The Effects of Heat Stress on Production, Metabolism and Energetics of Lactating and Growing Cattle

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Introduction

Heat stress negatively impacts a variety of dairy and beef parameters including milk yield, growth and reproduction and therefore is a significant financial burden (~$900 million/year for dairy and > $300 million/year in beef in the U.S.; St. Pierre et al., 2003). Advances in management (i.e. cooling systems; Armstrong, 1994; VanBaale et al., 2005) and nutritional strategies (West, 2003) have alleviated some of the negative impact of thermal stress on cattle, but production continues to decrease during the summer. Accurately identifying heat-stressed cattle and understanding the biological mechanism(s) by which thermal stress reduces milk synthesis, growth and reproductive indices is critical for developing novel approaches (i.e. genetic, managerial and nutritional) to maintain production or minimize losses during stressful summer months.

Biological Consequence of Heat Stress

The biological mechanism by which heat stress impacts production and reproduction is partly explained by reduced feed intake, but also includes altered endocrine status, reduction in rumination and nutrient absorption, and increased maintenance requirements (Collier and Beede, 1985; Collier et al., 2005) resulting in a net decrease in nutrient/energy available for production. This decrease in energy results in a reduction in energy balance (EBAL), and partially explains (reduced gut fill also contributes) why dairy cattle lose significant amounts of body weight when subjected to unabated heat stress.

Dairy

Reductions in energy intake during heat stress result in a majority of dairy cows entering into negative energy balance (NEBAL), regardless of the stage of lactation. Essentially, because of reduced feed and energy intake the heat-stressed cow enters a bioenergetic state, similar (but not to the same extent) to the NEBAL observed in early lactation. The NEBAL associated with the early postpartum period is coupled with increased risk of metabolic disorders and health problems (Goff and Horst, 1997; Drackley, 1999), decreased milk yield and reduced reproductive performance (Lucy et al., 1992; Beam and Butler, 1999; Baumgard et al., 2002, 2006). It is likely that many of the negative effects of heat stress on production, animal health and reproduction indices

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are mediated by the reduction in EBAL (similar to the transition period). However, it is not clear how much of the reduction in performance (yield, daily gain and reproduction) can be attributed or accounted for by the biological parameters affected by heat stress (i.e. reduced feed intake vs. increased maintenance costs).

Beef

In general, heat stress-induced production losses for beef cattle are not as severe as those for the dairy industry. It is not entirely clear why growing cattle tolerate higher THI conditions and exhibit a greater heat strain threshold than lactating dairy cows, but may involve: 1) reduced surface area to mass ratio, 2) reduced rumen heat production (because of the mostly grain diet), and 3) reduced overall metabolic heat production (on a body weight basis). In addition, beef cattle will often experience compensatory gain after mild or short periods of heat stress (Mitlöhner et al., 2001). The combination of these factors translate into heat-related reduced gain that is typically less than 10 kg, which amounts to ~7 extra days in the feed lot (St-Pierre et al., 2003). Furthermore, the impact of heat stress on reproductive indices is typically not as severe in beef cattle due to the seasonal nature of breeding programs (often occurring during the spring in the U.S.).

Metabolic Adaptations to Reduced Feed Intake

A prerequisite to understanding the metabolic adaptations which occur with heat stress, is an appreciation of the physiological and metabolic adjustments to thermal-neutral NEBAL (i.e. underfeeding or during the transition period). There is much less known about the metabolic and physiological effects of hyperthermia in beef cattle as compared to dairy cows, probably because the economic impact on the industry is less severe. Consequently, the changes in heat-related metabolism will be compared and contrasted primarily to the better-known changes in lactating dairy cows.

