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Adaptability of goats to heat stress: A review

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Abstract
In the environment, animals have to cope with a combination of natural factors. Extreme changes in these factors can alter homeostasis, which can lead to thermal or heat stress. This stress can be due to either high or low temperature. Energy transference for thermoregulation in homoeothermic animals occurs through several mechanisms: conduction, convection, radiation and evaporation. When animals are subjected to thermal stress, physiological mechanisms are activated which may include endocrine, neuroendocrine and behavioral responses. 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. Mechanisms which have developed through evolution to allow animals to adapt to high environmental temperatures and to achieve thermo-tolerance include physical, physiological and biochemical changes like reduced feed intake and metabolic heat production, increased surface area of skin to dissipate heat, increased blood flow to take heat from the body core to the skin and extremities to dissipate the heat, increased numbers and activity of sweat glands, panting, increased water intake and color adaptation of integument system to reflect heat. Chronic exposure to thermal stress can cause disease, reduce growth, decrease productive and reproductive performance and, in extreme cases, lead to death. This paper aims to briefly explain the physical and physiological responses of mammals to thermal stress, like a tool for biological environment adaptation, emphasizing knowledge gaps and offering some recommendations to stress control for the animal production system.

Keywords: Adaptability, climate change, thermal stress, heat dissipation, heat tolerance

Introduction
Small ruminants like goats play a predominant role in the economy of million people, and have provided meat, milk, skin and fibre for centuries. Animals undergo various kinds of stress, i.e. physical, nutritional, chemical, psychological and heat stress. Among all, heat stress is the most concerning at present in the ever-changing climatic scenario. Climate change is the most serious long-term challenge faced by owners of small ruminants worldwide. Heat stress results in decreased growth, reproduction, production, milk quantity and quality, as well as natural immunity, making animals more vulnerable to diseases, and even death. Thus, heat stress results in great economic losses, emphasizing the necessity to objectively assess animal welfare. The increasing demand for animal products paralleled by the frequent hot climate is a serious threat for the agricultural sector. The ability of goats to cope with heat stress without harming their welfare and productive performance has been often overrated. To date, little attention has been paid to comprehensive detailed data on the adverse effect of heat stress on goats. Therefore, this review discusses in detail the behavioural, physiological, molecular/cellular, haematological, biochemical and immunological responses of goats under heat stress conditions, and review suggests various strategies for alleviation of heat stress.
The demand for livestock (including small ruminants) products has been largely driven by the rapid growth of the human population, increases in income and urbanization [1]. The livestock sector is increasingly organized in long market chains that employ more than 1.3 billion people globally and directly support the livelihoods of 600 million poor smallholder farmers in the developing world [1]. Globally, small ruminants play an important role in the economy of millions of people who earn their livelihood by rearing these animals in different climatic conditions [2-4].
Goats are among the species with a wide geographical distribution, playing an important role in the agricultural revolution and advance of human civilization. The goats are thought to have been domesticated around 10,000 years ago [5, 6], in the socalled ‘Fertile Crescent’ of the Middle East [7]. This species plays a predominant role in the sustenance of the livelihoods of impoverished families, especially in rural areas [8, 9]. Goats owe their popularity to their multi-purpose ability to provide meat, milk, skin, offal, horn, dung for fuel, and fiber [2, 6].

Also, these animals are well adapted under different geographical and environmental conditions including extreme and harsh climates [10], and perform better than other domesticated ruminants. Goats tend to tolerate heat better than other species [11, 12]. Worldwide, there are more than 600 goat breeds [6], and these breeds differ in their capacity to overcome climatic conditions. Goats with loose skin and floppy ears are more heat tolerant than other goats [12]. Angora goats have a decreased ability to respond to heat stress (HS) as compared to other breeds of goats [12]. However, despite their extreme tolerance, the productivity of these animals often declines due to heat stress [10]. Therefore, proper breed selection is a very valuable tool for sustaining animal production under an increasingly challenging environment [13]. Nevertheless, importing high-producing breeds from temperate to desert and tropical areas does not always work well because of poor adaption to heat stress. Crossbreeding is a common strategy to resolve this problem.

Stress is described as the cumulative detrimental effect of a variety of factors on the health and performance of animals, or also described as the magnitude of forces external to the body which tend to displace its systems from their ground state [11]. Animals undergo various kinds of stressors, i.e. physical, nutritional, chemical, psychological and heat/thermal stress. Among all, heat stress is the most concerning issue nowadays in the ever-changing climatic scenario [14], and it is one of the most important stressors especially in the tropical, subtropical [15], arid [13], and semiarid [11, 16] regions of the world. Heat stress is the perceived discomfort and physiological strain associated with an exposure to an extreme and hot environment [4].

Climate change is the most serious long-term challenge faced by small ruminants' owners in much of the world, as it impacts animals' production and health [14]. Environmental factors such as ambient temperature, solar radiation and relative humidity have direct and indirect effects on animals [17]. High ambient temperature is the major concern that challenges the animal's ability to maintain energy, thermal, water, hormonal and mineral balance [13]. The intergovernmental panel on climate change reported that the period from 1983 to 2012 was the warmest 30-year period of the last 1,400 years in the Northern Hemisphere [18]. They predict that by the year 2100, the increase in global surface temperature may be 3.7–4.8°C [19]. It is expected 20–30% of livestock to be at risk of extinction [20] due to changes in weather and climate events. There is evidence that climate change, especially elevated temperature, has already changed the overall abundance, seasonality and spatial spread of farmed small ruminants [21]. Animal welfare outcomes show that heat stress compromises animal welfare and it is commonly stated that heat stress causes mortality in goats under inappropriate transport conditions [22]. However, unlike the situation in cows [23] there are no reports so far on mortality due to heat stress under conventional farming conditions, even in those raised in areas exposed to extreme heat stress (e.g., in Saudi Arabia).

Heat stress influences ovarian function and embryonic development which results in decreased fertility [4]. Heat stress redistributes the body resources including protein and energy at the cost of decreased growth, reproduction, production and health of animals [4]. In addition, heat stress reduces milk yield and quality [24]. The general homeostatic responses to heat stress in goats include raised respiration rate, body temperature and water consumption, decreased feed intake, and dry matter intake [4, 22, 25, 26]. Also, heat stress lowers natural immunity, making animals more vulnerable to diseases [27]. Collapse and even death can result if the heat load experienced by the animal becomes excessive [28].

Behavioural responses of goats to heat stress

Despite the numerous published studies on the impact of heat stress on productive and reproductive parameters, little is published about the behavioural changes in goats during heat stress. However, animals behave in various ways during heat stress, and that can provide insights on how and when to cool them. The range of behavioral responses affects heat exchange between the animal and its environment by reducing heat gain from radiation and increasing heat loss via convection and conduction [29]. Behavioral responses of goats under heat stress include: bunching in the shade [11], slobbering, panting, open-mouth breathing, decreasing feed intake and increasing water consumption [26]. In severe cases of heat stress in goats, lack of coordination, trembling and down animals may be seen. In addition, desert animals usually use nocturnal activity (most active during the night) to reduce heat load in hot conditions [30].

Seeking shade is a conspicuous form of behavioral adaptation. If shade is not available, animals will change their posture to the vertical position in respect to the sun in order to reduce the effective area for heat exchange [30]. Animals can change posture, i.e. stand or spread out to increase surface area for heat loss and reduce activity [31]. Under severe heat stress, animals moisten their body surface with water, saliva or nose secretions [11]. Animals may react either by physiological or behavioral responses, but most often a combination of both [32]. For example, goats expressed decreased urination and defecation under heat stress conditions [33, 34]. The reason for reduced urination frequency could be due to increased respiratory and cutaneous cooling mechanisms which might lead to severe dehydration, thereby leading to a reduction in their urination frequency [34]. Also, the reduction in defecating frequency could be an adaptive mechanism of these animals to conserve body water [34]. In extreme heat, grazing ruminants decrease their grazing time, tend to lie down to reduce their locomotion and spend more time in the shade [11, 35]. Standing and lying are behavioral adaptive mechanisms to prevent additional heat load from the ground and to facilitate effective heat dissemination [34, 33] concluded that heat-treated goats (4–8 h heat exposure for 18 days) showed an elevation in standing time (445 vs. 390 min), and spent less time lying down (50 vs. 90 min) as compared to control goats.

