

HEAT STRESS IN DAIRY CATTLE

A review, and some of the potential risks associated with the nutritional management of this condition

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Abstract

Heat stress occurs when animals are exposed to environmental temperatures in excess of 25°C (the upper critical temperature), particularly in combination with high relative humidity or sunshine. High humidity makes the sweating mechanism relatively ineffective, thereby making cattle unable to maintain their core body temperature. Affected cows attempt to reduce heat load by reducing exercise, feed intake and lactation. They actively seek shade and wet areas. As their body temperature rises animals become agitated and distressed, have laboured open-mouth breathing and eventually collapse, convulse and die. Heat stress that is not life-threatening leads to reduced milk production and impaired reproductive performance, and may predispose amongst others to subclinical acidosis. Treatment of severely affected animals is by cooling with cold water and/or fans. Prevention is by providing good-quality drinking water and shade (natural or artificial), and the use of water sprinklers and/or fans. Changes to the diet (i.e. high energy density and low protein) are also beneficial and often implemented. However, there may be some potential risks associated with the nutritional management of heat stress in dairy cattle; i.e. the animals are at increased risk of developing subacute rumen acidosis, with ensuing laminitis/lameness, and displaced abomasum. The first part of this paper provides a brief review of heat stress in dairy cattle. The second part discusses how increasing the energy density of the diet (i.e. increasing the grain/forage ratio), as part of the nutritional management of heat stress, may put the cows at greater risk of the above mentioned digestive disorders.

A brief review of heat stress in cattle

Introduction

Like all other mammals and birds, cattle are homeothermic animals. Despite wide fluctuations in environmental temperature, they are normally capable of maintaining a relatively constant body temperature (i.e. between 38.4 and 39.0°C), which is essential to preserve the multitude of biochemical reactions and physiological processes that occur with normal metabolism. As environmental temperatures increase, certain thermoregulatory responses (designed to stabilise body temperature) are initiated, including reduced feed intake, decreased activity and increased drinking, shade or wind seeking, increased peripheral blood flow, sweating and panting. However, these thermoregulatory activities may not be sufficient to maintain a normal body temperature during periods when ambient air temperature and humidity are particularly high.

¹ The information in this paper has been partly reproduced from: Parkinson TJ, Vermunt JJ, Malmo J. Diseases of Cattle in Australasia – A Comprehensive Textbook. The New Zealand Veterinary Association for Continuing Education (VetLearn™), Wellington, New Zealand, 2010

Hot weather conditions can have significant production effects on high producing dairy cattle during summer in Northern Australia. In these animals, high heat loads lead to

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depressed feed intake, decreased milk yield, milk fat and protein percentages, and elevated somatic cell counts (SCC). In severe conditions, immune function and animal welfare can also be compromised, unless the risk is wisely managed.

Environmental heat load has been described using various methods. However, since thermoregulation in cattle is affected largely by air temperature and relative humidity, the temperature humidity index (THI), which combines the effects of these parameters into one value, is the one most commonly used. Figure 1 shows THI values for a range of air temperature and relative humidity readings.

