MEDDOCS Open Access Publisher

ISSN: 2640-1223

Journal of Veterinary Medicine and Animal Sciences

Open Access | Review Article

Review on Hormonal Metabolic Adaptations of Farm Animals

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Received: Aug 21, 2020

Accepted: Oct 13, 2020

Published Online: Oct 16, 2020

Journal: Journal of Veterinary Medicine and Animal Sciences

Publisher: MedDocs Publishers LLC

Online edition: http://meddocsonline.org/

Keywords: Adaptation; Farm animals; Metabolism; Hormones.

Abstract

The general aim of this review is to summarize the roles of major hormones in the metabolic adaptations of farm animals. Farm animals maintain their physiological states by release of common hormones such as thyroxin, cortisol, leptin and catecholamine. These hormones influence the effects of the hypothalamus, adenohypophysis, pancreas, kidney, liver and thyroid hormones. These actions involve pathways that regulate appetite and energy expenditure as well as the secretion of metabolic hormones and regulate metabolic activities. They also regulate the body homeostasis. Heat and cold stresses have both direct and indirect impact on animal production and reproduction. Heat stress causes activation of hypothalamic-pituitary-adrenal axis and consequent increase in plasma glucocorticoid (cortisol) concentrations. Both short and long term heat stress affects the endocrine status of animals and cause release of hormones mainly: thyroxin, cortisol, leptin and catecholamine. The plasma levels of these hormones act as potential indicators of physiological adjustments taking place in the body of heat stressed animals. On the other hand, 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 the environmental temperature decreases below the thermal comfort level, heat loss must equal endogenous heat production (thermogenesis) to achieve homeostasis, mitigating the negative effects of climate change remains a major concern to maintain the economic viability and enhance profitability of the livestock sector. Hence, these hormones play significant roles for the metabolic adaptation of farm animals.

Cite this article: Getabalew M, Alemneh T, Zewdie D. Review on Hormonal Metabolic Adaptations of Farm Animals. J Vet Med Animal Sci. 2020; 3(1): 1037.

Introduction

The body systems, which are mostly involved in the process of adaptation to the environment are an endocrine system for long-standing responses and nervous system for sensory inputs and short term responses. When an environmental pressure exceeds to that which animal's adaptive mechanisms can provide somewhere to stay, the stress-related disease occurs. Many theories have been postulated to explain the organism's physiological response to stress [1] proposed the general adaptation syndrome which provided the first comprehensive biological theory of stress. He proposed three stages that constituted alarm, resistance and exhaustion. When the threat or stressor is identified or realized, the body's stress response is a state of alarm. In this stage that animals react by fighting or fleeing. If the stressor persists, the body tries to adapt to the strains or demands of the environment. Exhaustion is the third and final stage when all of the body's resources are eventually depleted and the body is unable to maintain normal function. If stage three is extended, the immune system is exhausted resulting in the development of the psychosomatic disease, immune suppression, reduced efficiency of production and reproduction. It affects ability to perform and make animals susceptible to physio-pathological disorders [2]. There are several 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 the normal estrous cycle of animals [1,2].

The biological mechanism by which heat and cold 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 [1]. Heat stress acclimation is accomplished by changes in homeostatic responses and may include homeostatic processes involving an altered endocrine status that ultimately affects target tissue responsiveness to environmental stimuli [1,2].

It has been reported that low-temperature stress activates the hypothalamic-pituitary-adrenal axis and causes the release of glucocorticoid hormone (cortisol) in vertebrates. Cold exposure leads animals to change carbohydrate metabolism, which is modulated by hormones such as insulin [3]. 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. Steroid hormones are dissociated from their receptors and metabolized by the target cell or the liver, which possess enzymes capable of altering the specific steroids and rendering them biologically inactive and water soluble [4]. Typically, inactivation involves reduction or removal of side-chain or attached groups or both, as well as the combination with other molecules (conjugation), such as glucose, to form a glucuronide or conjugation with sulfate. The relative emphasis on sulfate or glucuronide varies depending on the steroid and/or species [3,4]. The general objective of this review is to assess the role of the major hormone in metabolic adaptation of farm animals.