Early lactation dairy cattle enter a unique physiological state during which they are unable to consume enough nutrients to meet maintenance and milk production costs and animals typically enter NEBAL (Moore et al., 2005a). Negative energy balance is associated with a variety of metabolic changes that are implemented to support the dominant physiological condition of lactation (Bauman and Currie, 1980). Marked alterations in both carbohydrate and lipid metabolism ensure partitioning of dietary and tissue derived nutrients towards the mammary gland, and not surprisingly many of these changes are mediated by endogenous somatotropin which naturally increases during periods of NEBAL (Bauman and Currie, 1980). One classic response is a reduction in circulating insulin coupled with a reduction in systemic insulin sensitivity. The reduction in insulin action allows for adipose lipolysis and mobilization of non-esterified fatty acids (NEFA; Bauman and Currie, 1980). Increased circulating NEFA are typical in “transitioning” cows and represent (along with NEFA derived ketones) a significant source of energy (and are precursors for milk fat synthesis) for cows in NEBAL. Post-absorptive carbohydrate metabolism is also altered by the reduced insulin action during NEBAL with the net effect being reduced glucose uptake by systemic tissues (i.e. muscle and adipose). The reduced nutrient uptake coupled

with the net release of nutrients (i.e. amino acids and NEFA) by systemic tissues are key homeorhetic (an acclimated response vs. an acute/homeostatic response) mechanisms implemented by cows in NEBAL to support lactation (Bauman and Currie, 1980). The thermal-neutral cow in NEBAL is metabolically flexible, in that she can depend upon alternative fuels (NEFA and ketones) to spare glucose, which can be utilized by the mammary gland to copiously produce milk.

Heat Stress and Production Variables

Dairy

Heat stress reduces feed intake and both daily gain in beef cattle and milk yield in dairy cattle. The decline in nutrient intake has been identified as a major cause of reduced production (Fuquay, 1981; West, 2002, 2003). However, the exact contribution of declining feed intake to the overall reduced milk yield or average daily gain remains unknown. To evaluate this question in both dairy and beef cattle we designed experiments involving a group of thermal neutral pair-fed animals to eliminate the confounding effects of dissimilar nutrient intake. First we used lactating Holstein cows in mid-lactation that were either cyclically heat-stressed (THI = ~80 for 16 hrs/d) for 9 days or remained in constant thermal-neutral conditions (THI = ~ 64 for 24 hrs/d), but pair-fed with heat stressed cows to maintain similar nutrient intake (Rhoads et al., 2007). Cows were housed at the University of Arizona's ARC facility and individually fed ad libitum a TMR consisting primarily of alfalfa hay and steam flaked corn to meet or exceed nutrient requirements (NRC, 2001). Heat-stressed cows had an average rectal temperature of 40.6°C (~105.1°F) during the afternoons (maximum THI) of the treatment period. Heat-stressed cows had an immediate reduction (~5 kg/d) in dry matter intake (DMI) with the decrease reaching nadir at ~ day 4 and remaining stable thereafter (Figure 1). As expected and by design, thermal-neutral pair-fed cows had a feed intake pattern similar to heat-stressed cows (Figure 1). Heat stress reduced milk yield by ~14 kg/d with production steadily declining for the first 7 days and then reaching a plateau (Figure 2). Thermal neutral pair-fed cows also had a reduction in milk yield of approximately 6 kg/d, but milk production reached its nadir at day 2 and remained relatively stable thereafter (Figure 2). This indicates the reduction in DMI can only account for ~40-50% of the decrease in production when cows are heat-stressed and that ~50-60% can be explained by other hyperthermia-induced changes. We have repeated this experiment multiple times and the effects on DMI and milk yield are remarkably consistent (Wheelock et al., 2006; Shwartz and Baumgard, unpublished).

Beef

To evaluate the differential effects of heat stress vs. reduced nutrient intake in beef cattle we studied growing Holstein beef bulls (n=12, 4-5 months of age, 136-182 kg BW; O'Brien et al., unpublished). Bulls were either cyclically heat-stressed (29.4 to 40ºC, 25-40% humidity, and 12 hours of light [conditions slightly warmer than during our dairy experiments]) or were maintained in thermal-neutral conditions (18 to 20ºC, 12 hours of light), but pair-fed (86% concentrate, 14% protein, 2x/d) with heat-stressed bulls to maintain similar nutrient intake. Heat-stressed bulls had an average rectal temperature of ~ 40.6°C (105.1°F) during the afternoons (peak ambient THI). Heat

stress reduced DMI by ~12% (data not presented) and as expected (and by design) thermal-neutral pair-fed bulls had a feed intake pattern similar to heat-stressed cows (O'Brien et al., unpublished). Heat stress eliminated body weight gain and thermal neutral pair-fed animals had a similar reduction in performance (Figure 3; O'Brien et al., unpublished).

