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Endocrine changes in livestock during heat and cold stress

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Abstract
Livestock experience various types of stressors throughout their lifetime. Thermal stress imposed by fluctuations in environment temperature, that extend both above and below the thermoneutral zone of an animal, remains the major concern affecting animal production. The adaptation of the animals towards change in climate is mainly related to physiology and its adaptation to the environment. Climate change has adverse effects on animal reproduction and production when animal is subjected to heat and cold stress conditions. The endocrine system is responsible for controlling the stress response, and rapid and appropriate growth is one of the elements of greatest economic importance towards production. This review focuses on the endocrine responses to thermal stimuli during passive heat or cold exposure, with particular responses to the changes in the body core temperature. Activation of the neuroendocrine system affects the secretion of hormones and neurotransmitters which act collectively as response mechanisms that allow them to adapt to stress.

Keywords: heat stress, climate change, hypothalamo-pituitory -adrenal axis, renin-angiotensin system, endocrine profile, cold stress

1. Introduction
In developing nations, livestock plays a major role in the agricultural sector. Livestock sector contributes 40% to the agricultural GDP [1]. Livestock are adversely affected by the detrimental effects of extreme weather conditions [2]. Consequently, adaptation and mitigation of detrimental effects of climatic extremes on livestock has recently gained impetus [3]. Climate change refers to as rapid changes in the environment over short period (couple of years) or more subtle changes over decades, with general increase in global temperature.
Climate change is a major threat to the sustainability of livestock systems globally [4]. There are a number of climate related stressors such as cold, heat, humidity, rain, ice, and wind that can affect the endocrine system and influence the performance of an animal such as the reproductive system and normal estrous cycle of animals [5]. However, current knowledge available to scientist and producers is mostly based on heat stress research.

2. Effects of climate change on livestock
A) Direct effects
The direct effect of climate change on livestock production comes from heat stress. Livestock spend a lot of energy to get rid of excessive body heat, causing significant reductions in animal productivity and performance. In dairy herd it reduces milk yield and alters milk composition, in beef cattle in affects meat production, besides negatively affecting reproductive efficiency and animal health in general.

B) Indirect effects
Climate change alters patterns of precipitation rate, affecting quality, quantity and distribution of rainfall, melting snow resulting river flow and ground water recharge system and ultimately affect water availability, agriculture and drinking water supply. High global temperatures alter species composition and quality, as well as ecological structure of grasslands and forage production [6]. The water deprivation to animal, affects internal physiological homeostasis of body, leading to reductions in body weight, lowered reproductive efficiency and decreased disease resistance [7]. In addition, climate change may also flare up the occurrence and emergence of several vector borne diseases, which further hamper livestock economy.

3. Heat stress
Heat stress occurs when the body temperature is elevated, due to excessive metabolic heat production or high ambient temperatures, or reduced heat loss [8]. Extreme ambient conditions can negatively affect animal production. Temperature, humidity, ventilation and

light are related to the regulation of metabolic processes in animals, affecting the ability of the individual to adapt to its environment [9]. In this way, many productive and reproductive parameters can be compromised, like nutrient intake, weight gain, milk production and fertility [5]. Temperature Humidity Index combines effects of two variables temperature and humidity on animal production, serves as an indicator of effects of potential heat stress periods on livestock. Physiological responses to thermal stress in animals include the activation of endocrine, autonomic and central nervous systems, as well as cardiovascular mechanisms for redistribution of blood flow. All of them act in a synergistic way depending on the magnitude of the stress factor and they respond to it by generating physiological mechanisms to suppress or decrease the threat of the adverse effects of the stressor [6].