Interaction between stress and nutrition results in nutrient deficiency as heat stress is associated with marked reduction in feed intake [36]. There is also a direct effect of heat stress on the feeding centre of the hypothalamus, resulting in a hormonal response, which could also decrease metabolic rate [36, 37]. Heat-stressed animals decrease feed intake in an attempt to create less metabolic heat because the heat increment of feeding is an important source of heat production [38]. Also, the maintenance requirements increased by 30% because of heat stress [39] and the energy intake would not be enough to cover the daily requirements which results in an apparent body weight loss [40]. In fact, heat-stressed animals enter a bioenergetic state similar (but not to the same extent) to the negative energy balance observed in early lactation [41]. Negative energy balance is associated with a variety of metabolic and hormonal changes; it is likely that many of the negative effects of heat stress on production,

animal health and reproduction indices are mediated by the reduction in energy balance [41].
Literature reported a marked loss of feed intake and body weight of animals exposed to heat stress. Feed intake reduction due to heat stress has been reported in goats [40, 42]. In addition, body weight, daily feed intake and gain decreased under heat stress conditions in goats [9]. Loss of body weight during heat stress might be attributed to the increase in energy expended for heat dissipation through respiratory evaporation, and subsequently to the reduction in the amount of water available for storage [9, 43]. Dairy goats decreased their dry matter intake by 30% under heat stress conditions [24]. Numerous nutritional changes through researcher guidance to producers/farmers in diet, are needed during heat stress to maintain feed intake, increase nutrient density and minimize the negative effect of heat stress [36].
Water is one of the most important nutrients required for the maintenance of life, and it is involved in many physiological functions essential for performance of small ruminants. Water is essential for the adjustment of body temperature, growth, reproduction, lactation mechanisms, digestion pattern, nutrient exchanges and transport to and from cells in blood, excretion of waste products and heat balance [2]. Water requirements are regulated by dry matter intake (during drought, animals require more water as they are forced to select more fibrous and less digestible feed), environmental temperature (animals use more water for evaporative cooling in hot weather), and loss of water from body evaporation (body surfaces and the respiratory tract), urine, feces, and milk [44]. Unlike feed nutrients, water does not receive adequate consideration to ensure optimal performance of ruminant animals, mainly those raised under hot conditions. Small ruminants may experience moderate to severe water restriction during a variety of commonly occurring environmental conditions (i.e., drought periods, shipment, when grazing in areas far from watering sources), and their requirements for water in dry areas are high due to high temperature and radiation load from the sun [2]. Goats are characterized by a remarkable tolerance to drought conditions. In general, goats are better at conserving water than sheep and possibly due to their browse diet [6, 14, 35]. Goat breeds differ in their capacity to cope with hot periods without water, for example, Black Bedouin and Barmer goats can live on a once every four days watering regime [13, 45]; and desert goats raised under traditional systems may be watered only once every three to six days, when water is scarce [46]. Goats kept under heat stress conditions doubled their water consumption [22, 24]. Increased water intake was mainly used by heat-stressed goats for boosting heat loss by sweating and panting [40]. In this regard, total water evaporation from water input was three times greater in the heat-stressed goats than the controlled ones [40].

Physiological responses of goats to heat stress
High ambient temperature has potentially several physiological adverse effects that result in a tremendous economic loss for the goat industry. These include aberration of reproductive functions, oxidative stress, enzymatic dysfunction, electrolyte imbalances, promoting an unfavorable endocrine balance and reducing feed intake, and meat quality [47-49]. Physiological parameters like respiration rate, heart rate and rectal temperature give an immediate response to heat stress [50], and consequently the level of animal discomfort/comfort. Changes in respiration rate, heart and/or pulse rate and rectal temperature have been frequently used as indices of physiological adaptability to heat stress in small ruminants [4, 48, 51]. Increased body temperature and respiration rate are the most important signs for heat stress in goats [33]. However, it is important to mention that complexity and a suite of physiological changes due to heat stress response can differ from species to species, individual to individual, and the hormonal status of the animal.

Respiratory Rate: The respiration rate (breaths/min) can change frequently and it is indirectly influenced by the animal's activities (metabolism and muscle activity) [52], and environmental conditions [35]. Respiration rate is a practical and reliable measure of heat load and an indicator of heat stress [9, 35]. Respiration rate per minute was found to be increased by the effect of environmental temperature [53]. Increased respiration is an attempt to increase heat loss by evaporative cooling. The changes of metabolism and muscle activity of goats also changes pulsation and respiration rates [52]. Heat loss via high respiration rate was reported as higher than that via other ways [52]. The respiration rate is recorded by counting flank movements per minute from a distance of 4–5 m without disturbing the animals [34] or from a non-obstructive distance [48]. The basal reference respiration rate is 15–30 breaths/min in goats [54]. Thus, measuring respiration rate and deciding if an animal is panting, and qualifying the severity of heat stress according to panting rate (breaths/min) (low: 40–60, medium: 60–80, high: 80–120, and severe: >200) appears to be the most accessible and easiest method for evaluating the impact of heat stress on animals under extreme conditions [11, 35]. In this regard, [35] reported that the respiration rate approaching 300 breaths/min with open-mouthed panting is indicative of severe heat stress. Increased respiration rate following heat stress has been reported in goats: 22–162 breaths/min [55]; 23 breaths/min [9]. The increased respiration rate is probably indicating an effort of animals to maintain their normal body temperature by increasing their heat dissipation through increasing respiratory evaporation [40].

Heart and Pulse Rates: The heart rate is the regular beat rate of the arteries as the blood is pumped through them to the heart [56]. Heart rate (expressed through beats/min) can be rapidly altered due to animal biological activities or by external factors such as temperature. Normal heart rates range from 90 to 95 beats/min for goats [56]. Heat exposure showed a higher heart rate of 74 to 91 beats/min in goats [9, 33]. The heart rate increases under heat stress conditions, and this increases blood flow from the core to the surface of the body to give a chance for more heat to be lost by sensible (conduction, convention and radiation) and insensible (diffusion water from the skin) means [48]. However, heart rate reflects primarily the homeostasis of circulation along with the general metabolic status. Heat stress reduces the heart rate, and the marked acceleration of the heart rate occurred during the hottest part of the day [55] have suggested that heart rate decreased because of the general effort of the animal to decrease heat product. Pulsation rate per minute was found to be increased by the effect of environmental temperature [53]. Increase in heart rate and pulse rate is attributed to two causes. One is the increase in muscular activity controlling the rate of respiration, concurrent with elevated respiration rate. The second is the reduction in resistance of peripheral vascular beds and arteriovenous anastomoses. Increase in pulsation rate increases blood flow from the core to the surface as a result of it more heat is lost by sensible (loss by conduction, convention and radiation) and insensible (loss by diffusion water from the skin) means . The increase in cardiac output and cutaneous blood flow by heat stress, due to blood redistribution from deep splanchnic to more peripheral body regions, have been implicated in goat [11, 35].

Rectal Temperature: The body temperature is a good measure of heat tolerance in animals. It represents the resultant of all heat gain and heat loss processes of the body. Rectal temperature is considered as a good index of body temperature even though there is a considerable variation in different parts of the body core at different times of the day Rectal temperature of goats was found to be elevated with high environmental temperature in several studies [52]. Maintenance of body temperature is under neuronal control in a negative feedback system [58]. Temperature sensitive neurons (warm and cold), which are found in the preoptic region of the anterior hypothalamus, are considered a thermostat with a desired set point [59]. Temperature sensors are also found in the skin and deep tissues of the body (e.g., thorax, around the great veins of the abdomen and in the abdominal viscera) [59]. Fever and hyperthermia are two distinct causes of high body temperature. Fever is a complex reaction to pyrogens that not only cause the body's thermoregulatory set point to rise, but also stimulates an acute-phase reaction and activates numerous metabolic, endocrinologic and immunologic systems and behaviours [58, 60]. While hyperthermia represents a failure in thermoregulation (uncontrolled heat production, poor heat dissipation or an external heat load), this does not involve a thermoregulatory set point [58, 60], and the microbial products and pyrogenic cytokines are not directly involved [60]. It is likely that most cases of elevated body temperature after stroke are due to fever, and not to hyperthermia [60]. The temperature in the rectum is representative of deep body temperature [61]. It represents the result of all heat gain and heat loss processes of the body [4]. Rectal temperature is an indicator of thermal balance and may be used to assess the adversity of heat stress which can affect growth, lactation and reproduction [36]. A rise in rectal temperature of 1°C or less is enough to reduce performance in most livestock species [62], which makes body temperature a sensitive indicator of the physiological response to heat stress because it is nearly constant under normal conditions [11]. The physiological adjustments that animal makes to prevent body temperature from raising during heat stress help prevent death from heat stroke, but also reduce productivity [13].

Domestic animals are homeotherms which tend to maintain a constant body temperature through a balance of heat gain and loss. Its variation above and below normal is a measure of the animal's ability to resist stress environmental factors. However, [63] reported rectal temperature between 39.2°C and 39.8°C for goats. Heat exposure increased goats' rectal temperature from 37°C to 41°C [9, 64]. In contrast, no rectal temperature changes were reported in goats [33, 55] exposed to heat treatments. A daily change in respiration rate per minute from the effect of environmental temperature may not be parallel with change in body temperature and pulsation number (31). Higher values of means of these parameters (RR, RT, HR) have been reported than that of values in thermo-neutral zone [64, 65].

Haematological responses of goats to heat stress

The blood profile of animals is sensitive to changes in the environmental temperature and is an important indicator of physiological responses to the stressing agent [9]. Determination of blood parameters may be important in establishing the effect of heat stress. Heat stress has an effect on animals as revealed by changes in hematological parameters, i.e. red blood cells, white blood cells, hemoglobin, lymphocytes, neutrophil, eosinophil, monocyte, granulocytes, packed cell volume and blood pH. When exposed to heat stress, goats showed an increased amount of red blood cells, packed cell volume, haemoglobin, white blood cells, neutrophil, eosinophil, lymphocyte and monocyte [33]. Also, packed cell volume, haemoglobin and red blood cells were higher under heat stress in goats [9]. In contrast, heat stress decreases packed cell volume and haemoglobin [66], and white blood cells in goats [9]. Another explanation of the increase in packed cell volume and hemoglobin levels could be either increased un-attack of free radicals on the red blood cells membrane, which is rich in lipid content, and ultimate lysis of red blood cell [67] or availability of adequate nutrients for synthesis of haemoglobin as the animal consumes more feed or decreases voluntary intake under heat stress [4].

