

Effect of Stress on Animal Health: A Review

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Abstract

Stress, as it relates to bodily functions, has been defined as the sum of all biologic reactions to physical, emotional, or mental stimuli that disturb an individual's homeostasis. Stressor can be defined as any internal or external stimuli or threat that disrupts homeostasis of the body, and elicits a coordinated physiological response within the body in an attempt to reestablish homeostasis. The different stressors that enhance stress it can be chemical stressors: poor water quality low dissolved oxygen, improper pH, Pollution intentional pollution: chemical treatments, accidental pollution: insect spray, Diet composition - type of protein, amino acids, Nitrogenous and other metabolic wastes accumulation of ammonia or nitrite. Biological stressors: population density crowding, mixing of different species of animal aggression, territoriality, space requirements, Microorganisms pathogenic and nonpathogenic, Macroorganisms internal and external parasites. Physical stressors: Light, sounds, dissolved gases and temperature. The stressor can be any internal or external stimuli or threat that disrupts homeostasis of the body, and elicits a coordinated physiological response within the body in an attempt to reestablish homeostasis. The different stressors that enhance stress and disturb the normal physiological functions of the different organs of animal health in different ways. Finally stress increased incidence of diseases and suffering animals as well as if the stress level is chronic it leads to death.

Keywords: Health, Stressor and stress

1. Introduction

Stress is a broad term, generally used in negative connotation and is described as the cumulative detrimental effect of a variety of factors on the health and performance of animals. Stress is a condition in which an animal is unable to maintain a normal physiologic state because of various factors adversely affecting its well being. The term "stress" has been widely used in biology to describe a set of physiological and behavioural changes elicited by aversive stimuli (Agarwal and Prabhakaran, 2005). Cannon (1929) described stress as the sympatho adreno medullary (SAM) system's attempt to regulate homeostasis when threatened by a variety of aversive stimuli or stressors. Stress, as it relates to bodily functions, has been defined as the sum of all biologic reactions to physical, emotional, or mental stimuli that disturb an individual's homeostasis. Therefore, a stressor can be defined as any internal or external stimuli or threat that disrupts homeostasis of the body, and elicits a coordinated physiological response within the body in an attempt to reestablish homeostasis. A stressor is a chemical or biological agent, environmental condition, external stimulus or an event that causes stress to an organism and an event that triggers the stress response. The different stressors that enhance stress it can be chemical stressors: poor water quality - low dissolved oxygen, improper pH, Pollution - intentional pollution: chemical treatments, accidental pollution: insect spray, Diet composition - type of protein, amino acids, Nitrogenous and other metabolic wastes accumulation of ammonia or nitrite. Biological stressors: population density - crowding, mixing of different species of animal- aggression, territoriality, space requirements, Microorganisms - pathogenic and nonpathogenic, Macroorganisms - internal and external parasites. Physical stressors: Light, sounds, dissolved gases and temperature. Temperature is one of the most important influences on the immune system of animals and Procedural stressors are handling, shipping and disease treatments (Altan *et al.*, 2003).

Later on, Selye (1936) conducted some of his classic studies on the response of the hypothalamic-pituitary-adrenal (HPA) axis to noxious stimuli and suggested that the organism reacted in a non-specific manner to a wide variety of aversive stimuli, mainly with an increase in the HPA axis activity. Stresses broadly categorize Environmental (thermal), Physiologic (nutritional deficiency) Psychological/psychosocial i.e. overcrowding. Stressors can be conveniently divided into physical stressors, social stressors resulting from the interactions with individuals of the same species and stressors related to handling by humans. Stressors have additive effects. This means that when several stressors impinge upon the animal at the same time, the resulting stress response will be much higher than if the animal was exposed to one stressor only. Therefore, circumstances such as weaning and transport can be particularly difficult for the animals (Nienaber and Hahn, 2007). The stress response includes several changes that may have negative effects on the performance of farm animals. These effects include changes in the immune function and increased susceptibility to disease, decreased feed intake and rumination, inhibition of oxytocin release, and reduced fertility, among others (Nardone *et al.*, 2010).

2. Different Stress Effects on Animal Health

2.1. Effect of Stress on Immune Function and Susceptibility to Disease

It has long been observed that an association exists between stress and disease susceptibility in domestic farm animals, although a definitive causal factor has yet to be defined. Many researchers have implicated a suppression of the host's immune system by stress that allows opportunistic pathogens to invade. Furthermore, substantial evidence has suggested that this immune suppression is mediated by glucocorticoid following hypothalamic pituitary adreno cortical (HPA) axis activation by a stressor (Griffin, 1989).