Hormonal indicators

During stress, various endocrinal responses are involved to overcome stressful situations. The immediate endocrinal responses are the sympathy-adrenomedullary system releasing catecholamine. The main biological stress responses are related to the HPA axis releasing corticosteroids like cortisol, corticosterone, and aldosterone into the blood [3]. Corticotropin-Releasing Hormone (CRH), which acts on the anterior pituitary to synthesize and release ACTH, which in turn is released into the peripheral circulation to cause the release of glucocorticoids from the adrenal cortex. Plasma ACTH provides the direct determination of stress whereas cortisol provides the indirect criterion of stress. Besides plasma, salivary and urinary cortisol levels are good indicators of stress [4].

The disadvantage is that plasma cortisol levels naturally increase in the morning and decreases around midnight [5]. Another disadvantage is that all types of stressors do not induce an increase in cortisol levels. The complexity of diurnal activity, the natural increase in cortisol, individual variability suggests the use of other markers as well to evaluate the stress responses [5,6]. Fecal glucocorticoid analyses have been used in a wide range of studies as this is a non-invasive measure of these stress hormones. Salivary α-amylase has also been used as marker of stress [6].

Metabolic and hormonal acclimation to hot environment

The biological mechanism by which heat stress impacts production and reproduction is partly explained by reduced feed intake, but also includes an altered endocrine status, reduction in rumination and nutrient absorption, and increased maintenance requirements, resulting in a net decrease in nutrient/energy availability [7]. Naturally, a reduction in energy intake combined with increased energy expenditure for maintenance lowers energy balance and partially explains why lactating cattle lose significant amounts of body weight during severe heat stress [8,9].

Biological consequences of hormonal and metabolic acclimation

Animals start many acute acclimation responses to thermal stress [10]. But some of these adaptations may ultimately adversely affect long-term health and/or productivity. In the sections below, we will address how physiological and metabolic adjustments affect health variables and production parameters. Hence it is assumed that high ambient temperatures, directly and indirectly, affect the health status of farm animals. Direct influences include temperature-related illness and death [11].

Indirect influences include those derived from reduced nutrient intake, altered microbial populations around and in the animal, redistribution of vector-borne diseases, decreased host resistance to infections, water shortages and food-borne diseases. A series of studies have described a higher risk of mortality during the hottest months [12] and an increased death rate during extreme weather events [13]. High temperatures may cause heatstroke, heat exhaustion, heat syncope, heat cramps and ultimately organ dysfunction and these heat-induced complications occur when the body temperature raises approximately 38°C to 48°C. Our recent epidemiological dairy cow study indicates that 80 and 70 are the daily maximum and minimum THI values, respectively, above which heat-induced death rate increases [14].

The thyroid gland produces triiodothyronine (T_3) and tetraiodothyronine or thyroxin (T_4) in response to stimulation by TSH produced by thyrotrophic cells in the anterior hypophysis. Thyroid hormones control cellular metabolism that favours oxygen consumption and energy generation needed for tissue activities [14]. Oxygen consumption in cells is related to an 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 in cases of thermal stress secretion of releasing and tropic hormones are affected [14,15]. When an animal is subjected to high ambient temperatures, the 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 favour body thermogenesis. In this case, secretion of T_3 and T_4 is stimulated. When stress is chronic, physiologic functions of T_3 are decreased due to high levels of glucocorticoids that inhibit the transformation of T_4 to T_3 [15].

Adrenal gland hormone

Cortisol

Corticotropic releasing hormone reaches anterior pituitary via the hypothalamus-hypophyseal portal system and activates it for the synthesis and secretion of Adrenocorticotropic Hormone (ACTH) into the bloodstream. ACTH stimulates the zona fasciculata of the adrenal cortex to synthesize and secrete cortisol into blood to exert its physiological actions in target tissues like muscle, liver and adipose tissue [14]. 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 the metabolism and behavior of animals exposed to thermal stress. It helps to restore compromised energy homeostasis by stimulating glycogenolysis, lipolysis and proteolysis in the stressed animal [16]. High concentrations of cortisol in serum are associated with an increase in aggressive behavior in some animals [17].