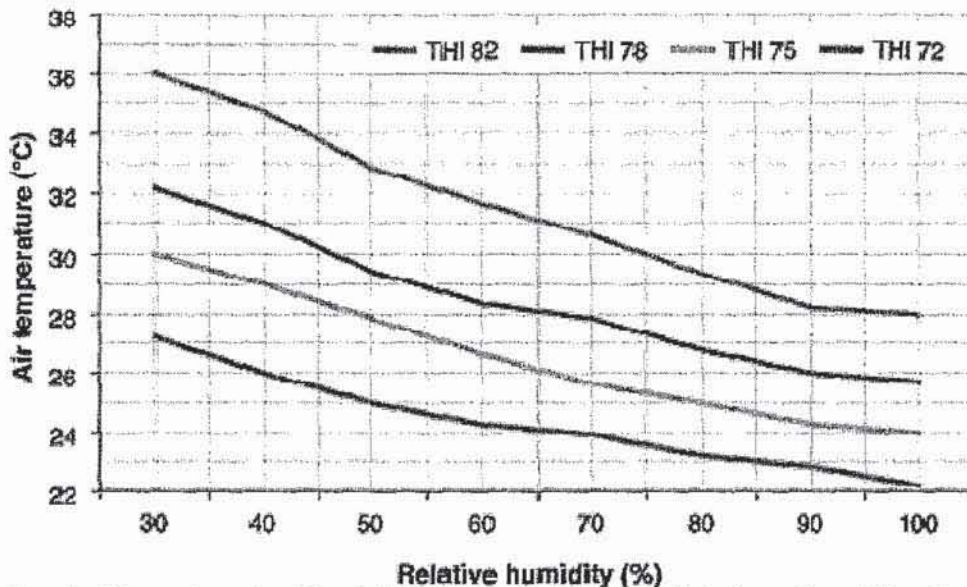


Fig 1. Temperature humidity index (THI) chart (downloaded from the Dairy Australia – Cool Cows website: <http://www.coolcows.com.au/go-on-alert/thi.htm>)

Factors other than air temperature and relative humidity can also impact heat stress. The presence of sunshine can add several degrees to the THI, whereas wind can lower it by a few points because it brings cooler air to the animal and carries away excess heat.

In dairy cattle, milk production is seriously affected once the THI rises above 78. As can be seen in this chart, a THI of 78 occurs at 31°C and 40% relative humidity, or 27°C and 80% relative humidity. Conception rates fall once THI is above 72, which is equivalent to 25°C at 50% relative humidity.

Aetiology and pathogenesis

For lactating dairy cattle, the most comfortable temperature range is between 5 and 25°C, also known as the thermal comfort zone. The lower critical temperature is the point at which an animal begins to feel cold and must increase its body heat production. It varies with age, physiological status (lactating or non-lactating), degree of insulation, level of milk production and acclimatisation. For example, a mature dairy cow in peak lactation may be quite comfortable at -25°C. The upper critical temperature is the point above which an animal begins to feel warm and must begin to compensate. Unlike the lower critical temperature it is constant at about 25°C, regardless of age or physiological status.

Total body heat load is a combination of heat derived from metabolism (internal body heat) and that obtained from environmental sources. In ruminants, the digestion of roughages produces a greater heat gain than that of concentrate feedstuffs. Other sources of internal body heat include those associated with daily physical activity and increased metabolic

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activity associated with lactational performance. The primary sources of environmental heat gain are solar radiation and high ambient air temperature. Black cattle absorb twice as much heat from the sun as white cattle. This is complicated by the fact that the flow of heat away from the animal's body is restricted by high ambient air temperature, which narrows the thermal gradient between the cow's body and the surrounding air.

The release of heat from any object into the environment is proportional to its exposed surface area. Further, the ratio of surface area to body mass decreases as overall size increases. Therefore, large cattle, such as adult cows, are at a disadvantage in losing excess body heat compared to calves; they are also at greater risk of becoming overheated.

Avenues for the dissipation of heat in cattle include non-evaporative and evaporative cooling mechanisms. Below 10°C, most heat loss from the body occurs by non-evaporative cooling (i.e. conduction, convection and radiation). However, when temperatures exceed 21°C, evaporative cooling (i.e. evaporation of water from the skin and respiratory tract) becomes the predominant mechanism of heat loss in cattle. *Bos indicus* cattle have larger and a greater number of sweat glands than *B. taurus* cattle; however, actual sweating rates are only slightly higher in the former. The evaporation of water from the cow's skin is a very effective cooling mechanism. It is enhanced by conditions that provide air movement, thereby moving water vapour away from the skin. The primary obstacle to evaporative cooling is high relative humidity, which in some environments may be exacerbated by limited air movement.