Stress can suppress immune function. However, the ways in which chronic stress suppresses the immune system are highly specific, and only some types of defense against disease are affected. When the stress response involves the release of glucocorticoid or catecholamines, the capacity of cellular immune mechanisms is reduced. In practical terms, this means that some disorders are more likely to be precipitated by chronic stress than others. These include respiratory infectious diseases and *Salmonella* sp. infection. For example, transport stress has been shown to increase pneumonia caused by bovine herpes virus-1 in calves, in beef cattle and exported animals. Pneumonia caused by *Pasteurella* sp. and result mortality in calves and sheep, and salmonellosis in sheep and horses (Mack *et al.* 2013)

Susceptibility to other diseases can also be increased as a result of situations which are likely to be stressful. For example, several studies have shown an increase in the prevalence of mastitis in dairy cows as a result of chronic fear. Although the precise mechanism explaining this effect is not know, it has been suggested that the function of the natural-killer cells could be impaired as a result of stress and this in turn could lead to an increased susceptibility of the mammary gland to infectious agents. The stress of weaning is known to increase the risk of digestive disease in several species (McDaniel *et al.*, 2004).

Cell mediated immunity since stressors have been associated with increased circulatory concentration of glucocorticoid; they also have been linked with decreased functioning of the cells of the immune system. Blecha *et al.* (1984) found that when cattle were exposed to stressful conditions, lymphocyte proliferative responses to concanavalin A (Con A) were reduced. High ambient temperature causes functional and metabolic alterations in cells and tissues including cells of immune system. The immune cell functions are associated with production of ROS such as that involved in the microbial activity of phagocytes or lympho proliferative response to mitogens (Bhar *et al.*, 1997). However excessive production of ROS due to heat stress renders harmful effect on cells of immune system. When an animal exposed to oxidative stress, polymorpho nuclear leukocytes (PMNs) change their pattern of oxygen uptake sharply while releasing large amounts of superoxide anion into the cell environment. PMNs play an important role as mediators of tissue destructive events in inflammatory diseases, ranging from rheumatoid arthritis and myocardial reperfusion injury to respiratory distress syndrome (Fedde, 1998). Heat stress reduced serum IgG1 in calves associated with an increased Cortisol concentration and extreme cold stress also reduced colostral immunoglobulin transfer. Thus, environmental extremes can influence disease resistance in dairy calves.

2.2. Effect of Stress on Metabolic Changes

High ambient temperature can adversely affect the structure and physiology of cells causing impaired transcription, RNA processing, translation, oxidative metabolism, membrane structure and function (Mashaly *et al.*, 2004). Cells generate small amounts of free radicals or reactive oxygen species (ROS) during their normal metabolism. Although low levels of ROS are essential in many biochemical processes, accumulation of ROS may damage biological macromolecules i.e. lipids, proteins, carbohydrates and DNA (Sandercock, *et al.*, 2001). External factors such as heat, trauma, ultrasound, infections, radiations, toxins etc. can lead to increased free radicals and other ROS and may lead to oxidative stress. Therefore heat stress increased lipid per oxidation which was associated with production of large number of free radicals which are capable of initiating per oxidation of polyunsaturated fatty acids. Renaudeau *et al.* (2012) also reported that lipid per oxidation is significantly increased during reticulo ruminal impaction in buffaloes. Heat stress may lead to increased production of transition metal ions (TMI), which can make electron donations to oxygen forming superoxide or H₂O₂ which is further reduced to an extremely reactive OH radical causing oxidative stress. Antioxidants, both enzymatic (viz. superoxide dismutase, glutathione peroxidase & catalase) and no enzymatic (vitamins C, E and A, glutathione, pyruvate etc) provide necessary defence against oxidative stress generated due to high ambient temperature. Catalase detoxifies H₂O₂ produced during different metabolic processes and also in stressful conditions by reducing it to H₂O and O₂ (Selye, 1976). Superoxide dismutase (SOD) in conjugation with catalase and glutathione peroxidase (GPx) scavenges both intracellular and extracellular superoxide radicals and prevents lipid per oxidation. GPx reacts with peroxides and requires glutathione (GSH) as the reductive substance donating an electron. GSH reduces oxygen toxicity by preventing O₂ formation. Lyte (2004) reported that catalase activity is reduced in oxydementon methyl induced oxidative stress in buffaloes. Lyte (2004) also reported similar findings in molybdenum induced oxidative stress in crossbred calves. Heat stress in lactating