Biochemical responses of goats to heat stress

The acid-base balance is a complex physiological process to maintain a stable pH in an animal's body. The body utilizes different mechanisms to combat any change in acid-base balance, i.e. chemical buffering, respiratory adjustment of blood carbonic acid (H_2CO_3) and excretion of hydrogen ions or bicarbonate (HCO^{3-}) by the kidneys [68]. Metabolic acidosis and alkalosis involving HCO^{3-} as well as respiratory acidosis and alkalosis related to partial pressure of CO_2 may occur under heat stress. The vital limits of pH variation for mammals are between 7.35 and 7.45 [68, 69] and regulated by a complex system of buffers (H_2CO_3 and HCO^{3-}). Blood pH increased under heat stress in goats (66). The increase in pH may be due to reduced H_2CO_3 (70), total CO_2, HCO^{3-} [40] and base excess in blood and extra-cellular fluid [66]. The secretion of HCO^{3-} in urine and its reabsorption suggest a large requirement and turnover of body HCO^{3-} to maintain blood pH during heat stress [40]. Blood biochemical parameters reflect the health (detecting possible diseases) and the metabolic status of an animal (evaluating the body's internal condition, the function of organs [e.g. kidneys and liver], and metabolic processes in the body), which are widely used in clinical situations. heat stress affects biochemical parameters, i.e. alkaline phosphatase, alanine aminotransferase, aspartate transaminase, lactate dehydrogenase, total protein, albumin, globulin, glucose, cholesterol, blood urea nitrogen, non-ester fatty acids, beta-hydroxybutyrate, creatinine, triiodothyroxine, thyroxine, cortisol, prolactin, sodium, potassium, chloride, calcium, magnesium, iron, manganese, copper, zinc and oxidative stress parameters (glutathione peroxidase, glutathione reductase, superoxide dismutase and lipid peroxides).

Enzymes: Metabolic regulators are important in elucidating a picture of modulation in physiological mechanisms during stressed conditions and are best assessed by determining the enzymes governing various metabolic reactions in plasma/serum [4]. Enzyme levels reflect the metabolic activities during stress. Heat stress reduces alkaline phosphatase and lactate dehydrogenase activity in goats [50].

The decrease in these enzymes during heat stress is due to the decrease in thyroid activity during heat stress [50]. Serum level of aspartate transaminase and alanine aminotransferase is helpful in the diagnosis of the welfare of animals. The serum alanine aminotransferase value increases during heat stress in goats [71].

Proteins: Significant decrease in total protein concentration in goats has been reported during heat stress [72]. Total plasma protein, albumin and globulin levels decrease in goats subjected to heat stress [50]. This may be due to an increase in plasma volume as a result of heat stress, which results in a decrease in plasma protein concentration. In contrast, heat stress increased total protein and albumin in goats [9] and could be due to dehydration which has been reported to occur as a result of increased respiration rate.

Glucose, cholesterol, blood urea nitrogen, non-ester fatty acids and beta-hydroxybutyrate: Studies on glucose, cholesterol, blood urea nitrogen, non-ester fatty acids and beta-hydroxybutyrate in response to heat stress are conflicting. Glucose and cholesterol levels show greater differences under heat stress conditions than in the comfort zone. Heat stress conditions decrease glucose and cholesterol levels in goats [43]. The decrease in glucose level could be related to the decrease in availability of nutrients and lower rate of propionate production [73], or due to the increase in plasma glucose utilization to provide energy for muscular expenditure required for high muscular activity associated with increased respiration rate [74]. The decrease in cholesterol level may have a relation with the increase in total body water or the decrease in acetate concentration, which is the primary precursor for the synthesis of cholesterol [4]. Heat stress had no effect on glucose and blood urea nitrogen in goats [40]. Non-ester fatty acids and betahydroxybutyrate are most indicative of the animal's energy status [40]. reported that a reduction in feed intake and body weight under heat stress was not accompanied by body fat mobilization as non-ester fatty acid concentration did not vary between heat-stressed and control goats. Exposure to heat stress resulted in higher beta-hydroxybutyrate concentration in goats [75].

Hormones: Hormones (i.e. thyroxine, triiodothyroxine, prolactin, leptin, adiponectin, growth hormone, glucocorticoids, mineralocorticoids, catecholamines and antidiuretic) are involved in thermal adaptation and could be important indicators for assessment of stress in animals [66, 76]. Decreased thyroid hormone level during heat stress is an adaptive response and affects the hypothalamic- pituitary-adrenal to decrease thyrotropin releasing hormone [37], which enables animals to reduce metabolic rate and heat production [36], and reduces the amount of heat produced by the cells [28]. In goats, [50] and [66] reported a decrease in plasma concentration of triiodothyroxine and thyroxine levels. Cortisol plays an important role in all types of stress. An increased cortisol level during heat stress was reported in goats [66]. The prolactin level increased in goats under heat stress [66].

Electrolytes: Heat stress challenges the animal's ability to maintain its mineral balance. The serum concentrations of sodium, potassium and chloride decreased in goats subjected to heat stress due to the fact that heat-stressed animals lost more potassium and chloride in sweat than non- heatstressed animals, and the blood volume expanded where water is transported in the circulatory system for evaporative cooling [66].

Oxidative stress parameters: Heat stress stimulates excessive production of free radicals such as reactive oxygen species (superoxide anion radicals, hydroxyl radical, hydrogen peroxide and singlet oxygen) which are continuously produced in the course of normal aerobic metabolism, and they can damage healthy cells if not eliminated [77]. Normal cells have the capacity to detoxify superoxide radicals using antioxidant enzymes (superoxide dismutase, glutathione peroxidase, glutathione reductase and catalase) and non-enzymatic antioxidants including vitamins (C, A and E), and proteins (albumin, transferrin, glutathione) [78, 79] reported that oxidative stress increases in goats during summer as superoxide dismutase increases.

Molecular / cellular responses of goats to heat stress

It is widely accepted that changes in gene expression are an integral part of the cellular response to thermal stress. Although the heat shock proteins (HSPs) are perhaps the best-studied examples of genes whose expression is affected by heat shock, it has become apparent in recent years that thermal stress also leads to induction of a substantial number of genes not traditionally considered to be HSPs. Some of these genes are affected by a wide variety of different stressors and probably represent a nonspecific cellular response to stress, whereas others may eventually found to be specific to certain types of stress. In mammalian cells, nonlethal heat shock produces changes in gene expression and in the activity of expressed proteins, resulting in what is referred to as a cell stress response [80]. This response characteristically includes an increase in thermotolerance (i.e., the ability to survive subsequent, more severe heat stresses) that is temporally associated with increased expression of HSPs. Thermal-induced changes in gene expression occur both during hyperthermia as well as hypothermia. About 50 genes not traditionally considered HSPs have been found to undergo changes in expression during or after heat stress. Many of these genes will likely to be proved as important mediators and effectors of the cell stress response.

The cellular response is one component of the acute systemic response to heat stress. High temperature results in alterations and damages at the cellular level. Cell components (i.e. unfolding and subsequent aggregation of proteins) are negatively affected by heat stress [81, 82]. Protein synthesis is particularly affected by heat stress but recovers quickly [77], whereas resumption of DNA synthesis requires a period of time [83]. The cellular response includes: activation of heat shock factors, increased expression of heat shock proteins, increased glucose and amino acid oxidation, reduced fatty acid metabolism, activation of endocrine and immune systems via extracellular secretion of heat shock proteins [84]. Heat shock factors exist in multiple isoforms in mammals, i.e. heat shock factors 1, 2 and 4. Heat shock factor 1 is the central transcription factor involved in the heat shock response [85]. Heat shock factor 1 is activated in response to heat stress and oxidative stress [86]. Heat shock factor 2 is primarily transcribed in response to the inhibition of proteasome activity and thus complements the response of heat shock factor 1 to an increase in misfolded proteins [87]. Heat shock proteins contribute to cell survival by reducing the accumulation of damaged or abnormal polypeptides within cells [88]. They play a crucial role in intracellular transport, maintenance of proteins in an inactive form, and the prevention of protein degradation [89]. Heat shock proteins 27, 60, 70, 90 and 110 are major heat shock proteins in mammalian cells [90], each with different functions and cellular locations [91]. Heat shock proteins are the best studied genes whose expressions are affected by heat stress. Expression of many heat shock proteins [32, 40, 60, 70, 90, 110] is increased during heat stress [51]. Heat shock protein 70 is used as a biomarker of cellular stress [77], and plays a heightened role in cryoprotection [92], and its expression level is indicative of magnitude and duration of heat stress [93]. In the nucleus, heat shock factor trimmer complexes bind promoters containing heat shock elements to activate heat stress target gene transcription [4, 84].

Immunological responses of goats to heat stress

Heat stress impairs immune function and increases disease susceptibility [94]. The immune system is classified into two categories: innate and adaptive immunity. Components of the innate defense include the physical barrier of the skin and mucous epithelia, leukocytes (macrophages, neutrophils and natural killer cells), non-immune cells (epithelial and endothelial cells), and certain soluble mediators (cytokines, eicosanoids and acute phase proteins). Inflammation is one of the hallmarks and first responses of the innate immune system to infection, and is associated with heat, redness, pain, swelling and impaired function. It has two main functions: remove the injurious agents and initiate the tissue healing process [95, 96]. The local inflammation that develops at the site of infection induces the acute phase response. The acute phase response is a complex systemic innate-defense system activated by trauma, infection, inflammation and stress to prevent tissue damage, eliminate any infective organisms and activate the repairing processes to restore homeostasis. It is induced by the release of inflammatory cytokines, especially Interleukin-1, Interleukin-6 and tumor necrosis factor-α from the macrophages or blood monocytes at the site of inflammatory lesions or infections. The liver is the main site of synthesis of most acute phase proteins. Cytokines therefore act as mediators between the local site of injury and the hepatocytes (liver) to produce and release the acute phase proteins [97].