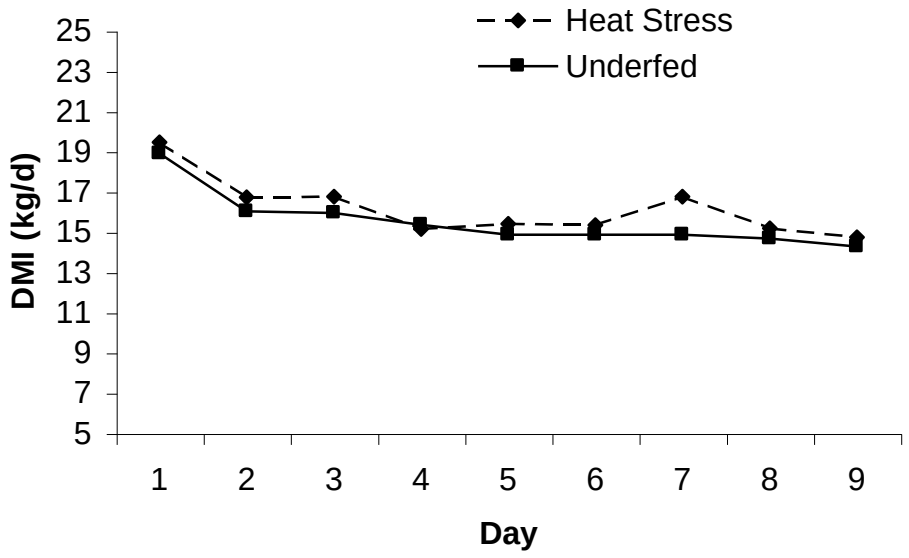

Figure 1. Effects of heat stress and underfeeding (pair-feeding) thermal-neutral lactating Holstein cows on dry matter intake (Rhoads et al., 2007).

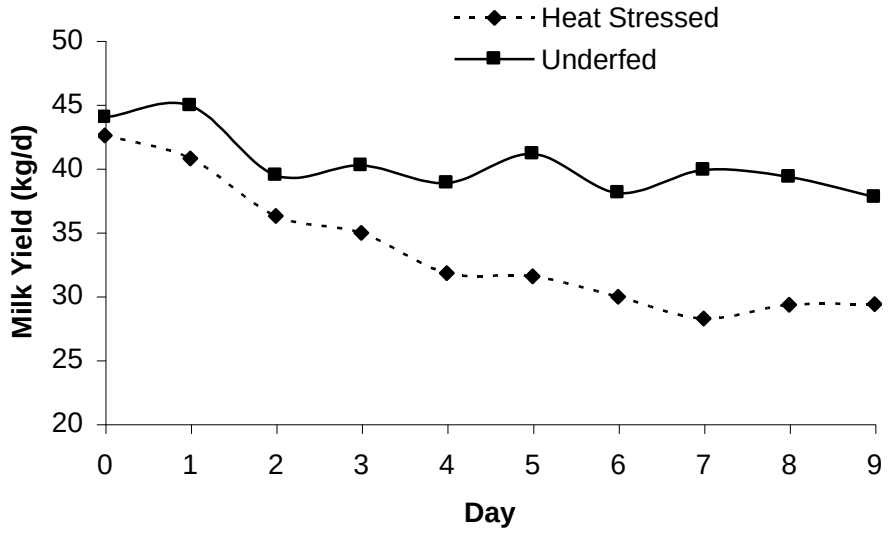

Figure 2. Effects of heat stress and underfeeding (pair-feeding) thermal neutral conditions on milk yield in lactating Holstein cows (Rhoads et al., 2007).