animals' results in dramatic reduction in roughage intake, gut motility and rumination which in turn contribute to decreased volatile fatty acid production and may contribute to alteration in acetate: propionate ratio. Rumen pH also declines during thermal stress (Collier *et al.*, 1982). Electrolyte concentrations, in particular Na⁺ and K⁺ are reduced in rumen fluid of heat stressed cattle. The decrease in Na⁺ and K⁺ are related to increase in loss of urinary Na⁺ and loss of skin K⁺ as well as decline in plasma aldosterone and increase in plasma prolactin (Collier *et al.*, 1982). Enhanced heat dissipation during heat stress may also lead to electrolyte losses through sweat, saliva, polypnea and urine. This may lead to fall in plasma Na⁺, K⁺ and Cl⁻ concentration (Coppock *et al.*, 1982). Dale and Brody (1954) reported that heat stress in lactating dairy cows caused significant loss of serum Na⁺ and K⁺ and fall in serum electrolyte concentration in dairy cows subjected to heat stress. Dale and Brody, (1954) suggested that a heat stressed animal, particularly a lactating cow, might experience metabolic ketosis as energy input would not satisfy energy need and thus accelerate body fat catabolism accumulating ketone bodies if they are not rapidly excreted. These ketone bodies deplete blood alkali reserves, possibly potentiating respiratory alkalosis. Thermal stress alters dietary protein utilization and body protein metabolism.

2.3. Effect of Stress on Hormonal Changes

It has been recognized that certain environmental stressors have the potential to activate the hypothalamus-pituitary-adrenal cortical axis (HPA) and sympatho-adrenal medullary axis (Elnagar *et al.*, 2010). There is increase in plasma concentration of Cortisol and corticosterone and less frequently an increase in plasma epinephrine and nor epinephrine concentration in heat stressed animals (Elnagar *et al.*, 2010). Collier *et al.* (1982) reported that thermal stress reduced birth weights of Holstein calves. Reduced birth weight of calves was associated with lower concentrations of estrone sulfate in plasma of heat stressed animals. Because estrone sulfate is produced by the gravid uterus and conceptus, its reduction indicates reduced conceptus function during thermal stress. Concentration of progesterone in plasma was also reported to elevate in heat stressed cycling cows by the same team workers. During short term exposure to high ambient temperature, the concentrations of glucocorticoid and catecholamines were found to be elevated (Novero *et al.*, 1991). Phillips and Santurtun, (2013) reported that the simultaneous relationship among thermal stress, plasma aldosterone level and urine electrolyte concentration in bovines. During prolonged heat exposure plasma aldosterone level was reported to decline. Concurrent with this, there were significant fall in serum and urinary K⁺. Robertshaw, (1985) also suggested that a fall in serum K⁺ depressed aldosterone secretion, which may also have reduced urinary K⁺ excretion. Sparke *et al.* (2001) reported an increase in plasma prolactin concentration during thermal stress in dairy cows. Alteration in prolactin secretion may be associated with altered metabolic state of heat stressed animals. One possibility is that prolactin is involved in meeting increased water and electrolyte frequently demands of heat stressed animals.

2.4. Effect of Stress on Feed Intake and Rumination

The negative effect of stress on feed intake has long been recognized, although the precise pathways involved are still debated. It is likely, however, that the inhibitory effect of stress on appetite results from a complex interplay among leptin, glucocorticoid and the CRF. There is some evidence suggesting that stress may have an inhibitory effect on rumination and this in turn may reduce feed digestibility and therefore performance, and may also increase the risk of ruminal acidosis. The precise mechanism underlying the effect of stress on rumination is not known, but it is interesting to highlight that brain activity during rumination is similar to that during sleep, and stress is known to interfere with sleep. Animals exposed for nutritional stress would result depressed appetite, altered body fat ratio, weight loss, nutrient deficiency and eating disorders. Heat stress has long been known to adversely affect rumen health. One way cows dissipate heat is via panting and this increased respiration rate results in enhanced CO₂ (carbon dioxide) being exhaled. In order to be an effective blood pH buffering system, the body needs to maintain a 20:1 HCO₃⁻ (bicarbonate) to CO₂ ratio. Due to the hyperventilation induced decrease in blood CO₂, the kidney secretes HCO₃⁻ to maintain this ratio. This reduces the amount of HCO₃⁻ that can be used (via saliva) to buffer and maintain a healthy rumen pH. In addition, panting cows drool and drooling reduces the quantity of saliva that would have normally been deposited in the rumen. Furthermore, due to reduced feed intake, heat-stressed cows ruminate less and therefore generate less saliva. The reductions in the amount of saliva produced and salivary HCO₃⁻ content and the decreased amount of saliva entering the rumen make the heat stressed cow much more susceptible to sub clinical and acute rumen acidosis (Yuan *et al.*, 2011). When cows begin to accumulate heat, there is a redistribution of blood to the extremities in an attempt to dissipate internal energy. As a consequence, there is reduced blood flow to the gastrointestinal track and nutrient uptake may be compromised (Mader *et al.*, 2006). Environmental factors influencing heat stress in feedlot. Therefore, fermentation end products (VFAs) probably accumulate and contribute to the reduced pH.

