes required for cortisol manufacture and they do exhibit glucocorticoid receptors, they are impacted by cortisol. The glucocorticoid receptor (GR) is a receptor through which cortisol works (Coutinho and Chapman, 2011; Hapgood *et al.*, 2016; Timmermans *et al*., 2019). Regardless of their level of maturity, bovine cumulus cells have been demonstrated to express GR (Anbo *et al*., 2022). In the development of cumulus oophorus, local cortisol synthesis increases concurrently with progesterone (P_4) production (Tetsuka *et al*., 2016; Tetsuka and Tanakadate, 2019). The steroidogenic enzyme 11β-hydroxysteroid dehydrogenase type1 (HSD11B1) mediates this process by converting the inert glucocorticoid cortisone into active cortisol (Krozowski *et al*., 1999; Michael *et al*., 2003; Seckl, 2004; Holmes and Seckl, 2006). Additionally, the locally synthesised P_4 in the cumulus cells increases the expression of HSD11B1 and cortisol production (Anbo *et al*., 2022). These findings imply that glucocorticoids are produced and function in bovine cumulus cells, and that P_4 can activate this system during the periovulatory phase. When follicular synthesis of progesterone and 17 alpha-hydroxyprogesterone reaches levels that displace cortisol from its binding proteins and make it available for biological action, ovarian follicles then regulate the biological activity of cortisol. Free cortisol, which is present in the pre-ovulatory follicle soon before ovulation, works to lessen the inflammatory-like processes that accompany ovulation. This explains why the presence of a dominant follicle during oestrus is associated with rise in serum levels of cortisol (Andersen, 2002; Ram *et al*., 2023).

Effects of stress-induced cortisol on folliculogenesis, oocytes and ovulation

Folliculogenesis is controlled at the endocrine level by a cascade mechanism involving the ovary, anterior pituitary and central nervous system. Gonadotropin-releasing hormone (GnRH) is secreted into the portal blood vessels by specialized hypothalamic neurons, which act on gonadotrophs to cause a pulsatile release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH), which in turn act on ovarian follicle cells to regulate folliculogenesis. Similarly, follicle derived hormones and growth factors can act locally to modify (amplify or attenuate) the activity of GnRH, FSH and LH which are crucial in controlling folliculogenesis (Kumar and Sharma, 2014; Hsueh *et al*., 2000). When high level of cortisol is sustained throughout the late luteal and early follicular phases of the oestrous cycle, there is inhibition of follicular growth and development, which prevents the preovulatory surge of LH (Doney *et al*., 1973; Welsh and Johnson, 1981; Wilson *et al*., 1998). Corticosterone alters the molecular pathways underlying oocyte maturation and competence for future development by reducing phosphorylated forms of extracellular signal-regulated kinases (ERKs), phospho-ERK (p-ERK)-1 and p-ERK-2 in exposed oocytes. This represents a change in the molecular pathways driving oocyte maturation and competence for later development (Gonzalez *et al*., 2010a).

Heat stress genes like HASP14 and Cx34 become active when the outdoor temperature climbs above 25°C in summer. Since these stress genes are linked to apoptosis among other processes, apoptosis can be important for both the development of the embryo and the follicle/oocyte from the start to metaphase II (Pavani *et al*., 2016; 2017). Insufficient gonadotropin production is the cause of follicular development and atresia during anestrus (Das and Khan, 2010).

According to Ranabir and Reetu (2011), cortisol has an adverse influence on oocyte maturation and subsequent embryonic development following IVF. Exposing oocytes to high concentrations of cortisol reduced their competence and impaired oocyte developmental potential by causing ovarian cells to undergo apoptosis through the activation of a transmembrane protein which is part of the tumour necrosis factor (TNF) and is commonly referred to as Fas ligand (FasL). Studies conducted on cortisol-injected mice showed that cortisol has a definite detrimental effect on oocyte nuclear maturation rate (NMR), blastocyst rates and the number of cells per blastocyst (Raghavendra *et al*., 2007; Yuan *et al*., 2016).

Effects of heat stress induced rise in cortisol levels on reproductive hormones

Glucocorticoids play a crucial role in mediating the inhibitory effect on reproduction during heat stress by directly preventing oocyte meiotic maturation. Additionally, corticotropic releasing hormone (CRH) inhibits ovarian steroidogenesis which is caused by a decrease in luteinizing hormone (LH) secretion (Al-Katanani *et al*., 2002). ACTH has been shown to prevent estradiol-induced sexual behaviour (Heinz and Allrich, 1992) and heat stress can enhance cortisol release (Roman-Ponce *et al*., 1981; Wise *et al*., 1988; Elvinger *et al*., 1992). Since this can inhibit GnRH and hence LH secretion, increased corticosteroid secretion has been proposed (Gilad *et al*., 1993).

Heat stress may inhibit oocyte growth by reducing the synthesis of pre-ovulatory surges in luteinizing hormone and oestradiol, which results in poor follicular maturation and ovarian inactivity in cattle (Hansen, 2007). High cortisol levels can affect the physiology of reproduction because it decreases the amplitude of GnRH and LH secretion as well as the levels of plasma oestradiol in the follicular phase (Wagenmaker *et al*., 2009; De Graaf

Roelfsema *et al*., 2007). According to Lotfi and Mendonca (2016) and Lee *et al*., (2007), cortisol suppression of the protein ERKs disrupts their functions in meiotic maturation of fully developed oocytes and/or arrest at metaphase of meiosis II before fertilization. Numerous studies suggest that glucocorticoids in general and cortisol in particular may contribute to the anti-gonadal action of stress, even if the exact causal relationship between stress and infertility has not been established (Dobson and Smith, 1995; Chrousos *et al*., 1998). Furthermore, elevated cortisol levels decrease the generation of oestradiol by potentially altering granulosa cell activities within the follicle leading to a decline in oocyte quality and a reduced capacity to develop following fertilization (Prasad *et al*., 2016).