Normal body temperature is maintained by matching heat production with heat losses to the environment. When the amount of heat produced by the cow exceeds the amount released to the environment the body temperature of the cow rises. The cow responds physiologically by reducing activities that produce heat (feed intake, milk yield and exercise) and increasing those that shed heat (sweating, increased respiration and salivation). Some responses of cattle to heat stress, such as increased respiratory rate and panting, may actually increase heat production in their bodies. Thus, with increasing heat loads the cow experiences more distress, eventually with life threatening consequences.

Clinical signs

Heat stress in cattle results in a change of behaviour with signs of agitation and distress becoming more severe with increasing heat loads. Early signs of mild heat stress include an increased respiratory rate, elevated rectal temperature, a tendency to seek shade, refusal to lie down, increased water intake and reduced feed intake. As heat load increases cows will tend to crowd around water troughs, lie down in any wet areas (including any muddy areas around troughs), become agitated and restless and have laboured open-mouth breathing with excessive salivation. In severe cases, frothy discharge from the mouth or nose can be indicative of pulmonary oedema. At this stage, the animals' core body temperature commonly exceeds 41°C. With very severe heat stress cows become ataxic, refuse to move, collapse with convulsions, coma and eventually experience physiological failure, resulting in death.

Effects on production

Reduced feed intake is a primary strategy for lowering body heat production. The effect of reduced appetite and feed intake in heat-stressed cows will be an immediate fall in production with reduced weight gain or decreased milk yield (10-20% or more). Similar

effects on milk fat and protein yield are observed. Milk quality parameters are also affected, with SCC and bacterial counts commonly increased during periods of hot, humid weather. Higher producing animals that have the highest feed intake typically are the most severely affected. The effects of excessive heat load on production usually build up over several days, so severe stress is generally associated with heat wave conditions that persist and where there is little relief from the high temperature and humidity at night. In order to maintain milk production in heat-stressed cows that have a decreased dry matter intake (DMI), the nutrient density of the diet should be increased.

Effects on reproduction

Environmental heat has a major impact on reproductive performance in cattle. Prolonged times to conception associated with hot weather are mediated through both reduced conception rates and submission rates. The reduction in conception rates in heat stressed cows appears to be related to the level of production, with larger declines occurring in higher producing cows. Consequently, the problem of reduced conception rates in dairy herds during the summer months has increased as production per cow has increased.

There is also some loss in fertility in the bull, particularly involving spermatogenesis. In addition, there can be interactions with reduced nutrient intakes that are superimposed on the direct effects of high temperatures on reproduction.

There is some breed variation as well, with Jerseys being slightly more tolerant to heat stress than Brown Swiss that, in turn, are more tolerant than Holstein-Friesians.

The hyperthermia resulting from maternal heat stress has a number of detrimental effects on the physiological processes that are important for the establishment and maintenance of pregnancy following fertilisation and may lead to an increased loss of early stage embryos. While older embryos (>7 days) appear to be more tolerant of heat load, there is some evidence that heat load in early gestation increases the risk of foetal loss out to Day 90 of gestation.

As well as the direct effects of environmental heat on reproductive physiology and the conceptus, reproductive performance may also be indirectly reduced by adverse effects on energy balance, as reduced appetites reduce dry matter intake (DMI) under hot conditions. In addition, a reduction in pasture quality is commonly observed during the hotter months in Australia, with the nutritive value of pastures on offer in the summer tending to be lower than that offered in winter. Thus, superimposed on the direct effects of high temperature on reproductive performance are the effects of a reduced plane of nutrition.

Effects on health

Hot, humid conditions, particularly following rain, favour environmental mastitis as udders are contaminated with mud and faeces and conditions are suitable for bacterial multiplication.

Cows wallow in mud to alleviate heat stress. Muddy udders need to be washed, increasing the likelihood of milking wet udders, which predisposes to new intramammary infections.

Heat stress can contribute to lameness. Heat stressed cows eat less frequently and often will not graze during the day. Reduced pasture intakes followed by slug feeding with concentrates at milking, lack of cud chewing, increased respiratory rates and excessive loss

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of saliva from drooling all contribute to the overall reduction in the natural buffering of the rumen contents, which is believed to be a significant factor of subacute rumen acidosis (SARA). Laminitis and other lameness conditions, such as white line lesions, may follow.