4. Neuroendocrine responses to thermal stress

Animals experience constant physiological changes in order to cope with diverse environmental situations (thermal, social and location), which can activate neuroendocrine responses. Stress leads to the activation of physiological mechanisms required to maintain homeostasis. Stress compromises important metabolic functions such as reproduction, immunity and growth [10]. Stress can be classified as acute or chronic depending on whether the exposure to drastic changes in temperature is for a short or long period of time. Both activate neuroendocrine pathways that modify physiological processes in order to maintain homeostasis to ensure survival of the animal [11]. Stress hormones, produced in response to an increase in environmental temperature, induce the following effects in animals: Mobilization of energy for maintenance of muscular and neural functions; increase in the perception of the environment; increase in brain perfusion for delivery of glucose; improvements in cardiovascular and respiratory functions; modulation of immune responses; decrease in reproductive and sexual functions; and decrease in appetite [12]. Stress induces several neuroendocrine responses in animal which include activation of various hormonal axis and release of tropic hormones which mediate the adaptive and behavioral responses in animals. Majorly released are adrenocorticotropic hormone (ACTH), thyrotrophic hormone (TSH), somatotropic or growth hormone (GH), follicle stimulating (FSH) and luteinizing (LH) hormones, and prolactin (PRL). Some functions of these hormones will be discussed.

I. Hypothalamus-hypophyseal-adrenal axis (HHAa)

Hans Selye was the first person to describe the neuroendocrine responses of the body to stress. He suggested that when individuals were submitted to stressful situations, they activated the sympathetic nervous system (SNS) and HHAa triggered the so-called “general adaptation syndrome” [13].

Activation of HHAa is initiated in the animal in response to changes in temperature that are outside of the thermal comfort zone of the species. Heat stress causes activation of hypothalamic -pituitary –adrenal axis and consequent increase in plasma glucocorticoid concentrations. Both short and long term heat stress affect endocrine status of animals and cause release of hormones mainly:

- Thyroxin
- Cortisol
- Growth hormone
- Catecholamine{Prolonged environmental stress}

The plasma levels of these hormones act as potential indicators of physiological adjustements taking place in the body of heat exposed animals.

Those changes are initially perceived by the peripheral nervous system and then assimilated by the central nervous system, wherein the paraventricular nucleus of the hypothalamus is stimulated to release all releasing hormones.

1. Growth hormone

There have been many consistent findings that hyperthermia induces a rapid rise in circulating growth hormone (GH) levels during heat exposure and exercise in humans. More severe heat stress (41°C, 3h) increased the plasma GH (eightfold, peaked at 45 min), epinephrine (E) (twofold, biphasically at 15 and 180min), and norepinephrine (NE) (80%) levels with a 1.2°C rise in men. The Teore threshold for the release of GH appears to be lower than that for catecholamine or cortisol.

2. Thyroid Gland Hormones

The thyroid gland produces triiodothyronine (T3) and tetraiodothyronine or thyroxine (T4) in response to stimulation by TSH produced by thyrotropic cells in the anterior hypophysis. Thyroid hormones control cellular metabolism that favors oxygen consumption and energy generation needed for tissue activities. Oxygen consumption in cells is related to increase in mitochondrial activity and generation of heat. High levels of thyroid hormones increase cellular respiration, ATP generation, cellular growth, cardiac and respiratory rates and catabolic pathways [14]. In cases of thermal stress, secretion of releasing and tropic hormones is affected. When an animal is subjected to high ambient temperatures, secretion of those hormones is inhibited in order to avoid thermogenesis. On the other hand, when an animal is subjected to ambient cold, those hormones are released to promote catabolic pathways that favor body thermogenesis. In this case, secretion of T3 and T4 is stimulated. When stress is chronic, physiologic functions of T3 are decreased due to high levels of glucocorticoids that inhibit transformation of T4 in T3.