Effects of heat stress-induced cortisol on bovine fertility and reproductive efficiency

Several in vivo studies have shown that rise in cortisol levels specifically impacts bovine metabolism and reproduction (Mudron *et al*., 2005; Pavani *et al*., 2016). Extreme weather, travel, laparoscopy and psychological stress are examples of stressful stimuli that suppress or decrease the manifestation of behavioural oestrus and ovulation which lowers fertility in domestic species. These stressors do not only lower fertility but also activate the hypothalamic-pituitary-adrenal (HPA) axis. Stressors related to management are frequently linked to a significant rise in cortisol levels in the blood (Prasad *et al*., 2016; Mahdy *et al*., 2017; Komesaroffet *et al*., 1998).

Earlier reports show that bovine fertility sharply declines to 35% during the summer and reaches 78% during the cooler seasons of spring and autumn (Pavani *et al*., 2017). A number of theories have been proposed to explain this closure during warmer seasons (Mahdy *et al*., 2017; Pavani *et al*., 2016; 2017). When calves are subjected to stressors like high temperatures for extended periods of time, cortisol levels can rise to a maximum of approximately 250 µM (Polsky and Von Keyserlingk, 2017; Gonzalez, 2010b). According to Pavani *et al*., (2015), the effects of high ambient temperatures on reproductive processes include decreased bovine fertility throughout the summer, altered physiological mechanisms, inhibition of in vitro oocyte maturation and low in vitro embryonic development. When cows were exposed to a mild heat stress (35°C) over an extended period of time, their plasma cortisol levels dramatically increased (Pavani *et al*., 2016; Mahdy *et al*., 2017). According to Khodaei-Motlagh *et al.,* (2011), heat stress lowers blood progesterone levels, which is a major factor in aberrant oocyte maturation, implantation failure, and ultimately early embryonic demise in dairy cattle.

Effect of cortisol levels on conception, gestation and embryo development in cows

In cattle breeds managed under extensive system, high cortisol levels directly interfere with the physiological processes that control ovulation, conception and pregnancy establishment (Cooke, *et al*., 2009; Faria *et al*., 2023). Heat stress affects the expression of several genes linked to the growth and development of embryos including connexin 43 (Cx43), E-cadherin (CDH1), DNA methyltransferase 1 (DNMT1) and heat shock protein family A member 14 (HSPA14). The down-regulation of these genes may be the cause of the lower maturation rate. In contrast, the HSPA14 gene is a crucial component of the cell's machinery for folding, unfolding, transport, protein localization and differentiation, controlling the embryonic cell cycle and aiding in stress protection. As a result, HSPA14 which is an apoptotic gene brought on by heat shock is linked to the death of embryos and plays a significant part in the regulation of processes related to

growth, cellular differentiation and embryonic development (Pavani *et al*., 2016). Additionally, Cx43 mediates the effects of heat stress on cells and is expressed in a variety of tissues including gonads (Mahdy *et al.,* 2017).

As an active glucocorticoid and anti-inflammatory hormone, cortisol modulates the synthesis and function of prostaglandins and cytokines necessary for ovulation, luteolysis, embryo implantation, foetal growth and placental development (Seth *et al*., 2016). Cortisol can also modify corpus luteum function by affecting prostaglandin F2α release (Duong *et al*., 2012). Low rates of embryo implantation and consequently pregnancy loss are caused by cortisol's detrimental effects on the corpus luteum's progesterone release during the early stages of pregnancy (Parent *et al*., 2002; Faria *et al*., 2023). Research on temperament and acclimatization effect of stress on reproductive performance in beef cattle has shown that aggressive animals have higher plasma cortisol concentrations, lower rates of pregnancy, calving and weaning, as well as lower calf weight at birth and weaning (Cooke *et al*., 2012). Excitable temperament also has a negative impact on pregnancy rates in cows subjected to natural breeding (Faria *et al*., 2023).

Conclusion

Stress induced cortisol release has a significant and complex impact on the ability of cows to reproduce. Any stressor that activates the hypothalamic-pituitary-adrenal (HPA) axis or pathway and causes elevated cortisol levels in circulation has an inhibitory effect on reproduction or infertility in cows. Physiological and environmental stressors such as heat stress activate the HPA blocking the hypothalamic-pituitary-gonadal (HPG) axis suppressing the reproduction efficiency in cows. Follicular development, ovulation and oestrous expression are ultimately hampered by this disturbance which results in decreased secretion of important reproductive hormones such as GnRH, LH, FSH, progesterone and oestradiol. Cortisol lowers oocyte quality and developmental competence at the ovarian level by adversely affecting folliculogenesis, oocyte maturation and steroidogenesis. Prolonged exposure to high cortisol concentrations also affects gene expression, impairs implantation and gestation processes thereby increasing embryonic mortality. Similarly, these physiological disruptions decrease conception rates, lower fertility and poor reproductive efficiency in cattle. Therefore, optimizing reproductive performance in cows requires effective management measures targeted at reducing stress, especially heat stress. A scientific foundation for boosting animal welfare, increasing productivity and creating focused interventions in cattle production systems is provided by comprehending the pathways through which cortisol disrupts reproductive processes.

Conflict of interest

The author declares that the research, authorship and publication of this review were all free from any conflicts of interest. There is no institutional, financial, personal or professional tie that might be interpreted as affecting the work's neutrality or integrity.

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