Heat stress affects calf viability by impeding foetal growth in the last trimester of pregnancy and by depressing colostrum quality and immunoglobulin transfer. Uterine blood flow and placental mass are reduced and endocrine profiles altered when cows are heat stressed during the last 90 days of gestation. As a consequence, calf birth weights are reduced by as much as 6 to 8%. Cows in late gestation (the last 3-4 weeks) during hot weather have reduced feed intake, which can also result in lower calf birth weights. The reduced DMI contributes to a negative energy balance at this time, which promotes mobilisation of body fat and subsequent ketogenesis, resulting in reduced body condition at calving.

Cows calving during daylight hours in hot climates with direct exposure to solar radiation are particularly vulnerable to heat stress and hyperthermia, especially cows suffering from hypocalcaemia in which the control of body temperature by natural mechanisms is greatly reduced.

Heat stress at parturition has significant consequences for calves. Once delivered, calves born in stressful conditions are weaker and slower to suck. Indeed, calves born during the hotter summer months have higher rates of failure of passive transfer. It has been suggested that an increase in serum corticosteroids in heat-stressed neonates reduces the permeability of the intestine to immunoglobulin absorption. Thus, it appears that both physical and physiological mechanisms are responsible for the high rates of failure of passive transfer in these calves.

Prognosis and treatment

Severe heat stress (heat stroke) is a life-threatening condition. Affected animals should be cooled by hosing their entire body with cool water. If available a large fan placed in front of the animal is a useful adjunct to water treatment as the increased airflow increases the rate of evaporative cooling. Cool drinking water administered by stomach tube into the rumen may also be useful.

The rectal temperature should be monitored closely; once the animal's body temperature returns to normal the measures taken to cool the animal can be discontinued.

Prevention and control

Water requirements parallel the increase in ambient temperature, and water intake may increase as much as 50% during periods of heat stress. Cows need ample access to fresh, clean, cool and good quality drinking water to allow for increased intakes in order to compensate for increased losses from sweating and increased respiration rates. The farm's drinking water system should be designed so that it is capable of providing the large amounts of water required during very hot conditions.

Tradition has held that cows should be kept away from streams and ponds because they pose a risk to the animals' health, most notably in the form of leptospirosis and mastitis. However, studies in the USA have found that cooling ponds not only effectively reduce body temperature but have no apparent adverse effect on udder health or milk quality. Cooling ponds, therefore, represent a controversial method for the management of heat stress.

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Provision of shade, either natural or artificial, is critical. With pasture-fed dairy herds, covered milking yards can provide some relief while cows are yarded at milking times. It should be noted that thermal radiation from the roof can add significant heat load to cattle, especially in the case of low structures without a ridge opening. This can be alleviated to some degree by cooling the roof with water, adding insulation or painting the roof with a reflective type of paint. Nevertheless, priorities should always be directed to cooling the cows rather than the roof.

Cooling by using sprinklers and fans is another option. In pasture-fed cattle, sprinkling cows as they stand in the milking yard prior to milking is a simple, low-cost strategy that can be used to cool cows on hot days. When feed pads are used, fans and sprinklers can be installed over the areas where cows stand while feeding. Sprinkling is most effective when combined with air movement, either from natural breezes or the use of fans. In warm, humid environments, sprinklers should disperse large water droplets to adequately wet the hair coat and skin. Cooling is accomplished as water evaporates from the hair and skin. If a misting system is used, which does not wet the coat through to the skin, an insulating layer of air can become trapped between the droplets of water on hair shafts and the skin. This will impede natural evaporative heat loss from the skin and may result in a harmful body heat build-up.

Exit lane sprayers, which automatically spray water on to cows as they pass through, can also be used. A less complex system can be made by simply locating an ordinary shower nozzle in the exit lane, so cows can be showered as they leave the bail area/milking platform.

