**Heat stress in cattle**

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**Introduction**

In the article published by Scholtz et al in 2013 it is suggested that global warming may have an effect on beef production particularly in the southern hemisphere where apparently the effects are likely to be more extreme.

Rust and Rust (2013) have speculated that the effects of climate change in South Africa will include a number of issues including rising feed cost due to less production as well as animal performance losses in weight gains, milk yield and reproductive performance. Incidentally they also suggest that vector borne diseases and internal parasites may increase in prevalence.

Du Preez et al (1990) were describing temperature humidity index (THI) values applicable to South Africa and Namibia in 1990 by means of computerized modelling and mapping techniques. Even at that time it is apparent that thermal conditions could be a constraint on the performance of farm animals, particularly in high-yielding dairy cows.

Heat stress has thus been previously described and with indications of global warming and more extreme weather conditions the livestock industry including sale yards, transport and production areas may become more affected. Welfare considerations should also be part of this focus of attention.

Twentieth century paradigms have been updated with newer concepts of heat stress or heat loading and the resulting pathophysiology are being described in the literature. This article attempts to summarize some of the aspects of this subject for the reader. This article is primarily with regard to beef cattle but much of the information may be adjusted or applied to dairy cattle.

**Terminology**

The following definitions and descriptions will assist with terminology:
Temperature humidity index

An index combining temperature and humidity (THI) has been used for some years to assess heat stress in cattle. However, according to Gaughan et al (2008) the THI neither includes important climatic variables such as solar radiation and wind speed (WS, m/s) nor does it include management factors (the effect of shade) or animal factors (genotype differences).

Gaughan et al (2008) suggest, also, that current indices do not account for the cumulative effects of heat load, natural cooling, or both. Cattle may accumulate heat during the day (the body temperature rises) and dissipate the heat at night. If there is insufficient night cooling, cattle may enter the following day with an accumulated heat load (AHL). The THI-hours model was developed to account for the impact of intensity × duration on thermal status and similarly models were developed using combinations of the maximum THI, daily duration of heat stress, and a heat load index (HLI).

Consequently Gaughan developed and validated a new HLI for cattle based on respiratory dynamics and tympanic temperature. Heat load thresholds were also determined for different genotypes, and an AHL model was developed to predict the heat balance of cattle.

Heat load index

The Gaughan HLI incorporating black globe (BG) temperature (°C), relative humidity (RH, decimal form), and WS was initially developed by using the panting score (PS) of 2,490 Angus feedlot steers. The HLI consists of 2 parts based on a BG temperature threshold of 25°C:

\[ \text{HLI}_{BG>25} = 8.62 + (0.38 \times \text{RH}) + (1.55 \times \text{BG}) - (0.5 \times \text{WS}) + e^{(2.4-\text{WS})}, \]
\[ \text{HLI}_{BG<25} = 10.66 + (0.28 \times \text{RH}) + (1.3 \times \text{BG}) - \text{WS}. \]

Where e is the base of the natural logarithm or 2.71828.

Black globe temperature is measured using a 150 mm (6 inch) black globe (usually copper) with a thermometer located at the center and located in the sun thus representing the integrated effects of radiation and wind.

A threshold HLI above which cattle of different genotypes gain body heat was developed for 7 genotypes. The threshold for unshaded black B. Taurus steers was 86, and for unshaded B. indicus (100%) the threshold was 96. Threshold adjustments were developed for factors such as coat colour, health status, access to shade, drinking water temperature and manure management. Upward and downward adjustments are possible; upward adjustments occur when cattle have access to shade (+3 to +7) and downward adjustments occur when cattle are showing clinical signs of disease (−5).

Accumulated heat load index

The AHL as defined by Gaughan is a 2-dimensional function incorporating time and animal heat balance (The amount of time the animal is exposed to a HLI above its upper threshold). When this occurs, the animal is not dissipating sufficient body heat into the environment and therefore core body temperature increases above its normal range. Alternatively, if the HLI falls below the upper threshold, then the animal is able to dissipate body heat into the environment, and core body temperature will return to the normal range. The threshold value is genotype specific and is
also affected by management factors such as access to shade and drinking water temperature. The upper threshold was defined as the HLI, where \( \geq 20\% \) of unshaded cattle had a panting score of \( \geq 1 \).