3. Effects of Stress on Reproduction Health

3.1. Effect of Stress on Semen Quality

In the Male Environmental stress can cause low sperm quality, which is closely related to low fertility in females, probably due to a combination of low fertilization rates and increased embryonic mortality (Yousef *et al.*, 1968). Direct exposure of the testis at high temperatures, causes changes in certain critical stages of spermatogenic cycle, which is also directly related to the quality of the ejaculate and reduced sperm motility (Thun *et al.*, 1996). Suggested that the effect of stress on sperm quality can be improved by implementation of the seminal freezing technology; however, the uterus of female stress may represent heat to sperm.

3.2. Effect of Stress on Sexual Behavior and Fertility Rate

In the female sexual behavior and fertility rate are the main indicators of the mammalian female reproduction that are negatively affected by environmental stress. A temperature increase of 0.5°C uterine during hot days caused a decrease in the rate of fertilization. In cattle, heifers' exposure to 32°C for 72 hours after insemination, inhibit embryonic development. The Copulatory behavior of both male and female animals in the time of stress exposed for depression, anxiety, decreased estrus in heat stressed cattle, zoo animals decreased reproductive capacity associated with captivity and chronic stress may interfere with central and peripheral pathways of the sexual response (decreased sex drive) (Wolkowitz *et al.*, 2001). Maternal behavior; Stress during gestation alters postpartum maternal care decrease and related to offspring prenatal stress induces developmental and behavioral disorders (Webster, 2001).

3.3. Effect of Stress on Fertility

Fertility in lactating cows varies by season. In the winter, decreases about 50%, 20% in the summer and fall is lower than in the winter. A few years earlier, Brown-Brandl *et al.*, (2005) reported that conception rates fell from 52% in winter to 24% in the summer. In summer, 80% of estrus may be undetectable. Brown-Brandl *et al.*, (2005) when indicated, the rectal temperature of the animals increased from 38.5°C to 40°C in 72 hours after insemination service, pregnancy rates can decrease up to 50%. Heifers and cows studies have indicated that the decline in oocyte quality in the early postpartum period is associated with negative energy balance and low body condition of the animals, which is expressed in developing embryos increased and abnormal having embryos resulting in loss of the hottest months of the year (Mitlöhner *et al.*, 2001) Male animal exposed for stress the gamete maturation or formation resulted in disturbance of spermatogenesis, decreased sperm fertility parameters but, in the case of female animals' disturbance of folliculogenesis and also may inhibit gonadotropic responsiveness in granulosa cells, given glucocorticoid receptor presence (Fernandes *et al.*, 1997)

Heat stress reduces the length and intensity of estrus. For example, in summer, motor activity and other manifestations of estrus are reduced (Hansen and Arechiga, 1999) and incidence of anestrus and silent ovulations are increased (Gwazdauskas *et al.*, 1981). Possible reasons for reduced estrous expression are from suppressed endocrine hormones such as luteinizing hormone and estradiol, important for follicle growth and triggering estrous behavior (Rensis and Scaramuzzi, 2003). Heat stress impairs follicle selection and increases the length of follicular waves; thus reducing the quality of oocytes and modulating follicular steroid genesis (Roth *et al.*, 2001).

3.4. Effect of Stress on Embryo

Several studies (Bhatia and Tandon, 2005) have indicated that in cattle, embryonic development is highly sensitive to high temperatures, in the top three to 11 days after service; acquiring more heat tolerance as the gestation period progresses. It is known that the embryos obtained by in vitro fertilization (IVF) are more susceptible to heat stress than those obtained under natural conditions. In this regard, (Freestone *et al.*, 2008) indicated that the greatest loss of bovine embryos from IVF, occur before 42 days, when females are under heat stress. Early embryonic loss in livestock and increased incidence of spontaneous abortion, preterm delivery, exhibit reduced uterine and umbilical blood flows, resulting reduced fetal oxygen, nutrients and fetal size, increased uterine a resistance with high anxiety scores and low birth weight (Fox and Tylutki, 1998).

4. Summery and Conclusions

The stressor can be any internal or external stimuli or threat that disrupts homeostasis of the body, and elicits a coordinated physiological response within the body in an attempt to reestablish homeostasis. The different stressors that enhance stress and disturb the normal physiological functions of the different organs of animal health in different ways. Finally stress increased incidence of diseases and suffering animals as well as if the stress level is chronic it leads to death.

5. Acknowledgement

We are deeply grateful and indebted to all sources of materials used for reviewed this manuscript have been duly acknowledged.

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