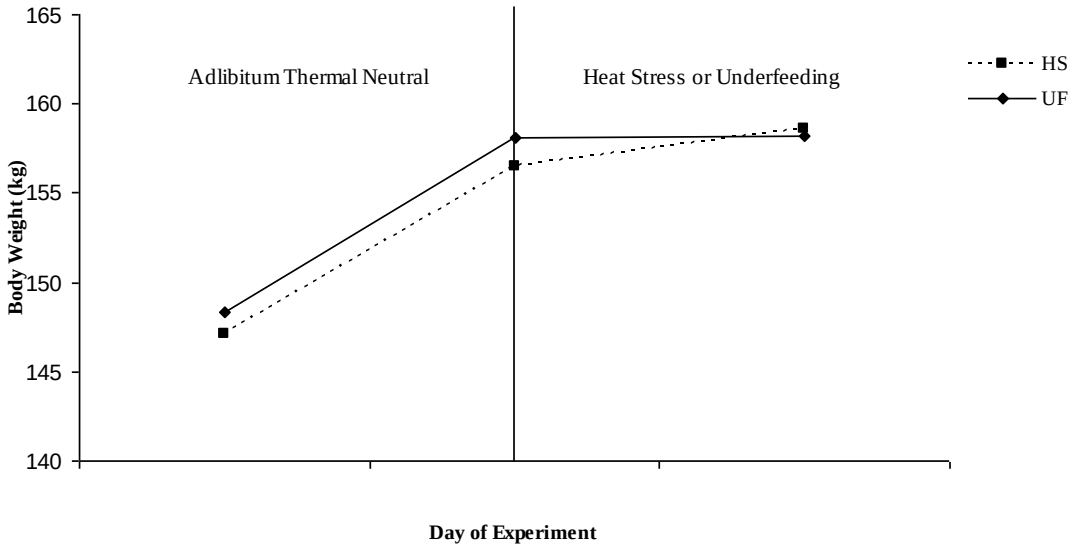

Figure 3. Effect of heat stress (HS) and underfeeding (UF; pair-feeding) during thermal neutral conditions on body weight gain (slopes of raw data) in growing Holstein beef bulls (O'Brien et al., unpublished).

Dairy vs. Beef

Despite being exposed to a slightly greater heat load, heat stress does not reduce DMI to the same extent in growing beef cattle as it does in lactating dairy cows (12 vs. 30%). In addition, the reduction in feed intake accounts for only ~50% of the decrease in milk yield (Figure 2), but appears to explain most (if not all) of the reduction in growth (Figure 3). Gaining a better appreciation for the biological reasons underlying the aforementioned discrepancy between beef and dairy may theoretically provide insight on how to prevent or ameliorate the exaggerated decrease in milk synthesis during hyperthermia.

Heat and Maintenance Costs

Estimating EBAL during heat stress (for both dairy and beef cattle) introduces problems independent of those that are inherent to normal EBAL estimations (Vicini et al., 2002). Considerable evidence suggest increased maintenance costs are associated with heat stress (7 to 25%; NRC, 2001), however due to complexities involved in predicting upper critical temperatures, no universal equation is available to adjust for this increase in maintenance (Fox and Tylutki, 1998). Maintenance requirements are thought to increase, as there is presumably a large energetic cost of dissipating stored heat. Not incorporating a heat stress correction factor results in overestimating EBAL and thus inaccurately predicting energy status.

In the beef study, the pair-fed thermal-neutral controls did not gain or lose body weight (Figure 3), suggesting nutrient and energy intake satisfied maintenance requirements. The heat-stressed bulls consumed similar quantities of the exact ration fed to the pair-fed thermal-neutral control animals and also had static body weight. This latter observation may indicate that, at least in growing bulls, heat stress does not increase overall maintenance requirements. If heat stress were to increase maintenance costs as reported (Fox and Tylutki, 1998; NRC, 2001) then the energy requirements of heat-stressed bulls should have exceed their pair-fed thermal-neutral counterparts. In turn, the heat-stressed bulls would have been consuming inadequate energy/nutrients and should have (by definition) lost body weight. However, this was not the case and heat-stressed bulls did not lose body weight (Figure 3), indicating that maintenance costs may not have been increased. Further research is necessary to evaluate the effects of heat on maintenance requirements and to determine if the physiological state (growth vs. lactation) influences energy partitioning during thermal challenges.