An alternative practice that can be used on dairy farms in hot weather is to milk earlier in the morning and later in the afternoon in order to avoid moving cattle and holding them in the milking yard during the hottest parts of the day. A further possibility is to return cows to the milking yard during the hottest parts of the day and stand them under sprinklers until mid-afternoon, milking late in the afternoon and returning cattle to pasture as the day starts to cool.

In dairy herds, it may be possible to modify the ration being fed in order to minimise the fall in milk production. The most limiting nutrient for lactating cows during periods of heat stress is energy intake and a common approach to increase the energy density is to reduce the forage and increase the concentrate content of the ration. The logic is that less fibre (less bulk) will encourage intake, while more concentrates increase the energy density of the diet. Although low fibre, high fermentable carbohydrate diets may lower the dietary heat load compared with higher fibre diets, this effect must be carefully balanced with the potential for rumen acidosis and displaced abomasum associated with such high-grain diets. Overall, increasing the energy density, i.e. the proportion of grain in the ration, will help maintain DMI, as long as some form of cooling with shade and sprinklers is available. If cooling is not provided for high-producing cows then a lower level of concentrate should be chosen. Another option to increase energy intake is to add supplemental fat (a non-starch energy source) in the form of whole cottonseed or tallow to the ration.

Over-feeding with protein should be avoided because it requires energy to excrete any excess nitrogen. Where pastures only are available the highest quality (lowest percentage neutral detergent fibre) pastures should be fed at night. In situations where a mixed ration

based on conserved forage is available, it is best to feed this under shade during the day and have pasture grazed at night. High-quality forages are digested faster and result in less heat production.

Hot weather can increase the need for mineral supplementation. Losses through sweating and increased urination increase the requirement for sodium and potassium in the diet. When environmental temperatures are above 30°C, the diet should contain at least 125 g salt/cow/day. Potassium should be at least 1.5% of the ration DM.

A longer term strategy to manage heat stress is the use of crossbreeding. Jerseys and Brown Swiss cattle are more tolerant to hot conditions than are Holsteins and could be considered in a future cross-breeding programme.

What are some of the potential risks associated with the nutritional management of heat stress in dairy cattle?

Although increasing the energy density of the diet (i.e. increasing the grain/forage ratio), as suggested above, is beneficial from the point of view of managing heat stress, it may put the animals at greater risk of digestive disorders such as SARA and displaced abomasum. SARA in turn has been associated with the occurrence of laminitis-associated claw lesions, such as sole haemorrhages and white line disease, and lameness. This part of the manuscript discusses how increasing the proportion of grain in the ration (and thus decreasing the amount of forage), as part of the nutritional management of heat stress, may lead to cows developing SARA and displaced abomasum.

SARA and laminitis/lameness

SARA plays an important role in the initiation of laminitis and subsequent lameness. Excessive grain or non-structural carbohydrate (NSC) feeding, slug feeding of grain, feeding sources of NSC that are rapidly fermented in the rumen, and feeding finely chopped silage (in particular maize silage) are common factors in the development of laminitis because of their propensity for inciting SARA.

Over the last 20 years or so there has been a marked increase in the feeding of starch-based concentrates to cows on pasture. In many herds, especially in Australia, 'slug feeding' occurs, in which 2 to 8 kg concentrates are fed twice daily at milking time. Unless such herds are well managed (possibly by including buffers and ionophors in the concentrate), there is a risk of SARA occurring, such that claw lesions associated with subclinical laminitis are often seen in these situations. The risk of SARA developing is less when the concentrate/forage ratio of the diet is kept well under 60:40, which is usually the case in pasture-based dairying systems. However, such a concentrate/forage ratio does not readily fit into the nutritional management of heat stress which involves increasing the proportion of grain in the diet, while feeding high-quality forages, i.e. those with a low percentage of neutral detergent fibre.