The HLI and the AHL were successful in predicting panting score (PS) responses of different cattle genotypes during periods of high heat load thus validating the measures. The reciprocal may therefore be true that accurate assessment of panting scores can predict HLI and AHL.

**Panting Scores**
A number of authors have described panting scores for quantifying heat stress in cattle. Meat and Livestock Australia (MLA) have presented guidelines for this as shown below:

<table>
<thead>
<tr>
<th>Panting score</th>
<th>Breaths/minute</th>
<th>Breathing condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>&lt; 40</td>
<td>Normal with no panting. Difficult to see chest movement.</td>
</tr>
<tr>
<td>1</td>
<td>40–70</td>
<td>Slight panting, mouth closed with no salivation. Easy to see chest movement.</td>
</tr>
<tr>
<td>2</td>
<td>70–120</td>
<td>Fast panting with salivation present. No open mouth panting.</td>
</tr>
<tr>
<td>2.5</td>
<td>70–120</td>
<td>As for 2 but with occasional open mouth panting. Tongue not extended.</td>
</tr>
<tr>
<td>3</td>
<td>120–160</td>
<td>Open mouth panting and some drooling. Neck extended and head usually up.</td>
</tr>
<tr>
<td>3.5</td>
<td>120–160</td>
<td>As for 3 but with tongue out slightly, occasionally fully extended for short periods and excessive drooling.</td>
</tr>
<tr>
<td>4</td>
<td>&gt; 160</td>
<td>Open mouth with tongue fully extended for prolonged periods and excessive drooling.</td>
</tr>
<tr>
<td>4.5</td>
<td>Variable</td>
<td>As for 4 but head down. Cattle breathe from flank. Drooling may cease</td>
</tr>
</tbody>
</table>

According to MLA If more than 10\% of the cattle exhibit panting scores of 2 or more, cattle handling should cease and only resume when conditions become cooler and cattle have returned to normal.

- Cattle with panting scores of 3.5 or more are in danger of death.
- If more than 10\% of the cattle exhibit panting scores of 3.5 or more, there is potential for a serious problem to develop unless measures are taken to cool the stock.
- The transition from 2.5 to 4.5 can happen quickly, in less than 2 h, under extreme conditions.

**Symptoms and pathophysiology of heat stress**

Heat stress affects livestock productivity in a number of ways including reduced feed intake, growth rate, milk production, fertility, embryo survival and increased susceptibility to disease
and parasites. There are also welfare issues concerning handling and management of livestock under heat loading conditions. As heat stress levels increase and more obvious clinical signs are seen, dry matter intake will be reduced, panting scores increase, water intake is increased and animals may seek shade. Ultimately animals may start to tremble, become uncoordinated, go down and death may follow.

Cronje (2005) published an extensive article on heat stress in cattle and salient points from this article are highlighted below:

Traditionally heat exhaustion was considered to be a result of either direct thermal damage to the brain or circulatory failure. This is consistent with the sudden rise in body temperature and drop in blood pressure that precede collapse from heat stroke, and with other symptoms such as delirium, convulsions and coma.

However, they do not adequately explain all of the pathophysiologic changes such as a systemic elevation of inflammatory cytokines, widely disseminated intravascular blood coagulation, kidney failure and injury to the liver and pancreas which are more consistent with multiple organ failure and sepsis than with brain damage or acute circulatory failure. Two of the pathologies least consistent with traditional theories are haemorrhage of the gut mucosa and peritoneum, and liver. In addition the delayed effect of heat associated deaths reported by some authors is difficult to explain with traditional theories.

According to Cronje there is mounting evidence that damage to the lining of the gut may be the source of a cascade of events leading to organ damage or failure through endotoxins released from the gut.