Metabolic Adaptations to Heat Stress

Dairy

Due to the reductions in feed intake and presumed increased maintenance costs, and despite the decrease in milk yield heat stressed cows enter into a state of NEBAL (Moore et al., 2005b). In a similar dairy trial to the one described above, heat-stressed cows entered into and remained in NEBAL (~4-5 Mcal/d) for the entire duration of heat stress (Figure 4; Wheelock et al., 2006). However, unlike NEBAL in thermal-neutral conditions, heat-stressed induced NEBAL doesn't result in elevated plasma NEFA (Figure 5). This was surprising as circulating NEFA are thought to closely reflect calculated EBAL (Bauman et al., 1988). In addition, using an IV glucose tolerance test, we demonstrated that glucose disposal (rate of cellular glucose entry) is greater in heat-stressed compared to thermal neutral pair-fed cows. Furthermore, heat-stressed cows have a much greater insulin response to a glucose challenge when compared to underfed cows (data not presented). Both the aforementioned changes in plasma NEFA and metabolic/hormonal adjustments in response to a glucose challenge can be explained by increased insulin effectiveness. Insulin is a potent anti-lipolytic signal (blocks fat break down) and the primary driver of cellular glucose entry. The apparent increased insulin action causes the heat-stressed cow to be metabolically inflexible, in that she does not have the option to oxidize fatty acids and ketones. As a consequence, the heat-stressed cow becomes increasingly dependant on glucose for her energetic needs and therefore less glucose is directed towards the mammary gland.

Beef

Although both the heat-stressed and pair-fed controls quit growing, neither mobilized adipose tissue (plasma NEFA remained <100 μEq/L), which is agreement with a lack of body weight *loss* (O'Brien et al., unpublished). However, despite similar changes in production and post-absorptive lipid variables, there were heat stress-induced changes in post-absorptive carbohydrate metabolism. Similar to lactating dairy cows, heat-stressed growing bulls appear to have an increase in glucose disposal rates

and have a much greater insulin response to a glucose challenge (Figure 6, O'Brien et al., unpublished).

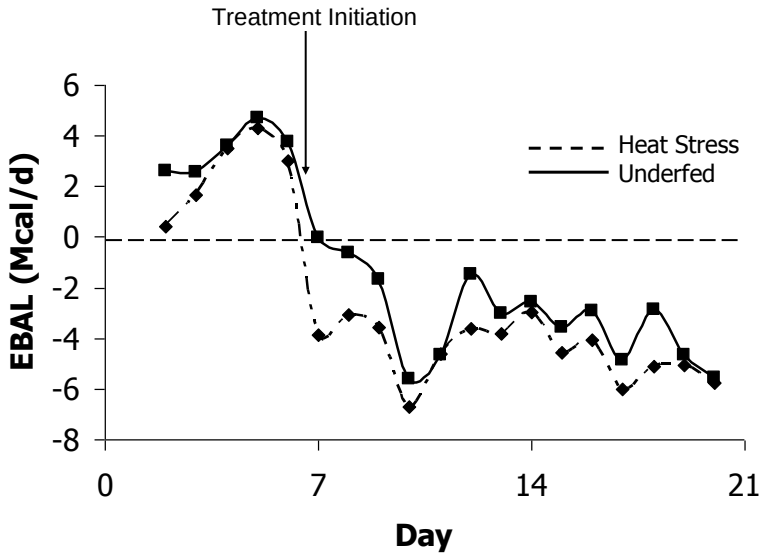

Figure 4. Effects of heat stress and underfeeding (pair-feeding) thermal-neutral conditions on calculated net energy balance in lactating Holstein cows (adapted from Wheelock et al., 2006.)

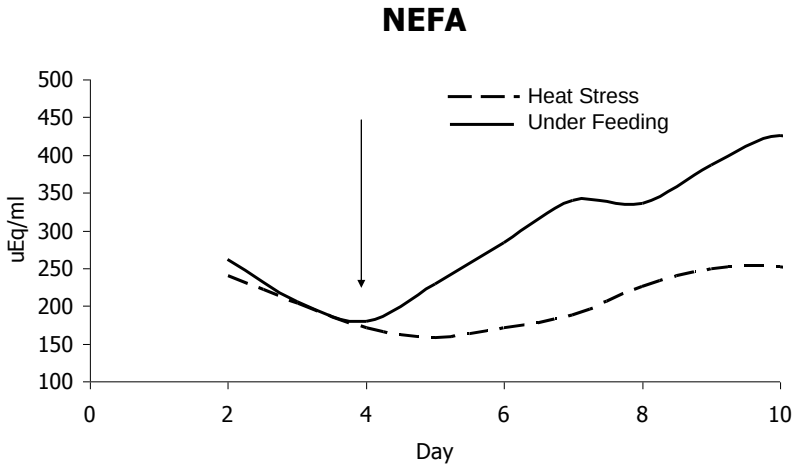

Figure 5. Effects of heat stress and underfeeding (pair-feeding) thermal-neutral conditions on circulating non-esterified fatty acids (NEFA) in lactating Holstein cows (adapted from Wheelock et al., 2006).

