

Effect of Heat Stress on Nutrient Metabolism and Feed Intake of Ruminant Animals: A Review

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Abstract: The objective of this review paper is to access the effect of heat stress on nutrient metabolism and feed intake of ruminant animals. Animals go through heat stress (HS) when the body temperature is higher than the optimal range specified for the normal activity because the total heat load is greater than the capacity for heat dissipation. Reduction in dry matter intake is generally associated with a decrease of rumen passage rate and an increase of diet digestibility in ruminants maintained in thermo neutrality. Endogenous heat production increases due to the metabolic utilization of crude proteins and this is higher than that for starch or fat. The greater heat increment from crude proteins is partially related to urea synthesis and to greater protein turnover. The changes in blood metabolites concentration due to water restriction should be separately considered in the acute and in the chronic restriction. During acute water restriction, serum protein and albumin increase due to the decreased blood volume; during chronic water restriction, both metabolites tend to decrease. Heat-stressed animals reduce feed intake, ostensibly as a survival strategy as digesting and processing nutrients generates heat, especially in ruminant animals. It has traditionally been assumed that inadequate feed intake caused by the thermal load was responsible for decreased milk production. Therefore this paper address the heat stress and there effect on nutrient metabolism of the ruminant animal.

Key words: Dissipation • Metabolism • Metabolites • Ruminant Animals • Synthesis

INTRODUCTION

Heat stress is one of the factors making animal production challenging in many parts of the world [1]. Heat Stress animals reduce dry matter intake (DMI), activity and metabolic rate in an attempt to decrease metabolic heat production [2]. In dairy cattle, HS decreases milk yield [3], which has been traditionally attributed to the heat-induced reduction in DMI [4].

Heat stress brings about changes in post-absorptive metabolism of animal independent of decreased feed intake and energy balance [5]. Animals go through heat stress (HS) when the body temperature is higher than the optimal range specified for the normal activity because the total heat load is greater than the capacity for heat dissipation [6].

Reduced feed intake occurring in animals exposed to hot environment partly explains the biological mechanism by which HS impacts production and reproduction [6]. This also includes an altered endocrine status, reduction in rumination and nutrient absorption and increased maintenance requirements Collier *et al.* [7] resulting in a net decrease in nutrient/energy availability. In a review, Kadzere *et al.* [8] concluded that exposure to a hot environment is responsible for an increase of digestibility that may be explained by the reduction of DMI and prolonged retention of feed in the gastrointestinal tract. Nevertheless, results available in literature on the effects of hot exposure on diet digestibility are often conflicting.

Milk composition is also discordantly altered during hyperthermia, which indicates that HS regulates component synthesis in addition to its overall effect on

milk yield [9, 10]. For instance, HS decreases milk protein content and yield, but the involved mechanisms remain largely unknown. Rhoads *et al.* [11] suggested that modest changes in the somatotrophic axis may explain a small portion of the reduction in milk protein yield during HS. Reduction in DMI is generally associated with a decrease of rumen passage rate and an increase of diet digestibility in ruminants maintained in thermo neutrality [12]. Therefore this paper address the heat stress and there effect on nutrient metabolism of the ruminant animal.

The Objective of this Paper Is:

- To understand the effect of heat stress on nutrient metabolism.
- To understand the effect of heat stress on feed intake.

Effect of Heat Stress on Nutrient Metabolism

Carbohydrate Metabolism: Carbohydrates are a readily available energy source that can be converted into a number of metabolic intermediates and used in synthesis reactions, or utilized for the production of ATP. ATP generation is a highly regulated process involving three distinct pathways generally termed glycolysis, the tricarboxylic acid cycle (TCA) and oxidative phosphorylation (via the electron chain transport). Pathway regulation is determined by several mechanisms affecting enzyme activity including synthesis and degradation rates, allosteric interactions and covalent modification and the energy charge of the cell [13]. Additionally, insulin and leptin concentrations, key hormones in energy metabolism, tended to decrease in water-restricted Awassi ewes [14]. Blood glucose levels are affected by the severity and the duration of heat stress.