Cytokines: Leptins are a kind of circulating adipocytokines whose concentration increases rapidly when the animal is exposed to hot environment [98]. The studies showed that expression of leptin mRNA is higher during winter season as compared to summer season [98]. The leptin is a pleiotropic hormone in caprine and changed leptin expressions in peripheral blood mononuclear cells (PBMCs) *in vivo* may represent an adaptive mechanism to environmental temperatures in goats suffering from severe thermal stress [98]. Under stress conditions Hypothalamus-Pituitary Axis gets stimulated which stimulates the production of catecholamine and glucocorticoids. Cytokine production and modulation of stress occur due to production of interleukins. Interleukins can act locally and systemically. Their action locally is to modulate cellular immune response. Systemically they change behavior, metabolism and neuro-endocrine secretion. IL-6 is considered as the primary mediator of metabolic response to inflammation by inducing production of a broad array of acute phase proteins. IL-1 and IL-6 inherently link muscle protein degradation with production of hepatic acute phase proteins as a response to inflammatory stimuli [99]. Production of these pro inflammatory cytokines directly affects bone growth and found to modulate the mechanism of proteins, fats and carbohydrates [100].

Acute Phase Proteins: The acute phase proteins are defined as proteins whose plasma concentrations increase or decrease classifying them into positive (i.e. C-reactive protein, serum amyloid A, haptoglobin), and negative (i.e. albumin, transferrin) acute phase proteins, respectively. The maximum concentration is usually reached within 24–48 hours after stimulation and declines with recovery from the infection [97]. In ruminants, there are two major acute phase proteins: haptoglobin and serum amyloid A which both increase during tissue injury and disease [101, 102]. Acute phase proteins are recognized as promising tools to assess welfare, health and performance in animal production [103]. The data on the use of acute phase proteins as biomarkers and potential indicators of stress in goats are rare. Goats exposed to heat stress increased circulating haptoglobin when they were metabolically challenged [40].

Nitric Oxide Synthases: Nitric oxide is a gaseous lipophilic free radical. It has three isoforms such as NOS type 1 or neuronal (nNOS), NOS type 2 or inducible (iNOS) and NOS type 3 or endothelial (eNOS) [104]. Nitric oxide regulates vascular hemostasis, hematopoeisis and peripheral immune response. Even in a small concentration, physiological and cellular activities are affected. Nitric oxide is required for full expression of active vasodilatation of the skin during hyperthermia. The effects of thermal stress on mRNA and protein expression of iNOS, eNOS and cNOS of goats *in vivo* during winter, moderate and summer season revealed that there is a higher relative mRNA expression of iNOS, eNos and cNOS during summer [105]. The interaction between nitric oxide and HSPs plays an important role in the adaptive enhancement of resistance to thermal stress. iNOS protects the heart from myocardial ischaemia occurring due to heat stress [106].

Role of interleukins in combating heat stress

Interleukins are a group of cytokines (secreted proteins and signal molecules) that were first seen to be expressed by white blood cells (leukocytes) [107]. The function of the immune system depends in a large part on interleukins, and rare deficiencies of a number of them have been described, all featuring autoimmune diseases or immune deficiency. The majority of interleukins are synthesized by helper CD4 T lymphocytes, as well as through monocytes, macrophages, and endothelial cells. They promote the development and differentiation of T and B lymphocytes, and hematopoietic cells.

T lymphocytes regulate the growth and differentiation of T cells and certain B cells through the release of secreted protein factors [108]. These factors, which include interleukin 2 (IL2), are secreted by lectin- or antigen-stimulated T cells, and have various physiological effects. IL2 is a lymphokine that induces the proliferation of responsive T cells. In addition, it acts on some B cells, via receipt or specific binding [109] as a growth factor and antibody production stimulant [110]. The protein is secreted as a single glycosylated polypeptide, and cleavage of a signal sequence is required for its activity [109]. Solution NMR suggests that the structure of IL2 comprises a bundle of 4 helices (termed A-D), flanked by

2 shorter helices and several poorly defined loops. Residues in helix A, and in the loop region between helices A and B, are important for receptor binding. Secondary structure analysis has suggested similarity to IL4 and granulocyte-macrophage colony stimulating factor (GMCSF) [110].

Interleukin 6 (IL6), also referred to as B-cell stimulatory factor-2 (BSF-2) and interferon beta-2, is a cytokine involved in a wide variety of biological functions [111]. It plays an essential role in the final differentiation of B cells into immunoglobulin-secreting cells, as well as inducing myeloma/plasmacytoma growth, nerve cell differentiation, and, in hepatocytes, acute-phase reactants [111, 112]. A number of other cytokines may be grouped with IL6 on the basis of sequence similarity [111-113]. These include granulocyte colonystimulating factor (GCSF) and myelomonocytic growth factor (MGF). GCSF acts in hematopoiesis by affecting the production, differentiation, and function of 2 related white cell groups in the blood [113]. MGF also acts in hematopoiesis, stimulating proliferation and colony formation of normal and transformed avian cells of the myeloid lineage. Cytokines of the IL6/GCSF/MGF family are glycoproteins of about 170 to 180 amino acid residues that contain four conserved cysteine residues involved in two disulphide bonds [113]. They have a compact, globular fold (similar to other interleukins), stabilised by the two disulphide bonds. One half of the structure is dominated by a 4-alpha-helix bundle with a left-handed twist; the helices are anti-parallel, with two overhand connections, which fall into a double-stranded anti-parallel beta-sheet [114]. The fourth alphahelix is important to the biological activity of the molecule [112].

The relative mRNA expression of IL-2 and IL-6 was found significantly higher in winter as compared to summer in tropical as well as temperate region goats [115]. Similar type of findings was also observed by [116, 117] in studies on rats, which explained that under stress conditions, the HPA axis gets stimulated, which stimulated secretion of catecholamine and glucocorticoides, which modulate immune cells and thus cytokine production and modulation of cold stress occurs due to higher expression of proinflammatory cytokines like IL-2 and IL-6. [118] observed that acute heat stress enhanced the secretion of IL-2 by splenic lymphocytes significantly in broiler chickens. Many studies indicated that acute or chronic stress induces immunomodulatory effects in animal models. The decrease in IL-6 expression during summer may be due to inhibition of expression of IL-6, mediated through activating transcription factor 3 by heat shock factor 1 as observed in murine cells [119]. Thermoregulatory protective mechanism develops with age of animal and is well developed in adults as compared to infants and aged ones [120]. The higher expression of IL-2 and IL-6 in adults as compared to young ones suggests that these genes helps in thermal protection in better way in adults as compared to young ones.

Estimation of degree of heat stress / measure of heat load

The degree of heat stress experienced by animals is estimated by the temperature-humidity index (THI) that includes both ambient temperature and relative humidity [121]. Measuring the heat load imposed on an animal using air temperature (dry bulb temperature) can be misleading. A more useful measure is the wet bulb temperature, which takes relative humidity into account [122]. When the temperature is expressed in °C, the equation to determine THI for goats according to (121) is as follows: THI = Dry Bulb Temperature °C − {(0.31 − 0.31 Relative Humidity) (Dry Bulb Temperature °C − 14.4)}. The values obtained indicate the following: <22.2 = absence of heat stress; 22.2 to <23.3 = moderate heat stress; 23.3 to <25.6 = severe heat stress and ≥25.6 = extreme severe heat stress [121].

When temperature is measured in °F, the equation is as follows: THI = Dry Bulb Temperature °F − {(0.55 − 0.55 Relative Humidity) (Dry Bulb Temperature °F − 58)}. The obtained values indicate the following: values <82 = absence of heat stress; 82 to <84 = moderate heat stress; 84 to <86 = severe heat stress and ≥86 = extreme severe heat stress [123]. Comparisons based on THI between dairy goats and dairy cows revealed that goats are more adapted than cows to the effect of heat stress i.e., extreme heat stress: THI ≥ 84 for cows and ≥ 90 for goats [14]. It is worth to mention here that THI does not take into account the effect of radiation [35], and since most goat farming occur under grazing situations, THI poorly reflected heat stress. In fact, heat stress is much higher under grazing situations than as reflected by the THI.

Thermoregulatory and heat dissipating mechanisms by goats

Goats are homeotherms, and can maintain near constant body temperature under a wide range of environmental conditions [124]. Thermoregulation is the mean by which an animal maintains its body temperature and it involves a balance between heat gain and loss [125]. The range of temperature when the animal needs no additional energy to maintainits body temperature is called thermoneutral zone. Animals function most efficiently within their thermoneutral zone, while above the upper and below the lower critical temperatures, the animals are stressed and the environment constrains the production process. The thermoneutral zone is about 12–24°C for goats in the hot regions of the world [126, 127]. Heat stress occurs in animals when there is an imbalance between heat production within the body and its dissipation.

Most of the adjustments made by an animal involve dissipating heat to the environment and reducing the production of metabolic heat [11, 35]. Exposure of animals to heat stress leads to drastic changes, including a decrease in feed intake efficiency and utilization, disturbances in water, protein, energy and mineral balances, enzymatic reactions, hormonal secretions and blood metabolites, reduction in fecal and urinary water losses and an increase in sweating, respiration and heart rates [11].