Laminitis has a complex aetiology and uncertain pathogenesis. The pathophysiology of laminitis in cattle has long been assumed to be analogous to that in the horse, namely a disturbance in the micro-circulation of the corium, with ensuing degenerative and possibly inflammatory changes at the dermal-epidermal junction, which may or may not be followed by rotation and 'sinking' of the distal phalanx. Sequelae include impaired horn production with diffuse softening and discolouration, and haemorrhages in the sole and heel (subclinical laminitis); double soles and walls; ulcers in the sole and toe; white line lesions

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(haemorrhages, separation and abscess); and in chronic cases, deformation of the whole claw.

For many years, the commonly accepted hypothesis regarding the aetiology and pathogenesis of subclinical laminitis has been that toxic substances, such as histamine, lactic acid, serotonin and endotoxin, are formed or released in the digestive tract (mainly as a consequence of rumen acidosis). These toxic, vasoactive components, together with a coagulopathy, are believed to severely disturb the micro-vasculature and haemodynamics of the corium, leading to tissue hypoxia and nutrient starvation, followed by ischaemic necrosis and degeneration of the horn-producing structures.

While the aetiology and pathogenesis of laminitis may not be as yet fully understood, an increased incidence of laminitis-associated lesions is commonly observed with increasing levels of concentrate feeding, particularly if no attempt is made to buffer the ration adequately in order to minimise the effect of SARA. This condition is likely to be a relatively common condition in dairy cows as they first enter the milking herd and many cases of subclinical laminitis are caused by relatively mild episodes of SARA.

The signs exhibited by animals with SARA are usually mild; namely reduction in milk yield, reduced butterfat percentage, reduced rumen contractions and some degree of lethargy. However, the associated changes in the animals' rumen environment are large enough to cause subclinical laminitis. No obvious changes are seen in the claws at the time of the insult, but lesions associated with subclinical laminitis (sole haemorrhages and yellowing and softening of the horn) may be detected some 6 to 8 weeks later.

Until recently, laminitis was rather uncommon in pasture-based dairy cattle of Australia. However, the incidence of laminitis (and to be more specific subclinical laminitis) is increasing, probably associated with the intensification and increasing amounts of concentrate feeding.

The role of nutrition and feeding management in the development of laminitis/lameness in the Australasian dairy scene is not well understood. However, it is likely that nutritional factors moderate the incidence and severity of lameness by contributing to the occurrence of subclinical laminitis. Therefore, there may be situations where the role of nutrition should be considered as a potential moderator of the extent and severity of lameness. The risk of nutrition contributing to laminitis/lameness will increase according to the amount of concentrate being fed, particularly when these concentrates are being 'slug-fed' in the bail while cows are being milked. In this situation, laminitis/lameness is associated with SARA and, therefore, any recommendations made to reduce the incidence of subclinical laminitis should be aimed at preventing SARA.

Dairy cows require a minimum amount of effective fibre and forage in their diet for proper chewing and rumination activity, proper rumen function and to maintain rumen pH >6.2. They need to chew (masticate and ruminate) for 10 to 12 hours/day in order to maintain normal rumen function. The effective fibre of a feed is directly related to the chewing time and, therefore, saliva production associated with that particular feed. High-fibre diets, such as hay and coarsely chopped silage, stimulate rumination, which in turn increases saliva flow. Saliva is rich in bicarbonate, which acts as a buffer by neutralising the acid produced in the rumen. Fine chopping reduces the effective fibre content of forages. Adding buffers to rations containing finely chopped silage may help if saliva production is low.

Under US feeding systems, it is recommended that the diet contains a minimum of 25% NDF. This recommendation, however, may be inadequate for diets in which pasture is the predominant forage. One reason that rumen pH may be low on high-quality pasture is that the NDF in pasture is not as effective as that in silage and hay. In this situation, adding a small amount of straw to the diet may be beneficial.

Finally, heat stress has been associated with an increased incidence of lameness in North America. Potentially, heat stress could also be a predisposing factor to lameness in Australasia. Heat stress alters the animals' breathing rate (it may double), heart rate, immune response (reduced) and behaviour (e.g. cows are standing for longer periods of time, which promotes pooling of blood in the digits). Heat-stressed cows also lose significant amounts of saliva from open-mouth breathing and drooling.