3. Adrenal gland hormone

a. Cortisol

Corticotropic releasing hormone reaches anterior pituitary via hypothalamus-hypophyseal portal system and activates it for the synthesis and secretion of ACTH into the blood stream. ACTH stimulates the zona fasciculata of the adrenal cortex to synthesize and secrete cortisol into blood to exert its physiological actions in target tissues (muscle, liver and adipose tissue) [15]. Cortisol is a member of the steroid hormone family whose common precursor is cholesterol. Due to its low solubility in blood it is transported by special proteins (corticosteroid binding globulin [CBG] to target tissues and only 10% of cortisol is in free form. It has been demonstrated that animals submitted to stress (noise, physical perturbation or changes in ambient temperature) have an increase in circulating concentrations of CBG and free cortisol. This finding shows that the secretion of cortisol is one of the most important hormonal responses to stress [16]. Cortisol mainly plays a role during heat stress by mobilization of energy for maintenance of muscular and neural functions, directly influencing metabolism and behavior of animals exposed to thermal stress. It helps to restores compromised energy homeostasis by stimulating glycogenolysis, lipolysis and proteolysis in the stressed animal. High concentrations of

cortisol in serum are associated with an increase in aggressive behavior in some animals [17].

b. Catecholamines (Epinephrine and Norepinephrine)

The concentration of catecholamine is elevated during both acute and chronic thermal stress [18]. An average increase of 45 and 42% in short term and 91 and 70% in long term heat exposure for epinephrine and norepinephrine respectively.

4. Antidiuretic hormone (ADH)

This is a hormone synthesized and secreted by supraoptic and paraventricular hypothalamic nuclei into the blood stream and transported to target tissues. It is also known as vasopressin due to its regulatory effect on blood pressure. ADH is the most important hormone controlling water homeostasis, stimulating kidneys to reabsorb more water during heat stress events. The pattern of ADH secretion during heat stress events varies in accordance with the type and nature of thermal stress, acute vs. chronic heat stress [19]. In heat stress, thermolytic routes get activated for heat dissipiation via sweating and evaporation. Baroreceptors in the atrium and greater blood vessels, as well as hypothalamic osmoreceptors get stimulated by constant fluctuations in body fluids during heat stress, which further induce ADH release to prevent dehydration. Whereas during cold stress polyurea is generated by inhibition of ADH which favors water loss by urination to avoid heat transference from tissues into water.

5. Renin-angiotensin-aldosterone system (RAA)

These hormones are related to the maintenance of homeostasis for electrolytes. RAA is activated when blood flow is reduced in the afferent arteriole of glomeruli of the kidney due to hypovolemia. Hypovolemia can result from dehydration as a consequence of high ambient heat. The decrease in blood flow to the kidney induces secretion of renin from the juxtaglomerular apparatus. Once renin is produced, it stimulates the synthesis and secretion of angiotensin, which induces the synthesis and secretion of aldosterone from the adrenal cortex. Aldosterone stimulates reabsorption of water and ions, principally sodium, in the kidney in order to avoid massive excretion of water and to maintain blood pressure. Exposure to cold stress causes inhibition of sensitivity of the juxtaglomerular apparatus, favoring water excretion (polyuria) [20]. Recent studies suggest that other hormones, like progesterone, insulin, oxytocin, androgens and estrogens, play a role in responses to thermal stress via undefined physiological mechanisms [13].

6. Insulin

Plasma insulin decreases on exposure to elevated temperature in cattle. Heat stress decrease feed intake. Reduction in DMI of heat stressed animals results in a negative energy balance, that may prolong the post partum period and decrease fertility in dairy cows.

7. Insulin like growth hormone (IGF-1)

The concentration of IGF-1 has been found to decrease during summer months. IGF-1 is associated with poor reproductive performance of cows during hot summer. The effect may be mediated through reduced feed intake as cows in negative energy balance have lower IGF-1[21].

8. Adiponectin and Leptin

In transition cows leptin plays a critical role in regulating energy metabolism. Nutritional changes induced by heat stress may influence leptin [22].

9. Reproductive hormone

In heat stress there is an activation of the thermoregulatory center in the central nervous system, with the activation of compensatory mechanisms by thermolysis (peripheral vasodilatation, increased respiratory rate, decreased food intake, increased water intake), inducing the animal to lose heat through sensitivity (conduction, convection and radiation) [23]. The efficiency of latent heat dissipation, depends on the function of the sweat glands.

