Rethink nutritional heat stress strategies

Bottom Line

with

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AS spring approaches, nutritionists and dairy managers start gearing up nutritional strategies and management practices designed to alleviate intake depression and production declines among heat-stressed cows.

Heat stress is not confined to the southern dairy sheds. In fact, heat stress that starts slowly, reaches a peak, plateaus for several months and then gradually declines may be more manageable than the spurts of high heat and humidity that plague cows in the North.

These cows tend to severely reduce intakes only to gorge once the weather cools off. Throw in the tendency to increase ration energy density to compensate for reduced intakes, coupled with increasing starch digestibility in long-stored fermented feeds like corn silage or high-moisture corn, and you have the ideal situation leading to bouts of acidosis.

Researchers from the University of Arizona are continuing their tradition of unraveling the biological mystery surrounding heat stress and leading us to rethink our approaches to managing this chronic and expensive problem.

Biology of reduced intakes

The biology of the heat-stressed cow can be compared and contrasted to that of the negative energy balance (NEBAL), early-lactation cow that is unable to consume enough feed to meet maintenance and milk production needs.

NEBAL cows in non-heat stress environments typically respond by losing weight and altering carbohydrate and lipid metabolism to support mammary nutrient demands. These cows exhibit reduced circulating insulin levels coupled with a reduction in systemic insulin sensitivity.

This reduces glucose uptake from systemic muscle and adipose tissue. It also allows for adipose mobilization of non-esterified fatty acids (NEFA) for energy and milk fat precursors (O'Brien et al., 2008). The net effect of increased NEFA-derived energy coupled with glucose sparing is that more glucose is made available to the mammary gland for the production of lactose to drive milk volume.

Heat-stressed NEBAL cows differ by displaying a reduction in rumination and nutrient absorption with increased maintenance requirements. Arizona researchers (Rhoads et al., 2007) conducted a novel experiment to determine how much the reduced intake in heat-stressed cows contributed to the loss in milk yield.

One group of mid-lactation cows were assigned to cyclical heat stress (thermal heat index of 80 for 16 hours with afternoon rectal temperatures of about 105.1°F; Figure). Paired cows exposed to thermal-neutral conditions (thermal heat index of 64 for 24 hours) were then fed the same level of intake as what the heat-stressed cows consumed.

The heat-stressed cows reduced dry matter intake (DMI) by about 5 kg per day, leveling off by day 4 of the experiment. Their daily milk production declined steadily for seven days, culminating with a reduction of about 14 kg of milk.

Thermal-neutral cows on restricted intakes to parallel the heat-stressed animals had a reduction of approximately 6 kg of milk and stabilized at this lower production level by two days into the trial.

The researchers concluded that the reduction in DMI by heat-stressed cows only accounted for about 40-50% of the decrease in production.

Biology of heat stress

Earlier Arizona research (Wheelock et al., 2006) in a similarly designed trial lends insight into why a reduction in DMI accounts for less than half of the loss in milk yield in heat-stressed cows. Surprisingly, this experiment showed that heat-stressed animals did not have the expected increase in circulating NEFA observed in the non-heat-stressed NEBAL group.

Researchers have also observed an atypical increase in plasma urea nitrogen in lower-intake, heat-stressed cows, indicating the possibility that muscle tissue may be contributing to more of the bodyweight loss than previously assumed (Baumgard, 2008).

Using an intravenous glucose tolerance test, the Arizona researchers have also shown increased insulin response to a glucose challenge in heat-stressed animals. Insulin reduces fat mobilization, explaining the reduced NEFA levels in the stressed group.

It has been theorized that heat-stressed cows respond metabolically differently in an attempt to reduce metabolic heat production. Increased insulin sensitivity and the oxidation of glucose for energy, although not as energy dense as fats, generate less metabolic heat than the oxidation of fatty acids (Baumgard, et al., 2007).

Without the ability to rely on energy from oxidized NEFA, heat-stressed cows are forced to utilize glucose for energetic needs in tissues like skeletal muscle. Lowered glucose flow to the mammary gland inhibits lactose synthesis, accounting for the reduced milk yield that could not be explained solely by a reduction in DMI.

Heat stress, acidosis

When cows are subjected to heat stress, they respond by sweating and panting. Increased respiration rates cause cows to exhale elevated levels of carbon dioxide. The increased blood pH with respiratory-induced alkalosis is actually the result of a deficiency of carbonic acid. When a cow pants, bicarbonate is converted to carbonic acid, which is broken down to carbon dioxide and water and expired via panting (West, 2003).