This progression of events begins with a redistribution of blood flow when heat load directs blood flow to the skin at the expense of blood supply to the gut. During heat load, blood flow to the gut is reported to be reduced by 40–50% and the reduction in visceral blood supply causes a substantial decrease in the supply of oxygen and nutrients and removal of waste products from these tissues. This condition is known as ischemia.

All cells have metabolic strategies that enable them to cope with limited periods of ischemia but they will incur structural damage if ischemia is prolonged. This leads to loss of function and ultimately to cell death (necrosis). The gut is particularly vulnerable to ischemia because it is one of the most metabolically demanding tissues of the body. There is now substantial evidence showing that hyperthermia and the reduction in blood supply to the viscera during hyperthermia result in damage to the cells of the gut particularly the villi of the intestines where increased permeability occurs.

The increased cellular permeability was attributed to reactive oxygen species (ROS) generated because of oxidative stress and depletion of ATP. ROS are a class of powerful oxidants that remove electrons from molecules, initiating destructive chain reactions that impair cell structure and membrane integrity. One of the main ways in which ROS are thought to exert their destructive effects is by impairing cellular ionic homeostasis. The enzyme, calcium ATPase is responsible for maintaining the intracellular Ca++ concentration at a level that is typically 10
times less than that outside the cell. This enzyme, however, contains thiol groups, which are susceptible to attack by ROS. Loss of calcium ATPase activity results in an increase in intracellular Ca++ which, in turn, activates proteases that attack the cytoskeleton of the cell. Under normal conditions, the membranes of cells lining the gut are impermeable to endotoxin and the cells themselves are joined together by ‘tight junctions’, effectively forming a barrier that prevents endotoxin from entering the body. Endotoxin is a lipopolysaccharide outer membrane of most gram-negative bacteria that inhabit the gut, and have exceedingly toxic effects when it enters the body.

When cells lining the gut are damaged this barrier is compromised and endotoxin invades the body where they induce the release of a variety of cytokines from the cells of the gut and liver. Cytokines are hormone-like chemical messengers that facilitate communication between cells responsible for initiating the acute phase immune response. Under normal conditions, this immune response promotes tissue repair by recruiting host defence mechanisms and facilitating access to the site of tissue damage.

Endotoxins, however, stimulate an inappropriate response in which there is an imbalance between inflammatory and anti-inflammatory cytokines which may result in either immunosuppression or inflammation. The inflammatory cytokines, tumour necrosis factor (TNF) and interleukin–1 (IL–1), are expressed at high levels, and are capable of particularly harmful effects. IL–1 may induce fever, depress appetite, stimulate bone resorption and cause capillaries to leak, while TNF causes cell death and blood clotting. Proteases that are activated by the influx of Ca++ ions during ischemia also convert the enzyme, xanthine dehydrogenase, to xanthine oxidase. In the presence of oxygen, xanthine oxidase converts the products of ATP depletion, xanthine and hypoxanthine, to a highly toxic species of ROS (reactive oxygen species), which damages cells.

In the ischemic cell, this enzyme and its substrate accumulate since the reaction is constrained by a lack of oxygen. However, subsequent resumption of blood flow to the gut (and hence oxygen supply) activates xanthine oxidase, releasing a flood of ROS. In addition one of the effects of endotoxin is to stimulate the release of nitric oxide (NO) which is a potent vasodilator. This together with the flood of ROS is known as reperfusion injury, and may be even more destructive than ischemia, since it not only aggravates damage at the site of the ischaemia but also causes damage throughout the body.

The endotoxin–mediated release of NO causes vasodilation of the visceral vasculature and since peripheral vasodilation is also present during heat load, this results in a sharp decrease in blood pressure, provoking an abrupt rise in heart rate and collapse. This is known as septic shock. Splanchnic vasodilation also facilitates the spread of endotoxin to other tissues of the body where further tissue damage is propagated by cytokine induction.

Pyrogenic cytokines acting on the thermoregulatory centre of the brain to induce a fever–like state which is thought to be the reason for the sudden rise in body temperature that precedes collapse from heat stroke. Other cytokines such as TNF and IL–1 are thought to be responsible for systemic damage to other tissues such as the brain, kidneys and lining of the vasculature, thus
providing an explanation for the observation that heat–induced tissue damage often persists after body temperature has been returned to normal.