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 subacute rumen acidosis (SARA) during periods of hot and humid weather. Considering the above, it can be argued that increasing the grain/forage ratio, as part of the nutritional management of heat stress, may increase the risk of SARA in heat-stressed dairy cows even more.

Displaced abomasum

A number of managerial, environmental and, possibly, hereditary risk factors are thought to play a role in the development of displaced abomasum. As pregnancy progresses, the growing uterus pushes the rumen cranially and dorsally and the abomasum may assume a position more to the left than normal. The uterus begins to slide under the caudal aspect of the rumen, reducing rumen volume by one-third at the end of gestation. The decrease in feed intake that occurs commonly during the peri-parturient period may reduce rumen volume even more, allowing the abomasum to move to the left. The pylorus, however, continues to extend across the abdomen to the right side of the cow.

Three factors are believed to be responsible for allowing the abomasum to shift further to the left side of the abdominal cavity. First, the rumen must fail to take up the void left by the involuting uterus following parturition, allowing the abomasum to fill that void, thus moving even further to the left. If the rumen moved into its normal position on the left ventral floor of the abdomen, the abomasum would not be able to slide under it. Second, the omentum attached to the abomasum must have been stretched to permit movement of the abomasum to the left side. These two factors provide opportunity for displacement. A third factor necessary to cause abomasal displacement is abomasal atony.

Normally, gases produced in the abomasum (from fermentation of feedstuffs) are expelled back into the rumen as a result of abomasal contractions. It is believed that these contractions are impaired in cows developing displacement of the abomasum. Trapped gas then causes the drift upwards, either along the left or the right abdominal wall. In most cases, the cause of abomasal atony is less clear. Potential causes include increased volatile fatty acids (VFA) and decreased smooth muscle tone associated with hypocalcaemia.

Nutrition undoubtedly plays an important role in the aetiology of displaced abomasum, with the use of concentrates and low-fibre diets generally being incriminated, probably through

an increase in the concentration of VFA, which inhibits abomasal motility. The flow of digesta from the abomasum to the duodenum is reduced and ingesta accumulate in the abomasum.

An observation from many practitioners in Australia is that as the level of concentrate feeding in a region increases, so does the incidence of LDA.

Decreased abomasal motility may result in ingesta accumulating in the abomasum. A high concentrate, reduced-forage diet can also promote the appearance of VFA in the abomasum by reducing the depth of the rumen raft or fibre mat (made up primarily of the long fibres of forages). The rumen raft captures grain particles so they are fermented at the top of the rumen liquor. In cows with an inadequate rumen raft, some grain particles may pass on to the abomasum, where they can then be fermented to some extent. The large volume of methane and carbon dioxide found in the abomasum following grain feeding may become trapped there, potentially causing distension and displacement.

The physical form of the ration must also be considered. A good rumen raft is required to stimulate rumen contractions in the cow. Abomasal contractions are closely linked to rumen contractions through the vagus nerve reflex. If the rumen has an inadequate raft to stimulate contractions, then the abomasum will not contract properly either. A thick rumen raft is generally present when cows are fed a high-roughage diet, but the depth of this raft is rapidly reduced as the amount of concentrates in the ration increases. Feeding high-carbohydrate rations, which contain inadequate levels of roughage (crude fibre levels below 17%), is an important risk factor for displaced abomasum. Fibre useful to the cow, in terms of rumen health, must stimulate chewing and saliva production in the cow. Not meeting the chewing requirements of the cow is a major problem contributing to abomasal displacement.

Feeding a diet containing adequate amounts of fibre of adequate length is one of the most important ways recommended to decrease the incidence of displaced abomasum. However, by and large, such a feeding regimen is not consistent with that recommended for heat-stressed dairy cows.

In conclusion, the nutritional management of lactating dairy cows may be particularly challenging during periods of intense heat. The main objective should be to maintain good levels of performance and homeostasis, but not add to the internal heat load or tendency toward SARA with subsequent subclinical laminitis, or displaced abomasum.

Further reading

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