Saunders *et al.* [15] reported that mild heat stress increased significantly intramuscular glycogen phosphorylase and pyruvate dehydrogenase activity, without affecting the intramuscular concentrations of glucose 6-phosphate, lactate, pyruvate, acetyl-coenzyme a (acetylCoA), creatine, phosphocreatine or ATP. Due to the increased reduction of fatty acid oxidation under chronic heat stress, heat-stressed animals become increasingly dependent on glucose for their energy needs. Taking into account the decreased feed intake and blood glucose levels, the use of glucose to meet production variables (lactation and growth) is declined

[16]. Hepatic glucose production typically decreases after ingesting carbohydrates; however, exogenous sugars are unable to blunt HS-induced liver glucose output [17]. The increased hepatic glucose output originates from increased glycogenolysis [18].

A large proportion of an animal's mass comprises skeletal muscle, which can have a profound impact on whole-animal energy metabolism and nutrient homeostasis, especially during periods of stress. To better understand how an environmental heat load influences the set points of several metabolic pathways within skeletal muscle, Rhoads *et al.* [19] examined heat stress effects on skeletal muscle during beef cattle adaptation to chronic heat stress using microarray analysis.

The exogenous sugars supplementation reduces hepatic glucose production, but do not blunt the heat-induced liver glucose output [20]. These findings indicate the increased rate of glycogenolysis and gluconeogenesis under chronic heat stress [21]. Similarly, acute heat stress results in an enhanced glycogenolysis but a decreased gluconeogenesis [21]. Interestingly, Wheelock *et al.* [22] and O'Brien *et al.* [23] reported that hepatic expression of the pyruvate carboxylase gene increases under chronic heat stress. Pyruvate carboxylase is a rate-limiting enzyme that regulates the entry of alanine and lactate into the gluconeogenesis pathway. Several authors reported increased blood lactate concentrations in different heat-stressed models [24].

Insulin is a potent regulator of carbohydrate and lipid metabolism and plays an important role in mediating the regulation the post-absorptive nutrient partitioning in heat-stressed animals. Although acute heat stress was shown to decrease insulin concentrations of lactating cows [25]. Increased insulin receptor abundance was reported by Tech *et al.* [26] in heat-stressed cows. Increased insulin blood level during chronic heat stress explains the decreased circulating glucose concentration, the blunted lipolytic activity and the increased lipogenesis of adipose tissue.

Protein Metabolism: Heat stress also affects post absorptive protein metabolism, as illustrated by changes in the quantity of carcass lean tissue in a variety of species [27]. Muscle protein synthesizing machinery and RNA/DNA synthesis capacity are reduced by environmental hyperthermia and similar effects apparently occur with regard to mammary a- and b-casein synthesis.

Skeletal muscle catabolism is also clearly increased during HS, because numerous studies have reported increased plasma markers of muscle breakdown in a variety of species [25]. Lot of studies demonstrated that heat-stressed cattle were in negative nitrogen balance, as consequence of the reduction in feed intake [28]. The reduction in the feed intake can be counteracted by the increase of protein content of the diet, which can lead to an excess of nitrogen intake. Endogenous heat production increases due to the metabolic utilization of crude proteins and this is higher than that for starch or fat. The greater heat increment from crude proteins is partially related to urea synthesis and to greater protein turnover. HS depresses RNA content, proteolytic rates and muscle protein turnover particularly acute heat stress increases protein catabolism. Increased protein catabolism under chronic heat stress is likely to produce glucose through the gluconeogenesis pathway. The inability of heat stressed animals to utilize glucose sparing mechanisms to prioritize milk synthesis results in inflexibility of metabolism [29]. Heat stress directly affects protein metabolism by increased skeletal muscle breakdown to afford amino acids which are necessary for energy metabolism [30].

Lipid Metabolism: Heat stress (HS) affects numerous physiological processes including nutrient partitioning and lipid metabolism. Lipid metabolism is affected by chronic heat stress. Ambient temperature-induced heat stress was shown to reduce fat oxidation in different species. Several studies demonstrated that during heat stress, basal levels of NEFA are typically reduced in dairy cows [31]. Although lipolytic enzyme activity is reduced under heat stress, the activity of the lipoprotein lipase of the adipose tissue is increased, which allows suggesting that hyper thermic animals have a greater storage capacity of intestinal and hepatic triglycerides [32]. Increased insulin sensitivity has been described for animals experiencing HS *in vivo* [24]. Therefore, this study investigated the interaction between thermal treatment and insulin administration to investigate their lipolytic and lipogenic effects on adipocytes cultured under HS conditions.