Animals maintain their body temperature within tight limits over a wide range of ambient temperatures by balancing heat loss or gain and heat production [125]. Elevation of body temperature is the most obvious measure indicating that an animal is exposed to an unacceptable heat load [128]. The exposure of goats to elevated ambient temperatures induces an increase in the dissipation of excess body heat in order to negate the excessive heat load. Dissipation of excess body heat is excluded by evaporation of water from the respiratory tract and skin surface via panting and sweating, respectively. As ambient temperature approaches skin temperature, the rate of heat dissipation through sensible heat loss decreases [22]. As heat stress progresses, there is recruitment of evaporative processes, primarily sweating and increased respiration rate [25]. When heat stress becomes more severe, the depth of respiration increases back to near normal tidal volume while the respiration rate remains elevated above normal [122]. Evaporation of water requires a vapour pressure gradient for loss of heat energy as water evaporates to the surrounding air, but in very humid conditions this gradient is reduced, and therefore evaporative heat loss from the skin is reduced [28]. When the air temperature rises to near body temperature, this means that heat loss from panting also becomes limited [122]. [14] and [35] provided an integrative explanation of the ability of goats to survive and produce better than other ruminants in harsh environments: (a) skillful grazing behavior (they eat diets composed of browse [tree-leaves and shrubs]); (b) small body size; (c) high digestive efficiency; (d) low metabolic requirements; (e) ability to reduce metabolism; (f) efficiency of utilization of high fiber forage; (g) higher surface area of absorptive mucosa than in grass and roughage eaters; (h) capacity to increase substantially the volume of the foregut when fed high-fibrous food; (i) effective urea recycling to the rumen allowing goats to effectively digest low-protein feeds; (j) efficient use of water; and (k) large salivary glands. The characteristics of the outer surface of an animal's body are of great importance in the relationship between the animal and its ambient temperature. The first defense layer protecting animals from direct sunlight is provided by the coat, and this protection differs according to coat color, depth and length [11, 12]. Animals with a dark coat – and therefore with greater absorption of thermal radiation – are more susceptible to heat than those with a light colored coat at the same temperature [11]. White or light brown goats do better than dark brown or black goats [129, 50] mentioned that the thermal insulation increases by increasing the coat depth and attributed to the air space between the hair fibers. Carpet-type wool, as compared with denser wool types, seems to confer protection from solar radiation while at the same time allowing effective cutaneous evaporative cooling [30]. The long hair serves as an insulator from the heat, providing a hair buffer zone between the outer environment and the animal's body [12, 50] showed that long haired goats (130 mm) tolerated heat better than short haired goats (97.5 mm).

Strategies for alleviation of heat stress

From the above discussed sections, it could be concluded that the changes in the biological functions of goats due to exposure to heat stress include depression in feed intake, disturbance in the metabolism of water, protein, energy and mineral balances, enzymatic reactions, hormonal secretions and blood metabolites. Therefore, a variety of methods should be adopted by small ruminant producers/owners to overcome the negative effects of heat stress, including the use of shades, feeding and grazing strategies, providing water, handling time, the use of fans and evaporative cooling, and site selection of animals' housing [22, 130, 131].

Shade is the easiest method to reduce the impact of high solar radiation, and it is applicable under extensive conditions. The use of shades, fans or evaporative cooling is not possible in semi-intensive systems as goats are grazed in the open during most of the day, and this necessitates other strategies (i.e. portable shades) to counteract the adverse effects of heat stress [3]. Accessibility of animals to shade during summer is simple, easy, cheap and an efficient tool to minimize heat stress [11, 64]. Providing goats access to shade allows a reduction in rectal temperature and respiration rate in goats [132]. A well-designed shade structure reduces heat load by 30–50% [133]. Shelters do not need to be complicated or elaborate, trees and shrubs can serve as shelters for animals from solar radiation [134], and are usually the least-cost alternative. If natural shelter is not available, many goat producers use Quonset huts, plastic calf hutches, polydomes and/or carports to provide shelters for grazing animals. In addition, hay or straw shades, solid shade provided by sheet metal painted with white on top [135], and aluminum sheets [136] are the most effective and cheap materials.

Ration modifications can greatly help in reducing the negative effect of heat stress, and these adjustments may include changes in feeding schedules (feeding at cool hours, feeding intervals), grazing time, and ration composition such as dietary fiber adjustment, the use of high-quality fiber forage, increased energy density (supplemental protected fat) and use of feed additives [buffers (sodium bicarbonate), niacin, antioxidants and fungal culture (yeast culture)]. During summer, the feeding behavior for most of the animals changes and they tend to consume more feed during the cooler periods of the day [36]. Therefore, feeding animals during the cooler periods of the day encourages them to maintain their normal feed intake and prevents the co-occurrence of peak metabolic and climatic heat load [128]. Also, feeding animals at more frequent intervals helps to minimize the diurnal fluctuation in ruminal metabolites and increase feed utilization efficiency in the rumen [137]. Another point to be taken into account to alleviate heat stress is the grazing time. In extreme heat, animals decrease their grazing time and spend more time in the shade, especially during the heat of the day. They graze during the period of the milder weather during the day, i.e. before sunrise, at dawn and during the night [6]. Careful ration modifications during heat stress are important in achieving the optimum animal performance. Decreasing the forage to concentrate ratio can result in more digestible rations that may be consumed in greater amounts [138]. Feed containing low fiber rations during hot weather is logical since heat production is highly associated with metabolism of acetate compared with propionate [139]. More nutrient-dense diets are usually preferred during the heat stress period [36]. Dairy goats supplemented with 4% fat during summer had lower rectal temperature. Soybean oil fed to goats kept under heat stress increased milk fat content [24]. Feed additives have been proposed to offset the consequence of heat stress. For example, antioxidants such as vitamin C and E protect the body defense system against excessive production of free radicals (antioxidants are free radical scavengers) during heat stress and stabilize the health status of the animal [3, 140, 141] found that vitamin C supplementations to goats are effective in alleviating heat stress. Vitamin E and C supplementations decreased rectal temperature and respiration rate [66], and alleviated heat stress in goats [142]. One of the best practices to reduce heat stress is to provide adequate fresh and cool drinking water [11, 139]. The water requirements of goats increase under heat stress conditions, thus, it is essential that animals have a continuous access to adequate, clean, cool and fresh water. This is done by having adequate watering devices (making sure pressure is adequate to refill waterers), and providing more water sources in the pasture [139].

In addition, handling animals should be kept at minimum. Goats can be handled (i.e. milking, transportation) in the early morning or late evening time [130], and the afternoon work should be avoided when body temperature is already high. One of the effective methods for prevention of heat stress is to delay afternoon milking for 1-2 hours [139]). Also, it may be necessary to install fans or other cooling systems in barns and similar structures [139, 143] Cooling goats by spraying could reduce heat stress symptoms and improve animal welfare [24]. Direct wetting of animals is often used as an emergency measure and can be an effective protective method [134]. Sprayed and ventilated heat-stressed goats for 1 h/day consumed more feed (18%) and water (7%) and produced more milk (21%) [144]. The site selection of animals' housing is fundamental to decrease exposure and minimize the effect of heat stress. Proper selection of the housing site to emphasize factors for enhancing heat dissipation (minimal radiation, air temperature and humidity, and maximal air velocity) will have long-term protection benefits [143]. Fully enclosed shelters are not recommended for hot climates because of the decreased natural air velocity, therefore, it is preferred to use partially enclosed shelters [143].

Apart from nutritional and managemental strategies, genetic selection is one important aspect. Improvement of adaptability by simultaneous heterosis and crossbreeding is a better option in the hand of goat breeders for producing better offspring capable of withstanding heat stress [145]. Genetic variability for the response to climatic change can be used to select the most tolerant and robust animal to cope up with future climatic changes [146].

Conclusion

Livestock genetic improvement should take into account not only production traits (milk yield, weight gain and wool production), but also the interaction of those traits with the environmental factors (i.e. air temperature, relative humidity and solar radiation). Heat stress exerts negative effects on productivity and well-being in small ruminants. The exposure of goats toheat stress negatively affects biological functions, changes antioxidant levels and various hormones which are reflected in the impairment of their health, production and reproduction. Goats show various responses to heat stress at behavioural, physiological, molecular/cellular, haematological, biochemical and immunological levels. Heat stress adversely affects animals' comfort, water consumption, feed intake, milk yield and quality, meat quality, and reproduction and fertility. Thus, management strategies must be applied to counter hot/humid environmental conditions. Control is based on the provision of drinking water, adjustments in animals' diets during heat stress (such as increase in the energy density of rations, the use of feed additives, etc.), use of cooling mechanisms (shade and fans), the use of strategies to reduce the impact of heat stress on fertility (i.e. timed mating programs during summer), and scheduling animal activities in the early morning and evening when temperatures are not as extreme. For optimal results, the people who care for animals should have appropriate education and experience, understand the species requirements and have good observational skills. It is to be noted that awareness of heat stress is the first step towards its management. In addition, effective participation, coordination and active cooperation among scientists, technicians, meteorologists, veterinarians, nutritionists and local agricultural organizations are required to successfully include these factors as a basis for strategic and operational management decisions to improve production systems. There is a need to intensify agricultural extension staff/ farmer relationships and researcher/agricultural extension staff linkage to improve farmers' knowledge, skills and practices on the use of heat stress management techniques. Finally, it is hoped that this review serves as guidance to researchers and contributes to ongoing efforts to promote heat stress management, and therefore will contribute to agricultural sustainability.