The heat stress directly affects the reproductive performance of animals through the hypothalamic-pituitary-gonadal axis by inhibiting gonadotropin-releasing hormone (GnRH) in the hypothalamus. In the anterior hypophysis interferes with the release of follicle stimulating hormone (FSH) and luteinizing hormone (LH) and in the gonads changes the stimulating effect of gonadotropin secretion of sex steroids. Consequently, the reproductive efficiency is affected, since spermatogenesis is controlled by the neuroendocrine system and is influenced by the mechanism of the scrotum testicular thermoregulation.

In females the activation of the hypothalamic-pituitary-adrenal (HHA) results in decreased secretion of gonadotropins (LH and FSH), in the production of estrogen, which consequently leads to various reproductive disorders such as failure to detect estrus (silent estrus), failures in oocyte development, in fertilization and insemination. The Embryo mortality is because of excretion of CRH which acts on GnRH inhibiting the release of LH and causing consequently fails in the oocyte release, changes in the endometrial cells and loss in pregnancy.

a) FSH and LH

The release of LH and FSH from the anterior pituitory gland is disrupted by heat stress. In summer the dominant follicle develops in a low LH environment and these results in reduced oestradiol secreation from the dominant follicle leading to poor expression of oestus and low fertility.

Reduction in plasma inhibin concentrations occurs in heat-stressed cows and cyclic buffaloes, specially during summer months, due to decline in folliculogenesis, which significantly affects growth of small and medium size follicles from which major proportion of plasma inhibin comes from. Increased concentration of FSH in heat may be due to decreased plasma inhibin production by compromised follicles as inhibin is an important factor in the regulation of FSH secretion.

b) Progesterone

The heat stress had no effect on plasma progesterone levels in lactating cows and dairy heifers during the second half of the oestrous cycle exposed to heat in a climatic chamber and luteolysis was delayed. [24]. reported that plasma progesterone concentration were lower in heat stressed cows compared to thermoneutral counterparts. Chronic heat stress possibly impairs follicle and corpus luteum and luteinised theca cells and more susceptible to heat stress than luteinised granulosa cells.

c) Gonadotrophins and Corticosteroids

Heat stress affects the hypothalamic pituitary axis and the sympathoadrenal system to initial and modulate most of the activities [25].

d) Prostaglandin

Heat stress affects reproductive performance of animals by negatively affecting uterine environment resulting in decreased blood flow to the uterus with corresponding increase in uterine temperature, which may lead to implantation failure and embryonic mortality. The production of heat shock proteins by the endometrium during heat stress and reduced production of interferon tau by the conceptus may contribute to these effects.

e) Androstenedione

Heat exposure induced impairment of LH receptor function as reflected in significant decline in LH –stimulated androstenedione production by thecal cells in the ovary [26].

f) Prolactin

Circulating prolactin levels are observed to increase during thermal stress in mammals [27]. Reduced nutrient intake decreases circulating prolactin concentration in ruminants and direct effect of heat stress on serum prolactin levels also been observed. [24].

10. Pineal gland hormon

Melatonin is mainly produced by the pineal gland at night or the dark phase of the light-dark cycle. Pineal gland is considered as a neuroendocrine transducer of cyclic photic input, which is responsible for the seasonal changes in reproductive capability of various species. Pineal gland is known to have anti-stressogenic effect in mammals and birds. Glucocorticoids could have stimulated the pinealocytes to release melatonin in these animals in order to relieve thermal-stress. Many biochemical reactions increase with high temperature; it is, thus, very likely that elevated body temperature, in heat stress conditions, would enhance the generation of free radicals via accelerated metabolic reactions in cells and tissues. Increased generation of oxygen-free radicals and cytotoxicity of oxidants may mediate in part, heat-induced cellular damage through the lipid peroxidation. In contrast, melatonin, a pineal gland-derived hormone, plays an important role in the antioxidant defense system and has an effective free-radical-scavenging activity. This additional mechanism has been effective in proving thermotolerance to the melatonin treated chickens. Thermal stress melatonin was able to successfully modify the adrenal cortex functions to relieve thermal stress. Exogenous melatonin further reduced cortisol level.