Water Metabolism: Water is a basic molecule in the body of vertebrates because it is essential for the maintenance of some vital functions: tonicity of tissue (by electrolyte balance and osmotic regulation); lubrication and

thermoregulation; nutrient transport; excretion. For the purpose of this review, it is pivotal the role of water in the homoeothermic subjects because it represents an important heat carrier for the regulation of thermal exchanges [33]. Water metabolism under heat stressful condition is closely linked to the thermoregulatory requirements of the ruminant. High-producing dairy cows have higher metabolic rate than lower-producing ones; this implies they experience more difficulties to dissipate body heat during the hot season [34].

The changes in blood metabolites concentration due to water restriction should be separately considered in the acute and in the chronic restriction. During acute water restriction, serum protein and albumin increase due to the decreased blood volume; during chronic water restriction, both metabolites tend to decrease [35]. At the same time, acute water restriction induces the kidney to slow glomerular filtration and increase urea reabsorption; this induces the increase of plasma levels of creatinine and urea, whereas chronic water restriction induce a reduction of these plasma metabolites [35]. The dietary level of some nutrients may affect the water requirement, namely for an increased demand for urine excretion. This is the case for dietary crude proteins and Kumar *et al.* [36]. Recently, a comprehensive model for water metabolism under thermo neutral conditions gave a driving role to the requirement for N excretion in determining water loss by urine [37]. HS influences water metabolism by increasing plasma and extracellular fluid volume in proportion to the thermoregulatory requirement of the cow [38].

Vitamins and Minerals Metabolism: Niacin helps to alleviate HS both by increasing evaporative heat loss from the body and also by reducing the effects of heat at the cell level [39]. Feeding protected niacin increased free plasma niacin levels, evaporative heat loss during peak thermal load and associated with a small but detectable reduction in rectal and vaginal temperatures in dairy cows experiencing a mild thermal load [40], with variable effect on milk production [41].

Mineral supplementation under hot climate must be viewed not only as a simple mean to cover the important (and increased) turnover of a specific nutrient, but also as a mean to buffer the effect of the diet and of climate [42]. Chromium is a micronutrient that facilitates insulin action on glucose, lipid and protein metabolism. Little is known about actual dietary chromium requirements; however, because glucose use is predominant during HS, chromium

supplementation could reduce the negative effects of HS. Dairy cows in early lactation supplemented with chromium under hot conditions have shown a reduction of weight loss, an improvement of milk production, a reduction of plasma NEFA concentrations and an improvement of rebreeding rates [43].

Effect of Heat Stress on Feed Intake: Many species limit their feed intake during HS to minimize the thermic effect of feeding, resulting in reduced growth [24, 44]. However, despite the reduced nutrient intake, heat-stressed animals actually retain more adipose tissue when compared with thermo neutral (TN) counterparts on the same plane of nutrient intake [24, 44]. The mechanisms responsible for the altered hierarchy in tissue accretion rates during HS are not clear. However, post absorptive changes in nutrient partitioning in some heat-stressed models do not reflect normal metabolic modifications observed in thermal-neutral (TN) animals on a similar restricted plane of nutrition. For example, using a pair-feeding model (to distinguish between the direct and indirect effects of HS), we have demonstrated that despite marked reductions in nutrient intake and BW loss, heat-stressed ruminants have increased basal and stimulated circulating insulin concentrations [25, 28].

Heat-stressed animals reduce feed intake, ostensibly as a survival strategy as digesting and processing nutrients generates heat, especially in ruminants [4, 44]. It has traditionally been assumed that inadequate feed intake caused by the thermal load was responsible for decreased milk production [4, 44]. However, recent results challenge this dogma and have demonstrated disparate slopes in feed intake and milk yield responses to a cyclical heat load pattern [31].

CONCLUSIONS

Generally, fully understanding how HS influences animal agriculture requires the consideration of three main components: 1) the environmental conditions, 2) the animal thermal sensitivity/tolerance and 3) available resources to employ heat mitigation and management strategies. In fact, the earth temperature is expected to be increased 1.1-6.4°C. Environmental research, particularly HS and its implications should be prioritized in order to ameliorate its negative effects of future climate change. Conducting more HS research is the foundation of better understanding thermal biology; a

prerequisite in developing more effective strategies to maximize food production in inhospitable environments. Animals housed under optimum environmental conditions (also known as thermo-neutral zone; TNZ) will achieve or be close to achieving their genetic potential. Thus, environments that are outside of the TNZ will reduce nutrient utilization into valuable products as animals will prioritize temperature regulation instead of production.

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