The body responds to a variety of insults that damage intracellular proteins by producing heat shock proteins which tend to stabilize proteins, protecting them against denaturation, and refold denatured proteins, restoring their functionality. Exposure to heat induces production of heat shock protein–70 (HSP–70) in the gut, liver and other tissues. HSP–70 attenuates heat–induced increases in membrane permeability and increases the resilience of cells to endotoxin and ROS. In addition, heat shock proteins inhibit the production of pro–inflammatory cytokines, up–regulate the expression of anti–inflammatory cytokines and induce protective growth factor responses that promote cell–healing. It has also been suggested that heat shock proteins assist with the maintenance of intracellular ion pumps by transporting key proteins. The beneficial effects of HSP are numerous supports the notion that protein denaturation is a critical component of heat injury.

The extension of the newer concepts in heat stress may have considerable relevance in livestock and Cronje considers it reasonable to assume that hyperthermia would result in damage to the cells of the digestive tract of livestock species. Evidence of macroscopic congestion of the mucous membranes of the intestine in cattle that died of heatstroke have been reported indicating that hyperthermia results in damage to the gut. The reported loss of bowel movements in hyperthermic cattle is suggestive of gut damage and this may be more severe in the rumen which is populated with predominantly potentially endotoxin producing gram negative bacteria. It has been established that intravenous injection with rumen bacterial endotoxin induces severe endotoxic shock in calves. The pathology that was observed in these endotoxic calves included haemorrhages and disseminated intravascular coagulation—symptoms that have also been observed in hyperthermic cattle and sheep where disseminated intravascular coagulation, renal failure, and myocardial necrosis have been reported as complications of hyperthermia in livestock species.

Cronje has reported a modest elevation of HSP concentrations during the heat load phase and a far greater increase subsequent to heat exposure and suggests that the increase in HSP concentrations that were observed following hyperthermia is indicative of tissue injury caused by reperfusion of the gut.

**Implications in the cattle industry:**

Rumen acidosis is common in feedlot and dairy animals fed diets that contain high levels of grain. Considerable quantities of endotoxin accumulate in the rumen under conditions conductive to the development of acidosis. Furthermore, acidosis damages the rumen epithelium. Grain–based diets can double the osmolality of rumen digesta, leading to a rise in the osmotic pressure gradient between the gut circulation and the rumen contents. This results in rapid movement of water from the blood across the rumen epithelium, causing epithelial cells to separate from the basement membrane. The resulting necrosis affords endotoxin and bacteria entry to the body. Liver abscesses in feedlot cattle may be a direct result of the entry of endotoxin and bacteria through a damaged rumen epithelial lining.
Although ruminants reduce their feed intake during hot weather, passage rate through the digestive tract is also reduced; the net result is that rumen acid production per unit of feed increases and rumen pH decreases. In addition to this, acidosis is associated with an increased level of lactic acid in the blood and dehydration; both these conditions would reduce resilience to heat stress.

Disruption of feeding patterns by changes associated with weather patterns have been implicated as a factor in acidosis, and feedlot deaths and reports of “sudden death” by Glock and DeGroot (1998) in feedlots related to digestive causes have been difficult to completely explain.

According to Shearer (2005) the primary avenues for heat loss during periods of hot weather are sweating and panting. In severe heat, panting progresses to open-mouth breathing characterized by a lower respiratory rate and greater tidal volume. The result is respiratory alkalosis as a result of the increased loss of carbon dioxide. The cow compensates by increasing urinary output of bicarbonate (HCO$_3^-$). Simultaneously, the salivary HCO$_3^-$ pool for rumen buffering is decreased by the loss of saliva from drooling in severely stressed cows. Water deprivation may contribute significantly to reducing saliva flow. The end result is rumen acidosis because of reduced rumen buffering and an overall reduction in total buffering capacity. Rumen pH is largely determined by the balance between the acids generated from the fermentation of feedstuffs and the bicarbonate and phosphate buffers in saliva which neutralize these acids. Physically effective fiber stimulates chewing and chewing stimulates saliva secretion. Consequently, consistent intake of feedstuffs with effective fiber and cud-chewing are essential for rumen buffering.