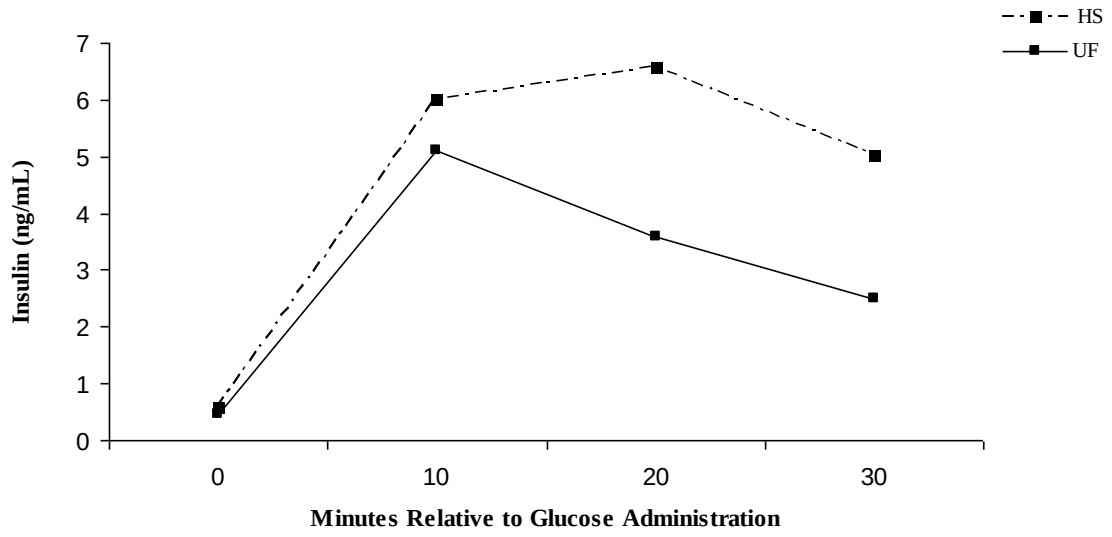

Figure 6. Effects of heat stress (HS) and underfeeding (UF; pair-feeding) in thermal-neutral conditions on plasma insulin response to a glucose challenge in growing beef cattle (O'Brien et al., unpublished).

Dairy vs. Beef

The changes in lipid and carbohydrate metabolism in heat-stressed dairy cattle may ultimately decrease the glucose availability needed for lactose synthesis. As a consequence, milk yield will decrease (milk synthesis is in large part dependant upon lactose synthesis) and this may quantitatively equal the extra amount of glucose that peripheral or extra-mammary tissues (primarily skeletal muscle) utilize. In beef cattle, the magnitude of heat-induced decrease in feed intake is not sufficient enough to signal adipose mobilization in pair-fed thermal neutral controls. However, beef animals do exhibit enhanced insulin response to supplemental glucose and an increase in exogenous glucose disposal. A possible reason why beef cattle appear to cope with heat stress better is that their production (tissue synthesis) does not rely on glucose to the extent that milk production does.

Theoretical Reasons for Altered Metabolism

Well-fed ruminants primarily oxidize (burn) acetate (a rumen produced VFA) as their principal energy source. However, during NEBAL cattle also largely depend on NEFA for energy. Therefore, it appears the post-absorptive metabolism of heat-stressed cattle markedly differs from that of thermal-neutral cattle, even though they are in a similar negative energetic state. The apparent switch in metabolism and the increase in insulin sensitivity is probably a mechanism by which cattle decrease

metabolic heat production, as oxidizing glucose is more efficient (Baldwin et al., 1980). In vivo glucose oxidation yields 38 ATP (assuming the ΔG of ATP hydrolysis is -12.3 kcal/mole under cellular conditions; Berg et al., 2007) or 472.3 kcal of energy (compared to 637.1 kcal in a bomb calorimeter) and in vivo fatty acid oxidation (i.e. stearic acid) generates 146 ATP or 1814 kcal of energy (compared to 2697 kcal in a bomb calorimeter). Despite having a much greater energy content, due to differences in the efficiencies of capturing ATP, oxidizing fatty acids generates more metabolic heat (~2 kcal/g or 13% on an energetic basis) compared to glucose. Therefore, during heat stress, preventing or blocking adipose mobilization/breakdown and increasing glucose "burning" is presumably a strategy to minimize metabolic heat production.