Leptin

Leptin is produced in the adipose tissue and other organs. Its main function is to maintain glucose homeostasis indicating the status of the reserve of energy to ventromedial nucleus of the hypothalamus; target of leptin which regulates eating behavior, so this is considered a sensor of energy balance. This hormone is involved in the onset of puberty and acts as a critical hormonal signal of nutritional status in the neuroendocrine regulation of pulsatile secretion of Growth Hormone (GH) and release of Gonadotropin-Releasing factor (GHRH) mediated by Neuropeptide Y (NPY) [18]. This hormone is involved in the onset of puberty and acts as a critical hormonal signal of nutritional status in the neuroendocrine regulation of pulsatile secretion of GH and release of GHRH mediated by NPY [17]. Leptin also controls the hypothalamic-hypophysis-gonadal axis through synthesis and release of hypothalamic and hypophyseal gonadotrophins (GnRH, FSH and LH) and potentiates the effects of insulin through which regulates the synthesis of blood glucose. The regulation of secretion of this hormone is at long term and depends on the variation in body mass and stimulating effects of insulin because of this, it has great importance in the transition period in dairy cattle in which during the time of drying the body condition and fat reserves are recovered for postpartum performance. The cattle breed tically determines the amount of body fat and leptin secretion which in turn determines the quality of the carcass [18].

Stress as a physiological mechanism

The find of metabolic, immunological and neuroendocrine mechanisms make it possible to describe the stress reaction in physiological terms [19]. A huge number of hormones like prolactin hormones are involved in the stress response [20]. The adrenal glands have a key-role in hormonal reactions to stress as they are involved both in the hypothalamic-pituitary adrenocortical axis and the symphatho-adrenomedullary system [4]. Difficult situations produce responses of the adrenals, which increase glucocorticoid and/or catecholamine secretion. These increases are the front-line endocrine mechanisms to defend the organism against stressful conditions. As a physiological mechanism, stress *per se* is not inherently bad [4]. For example, glucocorticoids are released in response to situations that are not normally regarded as stressful, including courtship, copulation and hunting [22].

In addition hormones, which increase during stress periods, are also part of the hormonal cascade causing parturition in some species [23]. During short-term stress, glucocorticoids improve fitness by energy mobilization and may change behavior [24]. However, severe chronic stress (prolonged periods of high cortisol concentrations) may decrease individual fitness by immune suppression and atrophy of tissues. Besides, the reproductive efficiency of the animals decreases [25,26]. There are also indications that stereo types might be related to stress. As data concerning the metabolism and the excretion of catecholamine are almost lacking so far in farm animals and relatively few studies have reported concentrations in sample materials other than the urine we have concentrated on glucocorticoids in feces. However, more investigations concerning these basic questions should be fostered to enable an evaluation of both “stress axes” in the future [27].

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 [28]. 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 [7]. In this way, many productive and reproductive parameters can be compromised, like nutrient intake, weight gain, milk production and fertility [29]. 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 redistributing 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 [30].

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 thermo neutral 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 the environmental temperature decreases below the thermal comfort level, heat loss must equal endogenous heat production (thermo genesis) to achieve homeostasis, i.e. maintenance of body temperature in a stable thermo neutral range [31].

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 [11]. In these regions of India, most of the lambs are born in January and February, and were exposed to cold stress. Newborn 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 [32].

Strategies for adapting cold stress

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 [31]. The metabolic thermogenesis is favoured by an increase in T3 secretion during cold stress, due to acute secretion of catecholamine and activation of HHA axis, similar to heat stress. Also the concentrations of adrenaline, noradrenaline, β-endorphin and cortisol are increased 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 thermo genesis. As energy intake is used mainly for thermoregulation, it is possible to observe a depression in body weight gain and an increase in mortality [32].

Inhibition of thermoregulatory hypothalamic center

This minimizes heat loss by evapo-transpiration. In cold stress, sensitivity of the juxtaglomerular apparatus (JGA) is inhibited, favouring 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 thermo neutrality 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 result in significant losses throughout the livestock industry every year [33].

Conclusions

In conclusion, farm animals maintain their physiological states by release of hormones; namely, thyroxin, cortisol, leptin and catecholamine. These hormones released when the body systems need adjustments to the environmental tempratures (homeostasis), to regulate appetite and energy expenditure, and to secret metabolic hormones.

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