The cow needs to maintain a 20:1 bicarbonate:carbon dioxide ratio in the blood. The decrease in blood carbon dioxide causes the kidneys to respond by increasing urinary excretion of bicarbonate to maintain this ratio. This also reduces the amount of bicarbonate in saliva to buffer the rumen pH (Baumgard et al., 2007).

Lowered saliva buffering capacity coupled with reduced saliva flow (due to drooling), reduction in rumination during periods of lowered intake and any potential ration sorting can conspire to make cows less efficient at buffering the rumen during periods of heat stress.

Nutritional considerations

Several nutritional strategies can be implemented during periods of heat stress. These include reformulations to account for reduced DMI, higher maintenance costs and metabolic heat production from various feedstuffs (West, 2003) and feeding in the early-morning or late-evening hours to reduce total heat load on the animal (Staples, 2007).

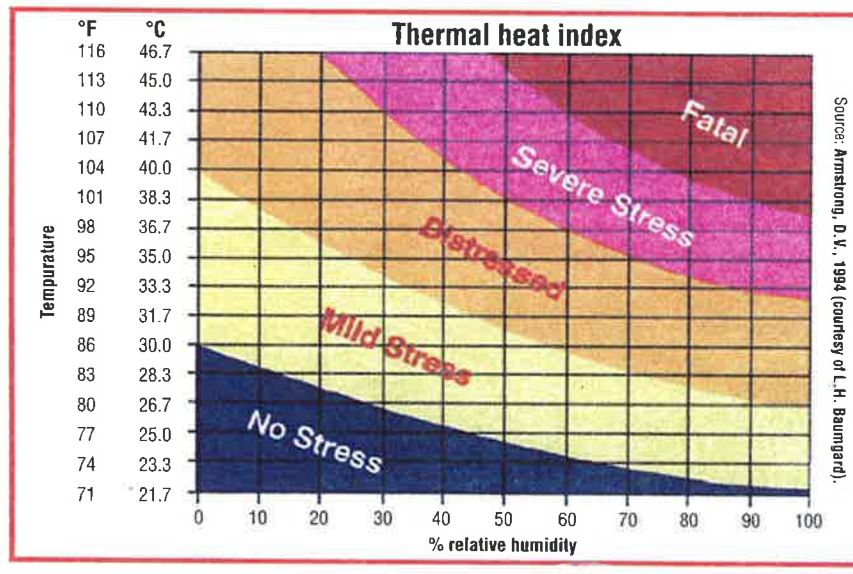
Feeding diets with a high dietary cation-anion difference has helped improve intake in heat-stressed cows (West, 2003). Feeding elevated levels of potassium (1.5-1.6% of dry matter), sodium (0.45-0.60% of dry matter) and magnesium (0.35-0.40% of dry matter) is also recommended (Staples, 2007) as they are the primary cations in bovine sweat.

Recent research indicates that the addition of 12 g per day of encapsulated niacin may improve heat tolerance through elevation of cellular heat shock proteins and peripheral vasodilation (Zimbelman et al., 2008).

Nutritionists often increase the energy or protein density of the ration during prolonged periods of heat stress. Caution should be exercised if increasing protein levels during hot weather. Studies have shown that the energetic cost associated with synthesizing and excreting urea can compromise milk production when feeding excess protein (West, 2003). Blood non-protein nitrogen content has been positively correlated with rectal temperatures, suggesting reduced energy efficiency and greater heat production with excess dietary nitrogen (West, 2003).

INCREASING energy density is typically accomplished with extra grain or fat and a reduction in forages. The trials from Arizona clearly indicate that heat-stressed cows do have an extra requirement for dietary or rumen-produced glucose precursors like propionate. However, lower-fiber/high-fermentable carbohydrate rations may drive propionate, increase energy density and lower heat increment, but these effects must be balanced with the potential for ruminal acidosis in animals already prone to acidosis due to intake variations and reduced rumen buffering capacity.

Consideration should be given to not only the quantity of fermentable carbohydrate but also to the dynamic aspects of starch digestibility in fermented feeds such as corn silage or high-moisture corn (Mahanna, 2007).



The Bottom Line

The reduction in DMI by heat-stressed cows only accounts for about 40-50% of the decrease in milk production. Heat-stressed cows appear to differ metabolically from non-heat-stressed cows experiencing similar levels of intake reduction by having a lower-than-expected level of circulating NEFA and higher insulin effectiveness.

This translates into an increased need for dietary or rumen-produced glucose precursors to meet the energy need of systemic tissues and the mammary demand for lactose precursors to drive milk volume.

Nutritionists must balance the use of concentrates and forages to fuel production without causing ruminal acidosis in animals already deficient in ruminal buffering capacity.

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