For dairy cattle, the mammary gland requires glucose to synthesize milk lactose and lactose is the primary osmoregulator and thus determinant of milk volume. However, in an attempt to generate less metabolic heat, the body (primarily skeletal muscle) appears to utilize glucose at an increased rate. As a consequence, the mammary gland may not receive adequate amounts of glucose and thus mammary lactose production and subsequent milk yield is reduced. This may be the primary mechanism which accounts for the additional reductions in milk yield beyond the portion explained by decreased feed intake (Figures 1 and 2).

Heat-stressed cattle require special attention with regards to heat abatement and other dietary considerations (i.e. concentrate:forage ratio, HCO_3^- etc; Baumgard et al., 2007). In addition they may also have an extra requirement for dietary or rumen-derived glucose precursors. Of the three main rumen-produced VFA's, propionate is the VFA primarily converted into glucose by the liver. One option to increase rumen propionate production is by feeding highly fermentable starches. However, this strategy may be risky as heat-stressed cattle are already susceptible to rumen acidosis. Further research is needed to identify safe methods of increasing dietary or rumen derived glucose precursors during heat stress conditions.

Summary

Clearly heat-stressed cattle implement a variety of post-absorptive changes in both carbohydrate and lipid metabolism (i.e. increased insulin action) that would not be predicted based upon their energetic state. The primary end result of this altered metabolic condition is that heat-stressed cattle have an extra need for glucose (theoretically due to its preferential oxidization in order to reduce metabolic heat). Therefore, any dietary component that increases propionate production (the primary precursor to hepatic glucose production), without reducing rumen pH, will probably increase production.

Note: This article has been partially adapted from a paper first published by the authors in the Proceedings in the 2007 University of Arizona Southwest Nutrition Conference.

References

Armstrong, D.V. 1994. Heat stress interaction with shade and cooling. J. Dairy Sci. 77:2044-2050.

Baldwin, R.L., N.E. Smith, J. Taylor and M. Sharp. 1980. Manipulating metabolic parameters to improve growth rate and milk secretion. J. Anim. Sci. 51:1416-1428.

Bauman, D.E. and W.B. Currie.1980. Partitioning of nutrients during pregnancy and lactation: a review of mechanisms involving homeostasis and homeorhesis. J. Dairy Sci. 63:1514-1529.

Bauman, D.E., C.J. Peel, W.D. Steinhour, P.J. Reynolds, H.F. Tyrrell, C. Brown and G.L. Harland. 1988. Effect of bovine somatotropin on metabolism of lactating dairy cows: influence on rates of irreversible loss and oxidation of glucose and nonesterified fatty acids. J. Nutr. 118:1031-1040.

Baumgard, L.H., C.E. Moore and D.E. Bauman. 2002. Potential application of conjugated linoleic acids in nutrient partitioning. Proc. Southwest Nutr. Conf. pp 127-141.

Baumgard, L.H., L.J. Odens, J.K. Kay, R.P. Rhoads, M.J. VanBaale and R.J Collier. 2006. Does negative energy balance (NEBAL) limit milk synthesis in early lactation? Proc. Southwest Nutr. Conf. 181-187.

Baumgard, L.H. and R.P. Rhoads. 2007. The effects of hyperthermia on nutrient partitioning. Proc. Cornell Nutr. Conf. pp 93-104.

Beam, S.W. and W.R Butler. 1999. Effects of energy balance on follicular development and first ovulation in postpartum dairy cows. J. Reprod. Fertility 54:411-424.

Berg, J.M., J.L. Tymoczko and L. Stryer. Biochemistry. 6th Edition. W.H. Freeman.