References

1. Thornton PK. Livestock production: Recent trends, future prospects. Phil. Trans. R. Soc. B. 2010; 365:2853-2867.
2. Ben Salem H. Nutritional management to improve sheep and goat performances in semiarid regions. R. Bras. Zootec. 2010; 39:337-347.
3. Silanikove N, Leitner G, Merin U, Prosser CG. Recent advances in exploiting goat's milk: quality, safety and production aspects. Small Rumin. Res. 2010; 89:110-124.
4. Gupta M, Kumar S, Dangi SS, Jangir BL. Physiological, biochemical and molecular responses to thermal stress in goats. Intern. J Livest. Res. 2013; 3:27-38.
5. Zeder M. Domestication and early agriculture in the Mediterranean Basin: Origins, diffusion, and impact. PNAS. 2008; 105:11597-11604.
6. Dwyer CM. The behavior of sheep and goats. In: The ethology of domestic animals: an introductory text, Jensen P. (ed.). CABI, 2nd ed., 2009, 161-174.
7. Silanikove N, Leitner G, Merin U. The interrelationships between lactose intolerance and the modern dairy industry: global perspectives in evolutional and historical backgrounds. Nutrients. 2015; 7:7312-7331.
8. Ben Salem H, Smith T. Feeding strategies to increase small ruminant production in dry environments. Small Rumin. Res. 2008; 77:174-194.
9. Okoruwa MI. Effect of heat stress on thermoregulatory, live body weight and physiological responses of dwarf goats in southern Nigeria. Europ. Sci. J. 2014; 10:255-264.
10. Banerjee D, Upadhyay RC, Chaudhary UB, Kumar R, Singh S, Ashutosh GJM *et al*. Seasonal variation in expression pattern of genes under HSP70 family in heat- and cold-adapted goats (*Capra hircus*). Cell Stress Chap. 2014; 19:401-408.
11. Silanikove N. Effects of heat stress on the welfare of extensively managed domestic ruminants. Livest. Prod. Sci. 2000a; 67:1-18.
12. Jakper N, Kojo IA. Effect of coat colour, ecotype, location and sex on hair density of West African Dwarf (WAD) goats in Northern Ghana. Sky. J Agric. Res. 2014; 3:25-30.
13. Silanikove N. Effects of water scarcity and hot environment on appetite and digestion in ruminants: A review. Livest. Prod. Sci. 1992; 30:175-194.
14. Silanikove N, Koluman N. Impact of climate change on the dairy industry in temperate zones: predications on the overall negative impact and on the positive role of dairy goats in adaptation to earth warming. Small Rumin. Res. 2015; 123:27-34.
15. Nardone A, Ronchi B, Lacetera N, Ranieri MS, Bernabucci U. Effects of climate changes on animal production and sustainability of livestock systems. Livest. Sci. 2010; 130:57-69.
16. Al – Dawood A. Adoption of agricultural innovations: Investigating current status and barriers to adoption of heat stress management in small ruminants in Jordan. Amer.-Euras. J Agric. Environ. Sci. 2015; 15:388-398.
17. Collier RJ, Beede DK, Thatcher WW, Israel LA, Wilcox CJ. Influences of environment and its modification on dairy animal health and production. J Dairy. Sci. 1982; 65:2213-2227.
18. IPCC. Intergovernmental Panel on Climate Change. Climate change: Synthesis report summary for policymakers, 2014a

(https://www.ipcc.ch/pdf/assessment-report/ar5/syr/AR5_SYR_FINAL_SPM. pdf.).
19. IPCC. Intergovernmental Panel on Climate Change. Summary for policymakers. In: Climate Change 2014: Mitigation of Climate Change. Contribution of working group III to the fifth assessment report of the intergovernmental panel on climate change, Edenhofer O, Pichs-Madruga R, Sokona Y, Farahani E, Kadner S, Seyboth K *et al*. (eds). Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA 2014b, 30.
20. FAO. Adaptation to climate change in agriculture, forestry, and fisheries: Perspective, framework and priorities. FAO, Rome, 2007, 24.
21. Van Dijk J, Sargison ND, Kenyon F, Skuce PJ. Climate change and infectious disease: Helminthological challenges to farmed ruminants in temperate regions. Animals. 2010; 4:377-392.
22. Caulfield MP, Cambridge H, Foster SF, Mc Greevy PD. Review: Heat stress: a major contributor to poor animal welfare associated with long-haul live export voyages. Vet. J. 2014; 199:223-228.
23. St - Pierre N, Cobanov B, Schnitkey G. Economic losses from heat stress by US livestock industries. J Dairy Sci. 2003; 86(E-Suppl.):E52–E77.
24. Salama AAK, Hamzaoui S, Caja G. Responses of dairy goats to heat stress and strategies to alleviate its effects. Proc. XI International Conference on Goats, Gran Canaria, Spain. 2012, 15.
25. Mortola JP, Frappell PB. Ventilatory responses to changes in temperature in mammals and other vertebrates. Ann. Rev. Physiol. 2000; 62:847-874.
26. Facanha DAE, Oliveira MGC, Guilhermino MG, Costa WP, Paula VV. Hemogasometric parameters of Brazilian Native Goats under thermal stress conditions. Proc. XI International Conference on Goats, Gran Canaria, Spain, 2012b, 72.
27. Schaefer AL, Jones SD, Stanley RW. The use of electrolyte solutions for reducing transport stress. J. Anim. Sci. 1997; 75:258-265.
28. Barnes A, Beatty D, Taylor E, Stockman C, Maloney S, Mc Carthy M. Physiology of heat stress in cattle and sheep. Project number LIVE.209, Australia. Meat and Livestock Australia Limited, 2004, 35.
29. Hafez ESE. Behavioral adaptation. In: Adaptation of domestic animals, Hafez E.S.E. (ed.). Lea and Febiger, Philadelphia, PA., 1968, 202-214.
30. Cain JWIII, Krausman PR, Rosenstock SS, Turner JC. Mechanisms of thermoregulation and water balance in desert ungulates. Wildlife. Soc. Bull. 2006; 34:570-581.
31. Bligh J. Temperature regulation. In: Stress physiology in livestock. Basic Principles, Yousef M.K. (ed.). CRC Press Inc., Boca Raton, FL, USA. 1985; 1:75-96.
32. Stull CL. Stress and dairy calves. University of California, Davis, 1997.
33. Alam MM, Hashem MA, Rahman MM, Hossain MM, Haque MR, Sobhan Z *et al*. Effect of heat stress on behavior, physiological and blood parameters of goat. Prog. Agric. 2011; 22:37-45.
34. Shilja S, Sejian V, Bagath M, Mech A, David CG, Kurien EK *et al*. Adaptive capability as indicated by behavioral and physiological responses, plasma HSP70 level, and PBMC HSP70 mRNA expression in Osmanabadi goats subjected to combined (heat and nutritional) stressors. Int. J Biometeorol. 2015. DOI 10.1007/s00484-015-1124-5.
35. Silanikove N. The physiological basis of adaptation in goats to harsh environments. Small Rumin. Res. 2000b; 35:181-193.
36. West JW. Nutritional strategies for managing the heat stressed dairy cow. J Anim. Sci. 1999; 77:21-35.
37. Johnson HD. Physiological responses and productivity of cattle. In: Stress physiology in livestock, Yousef M.K. (ed.). Ungulates. CRC Press Inc., Boca Raton, FL, USA. 1985; II:3-24
38. Kadzere CT, Murphy MR, Silanikove N, Maltz E. Heat stress in lactating dairy cows: a review. Livest. Prod. Sci. 2002; 77:59-91.
39. NRC. Nutrient requirements of small ruminants, sheep, goats, cervids, and new world camelids. National Academy Press, Washington, DC, 2007, 384.
40. Hamzaoui S, Salama AAK, Albanell E, Such X, Caja G. Physiological responses and lactational performances of late-lactation dairy goats under heat stress conditions. J. Dairy Sci. 2013; 96:6355-6365.
41. Moore CE, Kay JK, Van Baale MJ, Baumgard LH. Calculating and improving energy balance during times of nutrient limitation. Proc. Southwest Nutrition and Management Conference, Tempe, Arizona 2005, 173-185.
42. Salama AAK, Caja G, Hamzaoui S, Badaoui B, Castro Costa A, Facanha DAE *et al*. Different levels of response to heat stress in dairy goats. Small Rumin. Res. 2014; 121:73-79.
43. Ocak S, Darcan N, Cankaya S, Inal TC. Physiological and biochemical responses in German fawn kids subjected to cooling treatments under Mediterranean climatic conditions. Turk. J Vet. Anim. Sci. 2009; 33:455-461.
44. Giger – Reverdin S, Gihad EA. Water metabolism and intake in goats. In: Goat nutrition, Morand-Fehr P. (ed.). Pudoc: Wageningen, Netherlands, 1991, 37-45.
45. Khan MS, Ghosh PK, Sasidharan TO. Effect of acute water restriction on plasma proteins and on blood and urinary electrolytes in Barmer goats of the Rajasthan desert. J Agri. Sci. Camb. 1978; 2:395-398.
46. Ahmed MM, El Kheir IM. Thermoregulation and water balance as affected by water and food restrictions in Sudanese desert goats fed good-quality and poor-quality diets. Trop. Anim. Health Prod. 2004; 36:191-204.
47. Hall DM, Buettner GR, Oberley LW, Xu L, Matthes RD, Gisolfi CV. Mechanisms of circulatory and intestinal barrier dysfunction during whole body hyperthermia. Am. J Physiol. Heart Circ. Physiol. 2001; 280:509-521.
48. Adedeji TA. Effect of some qualitative traits and non-genetic factors on heat tolerance attributes of extensively reared West African Dwarf (WAD) goats. Inter. J. Appl. Agric. Apicul. Res. 2012; 8:68-81.
49. Kadim IT, Mahgoub O, Al - Marzooqi W, Al - Ajmi DS, Al - Maqbali RS, Al - Lawati SM. The influence of seasonal temperatures on meat quality characteristics of hot-boned, m. psoas major and minor, from goats and sheep. Meat Sci. 2008; 80:210-215.
50. Helal A, Hashem ALS, Abdel - Fattah MS, El - Shaer HM. Effects of heat stress on coat characteristics and physiological responses of Balady and Damascus goats in Sinai, Egypt. Amer.-Euras. J Agric. Environ. Sci. 2010; 7:60-69.