Saliva flow rates in beef and dairy cattle are estimated to be in the range of 108 to 308 liters (28 to 81 gallons) per day. At these rates of saliva flow it is estimated that the cow can contribute in the range of 390 to 1115 grams (.86 to 2.5 lb) of disodium phosphate and 1134 to 3234 grams (2.5 to 7.1 lb) of sodium bicarbonate for rumen buffering daily. Reduced feed intake, a preference for concentrates rather than forage, a loss of salivary buffering from increased respiratory rates and drooling, and a reduction in the total buffering pool all contribute to a greater potential for rumen acidosis during periods of hot and humid weather, and may explain in part, why some herds experience more acidosis and lameness despite being fed properly formulated rations.

The effect of ambient air temperature on rumen pH was evaluated in lactating Holstein cows fed either a high roughage or high concentrate diet in both a cool (65°F with 50% relative humidity) and a hot (85°F with 85% relative humidity) environment. Rumen pH was lower in cows exposed to the higher temperatures and those fed the higher concentrate diets.

Sudden death in ruminants as defined by O’Toole as an unexplained death in apparently healthy animals within 12 to 24 hours is an occurrence that may be seen in intensive livestock industries. Such deaths probably involve a complex interaction of animal, environmental factors, infectious including endotoxic agents and management. Heat loading may contribute to these deaths and should be kept in mind by investigating veterinarians.
Strategies for reducing heat stress in beef cattle

Lunn in a Shur Gain bulleting made the following recommendations:

Water:
Supplying cool, fresh, clean water is the most critical component to keeping cattle cool during hot weather. Water is important in regulating body temperature. Cattle drinking more water during hot weather are more likely to maintain normal body temperatures. Water requirements for heat stressed cattle can increase by up to 2x compared with non-heat stressed animals.

Increasing water consumption during hot weather will also improve DMI and will increase urine production and subsequently increase losses of some minerals (sodium, potassium and magnesium). Higher levels of these minerals should be provided in the diet to compensate for his loss. During normal weather conditions there should be a minimum of 0.1 to 0.2 m²/25 head of water surface area available. During hot weather additional water bowls and troughs should be available to allow for higher water intakes. Wetting down cattle is another option that has been proposed for keeping cattle cool but may also increase the humidity levels and aggravate a situation.

Handling Cattle:
Sorting and handling cattle during hot weather can increase their body temperature and heat load. It is not recommended to handle cattle during the hottest part of the day.

Feeding Patterns and Ration Changes:
Feeding cattle less in the morning and more in the evening can help keep cattle on feed and even out eating patterns. Cattle consuming more feed during the evening and night and less during the day may help reduce metabolic heat production during the hottest time of the day. Restricted feeding programs (such as slick bunk management) may also reduce metabolic heat production. Changing the ration itself may also reduce heat stress in cattle. The heat of digestion of forages is higher than that of grains. Animals on a higher forage ration are more inclined to heat stress than animals on higher grain diets. Changing nutrient densities in rations must be carefully considered due to possible reduced performance and possible aggravation of acidosis consequences. Feed additives may improve animal performance and reduce mortality during heat stress situations.

Air Flow:
Wind breaks can be detrimental to cattle in the summer. Increasing airflow will help keep cattle cool. House animals most susceptible to heat stress in areas with better airflow. Increasing pen and truck space (if cattle are being shipped during hot weather) should also be considered. Overcrowding increases the risk for animal mortality.

Provide Shade for Cattle:
Providing shade for cattle can also help reduce heat stress due to reduced exposure to solar radiation and thus reducing heat load on the animal. Providing shade has been shown to improve ADG and reduce death losses in feedlot cattle.
Conclusion:

With global warming and climate change being a topic of conversation, veterinary practitioners are encouraged to be aware of the possible changing situation and effects in livestock husbandry and react timeously to possible health and welfare issues.

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