Collier, R.J., L.H. Baumgard, A.L. Lock and D.E. Bauman. 2005. Physiological Limitations: nutrient partitioning. Chapter 16. In: Yields of farmed Species: constraints and opportunities in the 21st Century. Proceedings: 61st Easter School. Nottingham, England. J. Wiseman and R. Bradley, eds. Nottingham University Press, Nottingham, U.K. 351-377.

Collier, R.J. and D.K. Beede. 1985. Thermal stress as a factor associated with nutrient requirements and interrelationships. In *Nutrition of Grazing Ruminants*. (ed) by L. McDowell. Academic Press, New York, NY. pp 59-71.

Drackley, J.K. 1999. Biology of dairy cows during the transition period: the final frontier? J. Dairy Sci. 82:2259-2273.

Fox, D.G. and T.P. Tylutki. 1998. Accounting for the effects of environment on the nutrient requirements of dairy cattle. J. Dairy Sci. 81:3085-3089.

Fuquay, J.W. 1981. Heat stress as it affects production. J. Anim. Sci. 52:167-174.

Goff, J.P. and R.L. Horst. 1997. Physiological changes at parturition and their relationship to metabolic disorders. J. Dairy Sci. 80:1260-1268.

Lucy, M.C., C.R. Staples, W.W. Thatcher, P.S. Erickson, R.M. Cleale, J.L. Firkins, J.H. Clark, M.R. Murphy and B.O. Brodie.1992. Influence of diet composition, dry matter intake, milk production and energy balance on time of postpartum ovulation and fertility in dairy cows. Anim. Prod. 54:323-331.

McDowell, R.E., E.G. Moody, P.J. Van Soest, R.P. Lehmann and G.L. Ford. 1969. Effect of heat stress on energy and water utilization of lactating cows. J. Dairy Sci.. 52: 188-194.

Mitlöhner, F.M., J.L. Morrow, J.W. Dailey, S.C. Wilson, M.L. Galyean, M.F. Miller and J.J. McGlone. 2001. Shade and water misting effects on behavior, physiology, performance, and carcass traits of heat-stressed feedlot cattle. J. Anim. Sci. 79:2327-2335.

Moore, C.E., J.K. Kay, M.J. VanBaale and L.H. Baumgard. 2005a. Calculating and improving energy balance during times of nutrient limitation. Proc. Southwest Nutr. Conf: 173-185.

Moore, C.E., J.K. Kay, M.J. VanBaale, R.J. Collier and L.H. Baumgard. 2005b. Effect of conjugated linoleic acid on heat stressed Brown Swiss and Holstein cattle. J. Dairy Sci. 88:1732-1740.

National Research Council. 2001. Nutrient Requirements of Dairy Cattle, 7th rev. ed. Nat. Acad. Press, Washington, DC.

Rhoads, M.L, R.P. Rhoads, S.R. Sanders, S.H. Carroll, W.J. Weber, B.A. Crooker, R.J. Collier, M.J. VanBaale and L.H. Baumgard. 2007. Effects of heat stress on production, lipid metabolism and somatotropin variables in lactating cows. J. Dairy Sci. 90. Suppl. (1):230 (abst.).

St. Pierre, N.R., B. Cobanov and G. Schnitkey. 2003. Economic losses from heat stress by US livestock industries. J. Dairy Sci. 86:E52-E77.

VanBaale, M.J., J.F Smith, M.J. Brouk and L.H. Baumgard. 2005. Evaluate the efficacy of your cooling system through core body temperature. Hoards Dairyman: Western Dairy News. Aug 5:W147-W148.

West, J.W. 2002. Physiological effects of heat stress on production and reproduction. Proc. Tri-State Nutr. Conf. 1-9.

West, J.W. 2003. Effects of heat-stress on production in dairy cattle. J. Dairy Sci. 86:2131-2144.

Wheelock, J.B., S.R. Sanders, G. Shwartz, L.L. Hernandez, S.H. Baker, J.W. McFadden, L.J. Odens, R. Burgos, S.R. Hartman, R.M. Johnson, B.E. Jones, R.J. Collier, R.P. Rhoads, M.J. VanBaale and L.H. Baumgard. 2006. Effects of heat stress and rbST on production parameters and glucose homeostasis. J. Dairy Sci. 89. Suppl. (1):290-291 (abst.).

SESSION NOTES