5. Cold stress

All mammals are warm blooded and need to maintain a constant core body temperature. Animals experience cold stress when the temperature falls, below the lower limit of thermoneutral zone, called the "lower critical temperature". Increase in metabolic activity of body to generate more heat, is the first reaction of body to combat the effects of cold stress. When environmental temperature decreases below the thermal comfort level, heat loss must equal endogenous heat production (thermogenesis) in order to achieve homeostasis, i.e. maintenance of body temperature in a stable thermo neutral range [28]. Cold stress activates several physiological responses.

In the arid and semi-arid region of India, sheep are generally exposed to various climatic extremes like elevated temperature, feed and water scarcity during grazing [29, 30]. In these regions of India, most of the lambs are born in January and February, and were exposed to cold stress. New born lambs are more likely to be at risk from cold exposure and the lamb mortality by hypothermia is the main cause of economic loss in most of the sheep production systems. During early post-natal life, homeotherms undergo marked developmental changes to control their body temperature. Low survival rate because of cold stress is one of the main factors that adversely affect lamb production and resulting into sizeable economic losses in sheep farming.

6. Strategies for adapting cold stress

A. Activation of neuroendocrine response

Cold stress in vertebrates may activate the neuroendocrine pathway and cause the release of regulation hormones such as glucocorticoid and insulin related to energy metabolism. The metabolic themogenesis favoured by an increase in T3 secretion occurs during cold stress, due to acute secretion of catecholamines and activation of HHAa, similar to heat stress. Also the concentrations of adrenaline, noradrenaline, β-endorphin and cortisol are shown to increase in plasma in response to exercise in cool dry conditions in horses. Apart from fluctuations in the physiological and biochemical profiles, T3 and T4 also play a major role in thermogenesis. As energy intake is used mainly for thermoregulation, it is possible to observe a depression in body weight gain and an increase in mortality [31].

B. Inhibition of thermoregulatory hypothalamic center

This minimizes heat loss by evapotranspiration. In cold stress, sensitivity of the juxtaglomerular apparatus is inhibited, favoring water excretion (polyuria). Although livestock experience many stressors throughout the production cycle, one of the most commonly experienced, and most difficult to control, is stress caused by fluctuations in environmental temperatures that extend beyond thermoneutrality for an animal. Cold stress and infectious disease are suspected to contribute to the 13% to 15% mortality rate reported for piglets between farrowing and weaning. In addition, losses because of extreme changes in environmental temperature [heat stress (HS)] result in significant losses throughout the livestock industry every year.

C. Change in endocrine profile

Inhibition of ADH favors water loss by urination to avoid heat transference from tissue to water, generating polyurea [19]**.** It has been reported that low-temperature stress activates the hypothalamic-pituitary-adrenal axis and causes the release of glucocorticoid hormone in vertebrates. Cold exposure leads animals to change carbohydrate metabolism, which is modulated by hormones such as insulin and glucagon. There are marked interactions between glucocorticoid and insulin on most aspects of metabolism, and these interactions serve as a peripheral hormonal feedback loop that regulates the energy metabolism system**.** Cortisol enhances gluconeogenesis, promotes the breakdown of lipids and proteins, and mobilization of extrahepatic amino acids and ketone bodies, thus counteracting the effects of insulin. Cortisol does cause serum glucose to rise. The secretion of LH and FSH was lower in the cool environment, suggesting that, as in adult animals, adverse environmental conditions may reduce reproductive hormone secretion in the neonate. Thus, the secretion of pituitary LH and FSH may serve as a biochemical indicator of stress.

7. Conclusion

Mitigating the negative effects of climate change remains a

major concern to maintain the economic viability and enhance profitability of the livestock sector. The biological mechanism by which heat and cod stress impact animal production and reproduction is both direct and indirect. The decrease in feed intake and also because of an altered endocrine status reduction in nutrient absorption and increased maintenance requirements result in a net decrease in nutrient /energy availability. Heat stress acclimation is accomplished by changes in homeostatic responses and may include homeorhetic processes involving an altered endocrine status that ultimately affects target tissue responsiveness to environmental stimuli.

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