51. Sharma S, Ramesh K, Hyder I, Uniyal S, Yadav VP, Panda RP *et al*. Effect of melatonin administration on thyroid hormones, cortisol and expression profile of heat shock proteins in goats (*Capra hircus*) exposed to heat stress. Small Rumin. Res. 2013; 112:216-223.
52. Devendra C. Goats. In: Bioclimatology and the adaptation of livestock, Johnson H.D. (eds). Elsevier, Amsterdam, The Netherlands, 1987, 157-168.
53. Phulia SK, Upadhyay RC, Jindal SK, Misra RP. Alteration in surface body temperature and physiological responses in Sirohi goats during day time in summer season. Indian Journal of Animal Science. 2010; 80(4):340-342.
54. Robertshaw D, Dmiel R. The effect of dehydration on the control of panting and sweating in the black Bedouin goat. Physiol. Zool. 1983; 56:412-418.
55. Facanha DAE, Sammichelli L, Bozzi R, Silva WST, Morais JHG, Lucena RMO *et al*. Performance of Brazilian native goats submitted to a mix supply under thermal stress conditions. Proc. XI International Conference on Goats, Gran Canaria, Spain. 2012a, 343.
56. Heath E, Olusanya S. Anatomy and physiology of tropical livestock. Intern. Tropical Agric. Series, Longman, 3rd ed. 1985, 138.
57. Aharoni Y, Brosh A, Kourilov P, Ariel A. The variability of the ratio of oxygen consumption to heart rate in cattle and sheep at different hours of the day and under different heat load conditions. Livest. Prod. Sci. 2003; 79:107-117.
58. Fecteau ME, White SL. Alteration in body temperature. In: Large animal internal medicine, Smith B.P. (ed.). Elsevier Health Sciences, 5th ed., 2014, 31-39.
59. Robinson NE. Thermoregulation. In: Textbook of veterinary physiology, Cunningham J.G. (ed.). WB Saunders, Philadelphia, PA. 2002, 533–542.
60. Wrotek SE, Kozak WE, Hess DC, Fagan SC. Treatment of fever after stroke: conflicting evidence. Pharmacotherapy. 2011; 31:1085-1091.
61. Bligh J. The relationship between the temperature in the rectum and of the blood in the bicarotid trunk of the calf during exposure to heat stress. J Physiol. 1957; 36:393-403.
62. Shebaita MK, El - Banna IM. Heat load and heat dissipation in sheep and goats under environmental heat stress. Proc. VI International Conference on Animal and Poultry Production, University of Zagazig, Zagazig, Egypt, 1982, 459-469.
63. Fasoro BF. Heat stress index in three breeds of goats. Agric. Project, Dept. of Animal Breeding and Genetics, University of Agriculture, Abeokuta, Nigeria, 1999,6-28.
64. A l - Tamimi HJ. Thermoregulatory response of goat kids subjected to heat stress. Small Rumin. Res. 2007; 7:280-285.
65. Mc Dowell RE, Woodward A. Concepts in animal adaptation. comparative suitability of goats, sheep and cattle two tropical environments. Proceedings 3rd Int. Conf. on Goat Production and Disease, 10-15 January 1982, Tucson, USA. 1982, 384-393.
66. Sivakumar AVN. Singh G, Varshney VP. Antioxidants supplementation on acid base balance during heat stress in goats. Asian-Aust. J Anim. Sci. 2010; 23:1462-1468.
67. Leonart MS, Weffort Santos AM, Munoz EM, Higuti IH, Fortes VA, Nascimento AJ. Effect of vitamin E on red blood cell preservation. Braz. J Med. Biol. Res. 1989; 22:85-86.
68. Houpt TR.. Water, electrolytes and acid base balance. In: Dukes' physiology of domestic animals, Swenson M.J. (ed.). Cornell University Press, Ithaca, NY, USA, 10th ed., 1989, 486-506.
69. Constable PD. Clinical assessment of acid-base status: strong ion difference theory. Vet. Clin. North Am. Food Anim. Pract. 1999; 15:447-471.
70. Benjamin MM. Fluid and electrolytes. In: Outline of veterinary clinical pathology. Iowa State Univ. Press, Ames, USA, 1981, 213-238.
71. Sharma AK, Kataria N. Effects of extreme hot climate on liver and serum enzymes in Marwari goat. Indian J. Anim. Sci. 2011; 81:293-295.
72. Dangi SS, Gupta M, Maurya D, Ya dav VP, Panda RP, Singh G *et al.* Expression profile of HSP genes during different seasons in goats (*Capra hircus*). Trop. Anim. Health Prod. 2012; 44:1905-1912.
73. Mohamad SS. Effect of level of feeding and season on rectal temperature and blood metabolites in desert rams. Acad. J Nutr. 2012; 1:14-18.
74. Sejian V, Srivastava RS. Effects of melatonin on adrenal cortical functions of Indian goats under thermal stress. Vet. Med. Int., 2010. doi: 10.4061/2010/348919.
75. Salama AAK, Caja G, Hamzaoui S, Badaoui B, Castro Costa A, Facanha DAE *et al*. Different levels of response to heat stress in dairy goats. Small Rumin. Res. 2014; 121:73-79.
76. Minton JE. Function of hypothalamic pituitary adrenal axis and sympathetic nervous system in models of acute stress in domestic farm animals. J Anim. Sci. 1994; 72:1891-1898.
77. Rhoads RP, Baumgard LH, Suagee JK, Sanders SR. Nutritional interventions to alleviate the negative consequences of heat stress. Adv. Nutr. 2013; 4:267-276.
78. Kumar M, Jindal R, Nayyar S. Influence of heat stress on antioxidant status in Beetal goats. Indian J Small Rumin., 2011a; 17:178-181.
79. Kumar SBV, Kumar A, Kataria M. Effect of heat stress in tropical livestock and different strategies for its amelioration. J. Stress Physiol. Biochem 2011b; 7:45-54.
80. Lindquist S. The heat-shock response. Annu. Rev. Biochem. 1986; 55:1151-1191.
81. Caspani ML, Savioli M, Crotti S, Bruzzone P, Gattinoni L. Heat stress: characteristics, pathophysiology and avoidable mistakes. Minerva Anest. 2004; 70:617-624.
82. Roti RJL. Cellular responses to hyperthermia (40–46ºC): cell killing and molecular events. Int. J Hyperthermia. 2008; 24:3-15.
83. Henle KJ, Leeper DB. Effects of hyperthermia (45 degrees) on macromolecular synthesis in Chinese hamster ovary cells. Cancer Res. 1979; 39:2665-2674.
84. Collier RJ, Collier JL, Rhoads RP, Baumgard LH. Invited review: genes involved in the bovine heat stress response. J Dairy Sci. 2008; 91:445-454.
85. Baler R, Dahl G, Voellmy R. Activation of human heat shock genes is accompanied by oligomerization, modification, and rapid translocation of heat shock transcription factor HSF1. Mol. Cell. Biol. 1993; 13:2486-2496.
86. Westerheide SD, Morimoto RI. Heat shock response modulators as therapeutic tools for diseases of protein conformation. J Biol. Chem. 2005; 280:33097-33100.
87. Mathew A, Mathur SK, Morimoto RI. Heat shock

response and protein degradation: regulation of HSF2 by the ubiquitin-proteasome pathway. Mol. Cell. Biol. 1998; 18:5091-5098.
88. Parsell DA, Lindquist S. The function of heat-shock proteins in stress tolerance: degradation and reactivation of damaged proteins. Annu. Rev. Genet. 1993; 27:437–496.
89. Neuer A, Spandorfer SD, Giraldo P, Dieterle S, Rosenwaks Z, Witkin SS. The role of heat shock proteins in reproduction. Hum. Reprod. Update. 2000; 6:149-159.
90. Park SH, Lee SJ, Chung HY, Kim TH, Cho CK, Yoo SY *et al*. Inducible heat-shock protein 70 is involved in the radioadaptive response. Radiat Res. 2000; 153:318-326.
91. Feige U, Polla BS. Hsp70 – a multi-gene, multi-structure, multifunction family with potential clinical applications. Experientia. 1994; 50:979-986.
92. Volloch V, Rits S. A natural extracellular factor that induces Hsp72, inhibits apoptosis, and restores stress resistance in aged human cells. Exp. Cell. Res. 1999; 253:483-492.
93. Mizzen LA, Welch WJ. Characterization of the thermotolerant cell. I. Effects on protein synthesis activity and the regulation of heat-shock protein 70 expression. J Cell. Biol. 1988; 106:1105-1116.
94. Kelley KW. Immunological consequences of changing environmental stimuli. In: Animal stress, Moberg G.P. (ed.). Am. Physiol. Soc., Bethesda, MD, 1985, 193-223.
95. Lippolis JD. Immunological signaling networks: integrating the body’s immune response. J Anim. Sci., 2008; 86:53–63.
96. Sordillo LM, Contreras GA, Aitken SL. Metabolic factors affecting the inflammatory response of periparturient dairy cows. Anim. Health Res. Rev. 2009; 10:53-63.
97. Jain S, Gautam V, Naseem S. Acute-phase proteins: As diagnostic tool. J Pharm. Bioallied. Sci. 2011; 3:118-127.
98. Bernabucci U, Badirico L, Morera P, Lacetera N, Ronchi B, Nardone A. Heat Shock modulates adipokines expression in 3T3-L1 adipocytes. J Mol Endocrinol. 2009; 42:139-147.
99. Maurya D, Gupta M, Dangi SS, Yadav VP, Mahapatra RK, Sarkar M. Expression of gene associated with thermal stress in goats during different seasons. Indian J Anim Sci. 2013; 83(6):604-608.
100. Johnson HD. Bioclimate effects on growth, reproduction and milk production. In: Bioclimatology and the adaptation of livestock, Johnson H.D. (ed.). Elsevier Science, Amsterdam, The Netherlands, Part II, 1987, 3-16.
101. Grönlund U, Sandgren CH, Waller KP. Haptoglobin and serum amyloid A in milk from dairy cows with chronic sub-clinical mastitis. Vet. Res. 2005; 36:191-198.
102. Gonzalez FH, Tecles F, Martınez - Subiela S, Tvarijonaviciute A, Soler L, Cerón JJ. Acute phase protein response in goats. J Vet. Diagn. Invest. 2008; 20:580-584.
103. Petersen HH, Nielsen JP, Heegaard PM. Application of acute phase protein measurements in veterinary clinical chemistry. Vet. Res. 2004; 35:163-187.
104. Chatterjee A, Black SM, Catravas JD. Endothelial Nitric Oxide (No) and Its Pathophysiologic Regulation. Vascul Pharmacol. 2008; 49(4-6):134-140.
105. Yadav VP, Dangi SS, Chouhan VS, Gupta M, Dangi SK, Singh G *et al*. Expression Analysis of NOS Family and HSP Genes during Thermal Stress in Goat (Capra hircus). Int J Biometeorol. 2016; 60:381-389.
106. Arnaud C, Godin-Ribuot D, Bottari S, Peinnequin A, Joyeux M, Demenge P *et al*. iNOS is a mediator of the heat stress-induced preconditioning against myocardial infarction *in vivo* in the rat. Cardiovasc Res. 2003; 58:118-125.
107. Brocker C, Thompson D, Matsumoto A, Nebert DW, Vasiliou V. Evolutionary divergence and functions of the human interleukin (IL) gene family. Human Genomics. 2010; 5(1):30-55.
108. Yokota T, Arai N, Lee F, Rennick D, Mosmann T, Arai K. Use of a cDNA expression vector for isolation of mouse interleukin 2 cDNA clones: expression of T-cell growth-factor activity after transfection of monkey cells. Proceedings of the National Academy of Sciences of the United States of America. 1985; 82(1):68-72.
109. Cerretti DP, McKereghan K, Larsen A, Cantrell MA, Anderson D, Gillis S *et al*. Cloning, sequence, and expression of bovine interleukin 2. Proceedings of the National Academy of Sciences of the United States of America. 1986; 83(10):223-7.
110. Mott HR, Driscoll PC, Boyd J, Cooke RM, Weir MP, Campbell ID. Secondary structure of human interleukin 2 from 3D heteronuclear NMR experiments. Biochemistry. 1992; 31(33):7741-4.
111. Hirano T, Yasukawa K, Harada H, Taga T, Watanabe Y, Matsuda T *et al*. Complementary DNA for a novel human interleukin (BSF-2) that induces B lymphocytes to produce immunoglobulin. Nature. 1986; 324(6092):73-6.
112. Lütticken C, Krüttgen A, Möller C, Heinrich PC, Rose-John S. Evidence for the importance of a positive charge and an alpha-helical structure of the C-terminus for biological activity of human IL-6. FEBS Letters. 1991; 282(2):265-7.
113. Clogston CL, Boone TC, Crandall BC, Mendiaz EA, Lu HS. Disulfide structures of human interleukin-6 are similar to those of human granulocyte colony stimulating factor. Archives of Biochemistry and Biophysics. 1989; 272(1):144-51.
114. Walter MR, Cook WJ, Zhao BG, Cameron RP, Ealick SE, Walter RL *et al* Crystal structure of recombinant human interleukin-4. The Journal of Biological Chemistry. 1992; 267(28):20371–6.
115. Marai IF, Ayyat MS, Abd El - Monem UM. Growth performance and reproductive traits at first parity of New Zealand White female rabbits as affected by heat stress and its alleviation under Egyptian conditions. Trop. Anim. Health Prod. 2001; 33:451-462.
116. Sparke EJ, Young BA, Gaughan JB, Holt M, Goodwin PJ. Heat load in feedlot cattle. MLA Project FLOT. 307, 308, 309, Australia. Meat and Livestock Australia Limited, 2001, 34.
117. LPHSI. Livestock and poultry heat stress indices. Agriculture Engineering Technology Guide, Clemson University, Clemson, SC, USA, 1990.
118. Lefcourt AM, Adams WR. Radiotelemetric measurement of body temperature of feedlot steers during winter. J Anim. Sci. 1998; 76:1830-1837.
119. Crawshaw LI. Temperature regulation in vertebrates. Ann. Rev. Physiol. 1980; 42:473-491.
120. Nikitchenko IN, Plyaschenko SI, Zenkov AC. Stresses and productivity of farm animals. Urajai Publishing House, Minsk, 1988, 200.

121. Mishra RP. Role of housing and management in improving productivity efficiency of goats. In: Goat production-processing of milk and meat. Central Institute for Research on Goats (CIRG), India, 1st ed., 2009, 45.
122. Mader TL, Davis MS. Effect of management strategies on reducing heat stress of feedlot cattle: feed and water intake. J Anim. Sci. 2004; 82:3077-3087.
123. Acharya RM, Gupta UD, Sehgal JP, Singh M. Coat characteristics of goats in relation to heat tolerance in the hot tropics. Small Rumin. Res. 1995; 18:245-248.
124. Morrison SR. Ruminant heat stress: effect on production and means of alleviation. J Anim. Sci. 1983; 57:1594-1600
125. Al - Dawood A. Adoption of agricultural innovations: Investigating current status and barriers to adoption of heat stress management in small ruminants in Jordan. Amer.-Euras. J Agric. Environ. Sci. 2015; 15:388-398.
126. Hammadi M, Fehem A, Harrabi H, Ayeb N, Khorchani T, Salama AAK *et al*. Shading effects on respiratory rate and rectal temperature in Tunisian local goat kids during summer season. Proc. XI International Conference on Goats, Gran Canaria, Spain, 2012, 127.
127. Muller CJC, Botha JA, Coetzer WA, Smith WA. Effect of shade on various parameters of Friesian cows in a Mediterranean climate in South Africa. 2. Physiological responses. South Afric. J Anim. Sci. 1994; 24:56-60.
128. Onyewotu LOZ, Stigter CJ, Abdullahi AM, Ariyo JA, Oladipo EO, Owonubi JJ. Reclamation of desertified farmlands and consequences for its farmers in semiarid northern Nigeria: A case study of Yambawa rehabilitation scheme. Arid Land Res. Manage. 2003; 17:85-101.
129. Bond TE, Kelly CF, Garrett WN, Hahn L. Evaluation of materials for livestock shades. Calif. Agric, 1961; 15:7-8.
130. Bond TE, Morrison SR, Givens RL. Influence of surroundings on radiant heat load of animals. Trans. ASAE. 1969; 12:246-248.
131. Soto- Navarro SA, Krehbiel CR, Duff GC, Galyean ML, Brown MS. Steiner RL. Influence of feed intake fluctuation and frequency of feeding on nutrient digestion, digesta kinetics, and ruminal fermentation profiles in limit-fed steers. J Anim. Sci. 2000; 78:2215-2222.
132. Aggarwal A, Upadhyay R. Heat stress and animal productivity. Springer India, 2013, 188.
133. Atrian P, Shahryar HA. Heat stress in dairy cows. Res. Zool. 2012; 2:31-37.
134. Ayo JO, Minka NS, Mamman M. x Excitability scores of goats administered ascorbic acid and transported during hot-dry conditions. J Vet. Sci. 2012; 7:127-131.
135. Ghanem AM, Jaber LS, Abi - Said M, Barbour EK, Hamadeh SK. Physiological and chemical responses in water deprived Awassi ewes treated with vitamin C. J Arid Environ. 2008; 72:141-149.
136. Kobeisy S. Effect of vitamin C and E on rectal temperature and respiratory rates in heat stressed goats. Assiut. Vet. Med. J. 1997; 37:120-132.
137. Sejian V, Hyder I, Malik PK, Soren NM, Mech A, Mishra A *et al.* Strategies for alleviating abiotic stress in livestock. In: Livestock production and climate change, Malik P.K, Bhatta R., Takahashi J., Kohn R., Prasad C.S. (eds). CAB International, 2015, 25-60.
138. Darcan N, Güney O. Alleviation of climatic stress of dairy goats in Mediterranean climate. Small Rumin. Res. 2008; 74:212-215.
139. Samara EM, Abdoun KA, Okab AB, Al-Badwi MA, El-Zarei MF, Al-Saef AM *et al*. Assessment of heat tolerance and production performance of Aradi, Damascus, and their crossbred goats. Int J Biometeorol. 2016; 60:1377-1387.
140. Menendez-Buxadera A, Seradilla JM, Arrebola F, Clemente I, Castro JA, Osorio J *et al*. Genetic variations for tolerance to heat stress in dairy small ruminants: Results obtained in Spain. FAO-CIHEAM Network on sheep and Goats. 8th International seminar. Tangier, Morocco, 11-13th June, 2013.