Feeding and Managing Cows to Minimize Heat Stress

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Abstract

Environmental-induced hyperthermia compromises efficient animal production and jeopardizes animal welfare. Reduced productive output during heat stress was traditionally thought to result from decreased nutrient intake. Our observations challenge this dogma and indicate heat-stressed animals employ novel homeorhetic strategies to direct metabolic and fuel selection priorities independently of nutrient intake or energy balance. Thus, the heat stress response markedly alters post-absorptive carbohydrate, lipid, and protein metabolism, independently of reduced feed intake through coordinated changes in fuel supply and utilization by multiple tissues. There may be nutritional, pharmaceutical, and managerial options to take advantage of these aforementioned metabolic changes to improve productivity and animal welfare during the warm summer months.

Introduction

The term "stress" is defined in different ways, but it is used to describe influences outside of a body system, which can shift the internal mechanisms away from their normal or resting state (Lee, 1965). Therefore, the term heat stress is used to describe the effects of increasing environmental temperature on different physiological systems. This is of interest to the dairy industry because of the detrimental changes (production, metabolic, and reproductive) induced by heat stress (West, 2003; Bernabucci et al., 2005).

Heat stress negatively impacts a variety of dairy parameters, including milk yield, milk quality and composition, rumen health, growth, and reproduction, and therefore it is a significant financial burden (~$900 million/year for dairy 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 heat stress on cattle, but productivity continues to decline during the summer. In the upper Midwest, heat-induced poor reproduction may be the costliest issue. For example, pregnancy rates at the Iowa State University Dairy decreased 19% during the 2010 summer and did not return to spring levels until the middle of December.

Biological Consequences 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

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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 (Rhoads et al., 2009b; Shwartz et al., 2009; Wheelock et al., 2010).

Reductions in energy intake during heat stress results in a majority of dairy cows entering into a negative energy balance **(NEBAL)**, regardless of the stage of lactation. Essentially, 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), and 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 are mediated by the reduction in EBAL (similar to the transition period). However, it is not clear how much of the reduction in performance (milk yield 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).

Rumen health

The heat-stressed cow is prone to rumen acidosis, and many of the lasting effects of warm weather (laminitis, low milk fats, etc.) can probably be traced back to a low rumen pH during the summer months. This may be explained by increased respiration rate, which results in enhanced carbon dioxide **(CO_2)** exhalation. In order to be an effective blood pH buffering system, the body needs to maintain a 20:1 bicarbonate **(HCO_{3-})** to CO_2 ratio. Due to the hyperventilation induced decrease in blood CO_2, the kidney secretes HCO_{3-} to maintain this ratio. This reduces the amount of HCO_{3-} that can be used (via saliva) to buffer and maintain a healthy rumen pH. In addition, the heat-stressed cow ruminates less (because of the reduced feed intake and increased time respiring), and rumination is a key stimulator of saliva production. Furthermore, heat-stressed cows drool, and this, coupled with reduced saliva production, reduces the amount of buffering agents entering the rumen. Consequently, care should be taken when feeding "hot" rations during the summer months. In addition, fiber quality is important all the time, but it is paramount during the summer as it has some buffering capacity and stimulates saliva production (Baumgard and Rhoads, 2007).

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). 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., 2005). 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. One classic response is a reduction in circulating insulin, coupled with a reduction in systemic insulin sensitivity. The reduction in insulin action activates adipose lipolysis, leading to the 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 reduced insulin action during NEBAL which results in reduced glucose uptake by systemic tissues (i.e., muscle and adipose). 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. The thermal-neutral cow in NEBAL is metabolically flexible and can depend upon alternative fuels (NEFA and ketones) to spare glucose (Baumgard and Rhoads, 2013). Glucose can then be utilized by the mammary gland to copiously produce milk (Bauman and Currie, 1980).

Heat Stress and Production Variables

Heat stress reduces feed intake 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 reduced 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 have conducted experiments involving a group of thermal neutral pair-fed animals to eliminate the confounding effects of dissimilar nutrient intake. The pair-feeding model is necessary in order to differentiate between the direct and indirect effects of heat stress (mediated by reduced feed intake) on production and metabolism. Utilizing this model has allowed us to determine that the heat-induced decrease in nutrient intake only accounts for approximately 50% of the decrease in milk yield (Figures 1 and 2: Rhoads et al., 2009a or b; Wheelock et al., 2010). The model indicates that direct effects of heat explain ~50 to 60% of decreased milk synthesis. Therefore, identifying hyperthermia-induced direct changes is likely a prerequisite to develop mitigation strategies to maximize milk yield during the warm summer months.

Pre-partum heat stress

The effects of heat stress during established lactation are well-characterized (Baumgard et al., 2012; Baumgard and Rhoads, 2013), but the effects of environmental hyperthermia prior to calving on post-parturition production parameters is not as clear. It was demonstrated that heat stress during the "far-off" period reduced gestation length, and calf body weight and subsequent milk yield, even in intensely cooled cows following calving (Collier et al., 1982). This has recently been confirmed and results indicate that future milk production is substantially reduced in heat-stressed dry cows (Tao et al., 2012). Interesting, it appears that the heat-induced blunted adipose tissue mobilization "lingers" into lactation, and dry cows that were heat-stressed were not able to enlist glucose sparing mechanisms necessary to support maximum milk yield, even though they were intensely cooled after calving (Tao et al., 2012). In addition, future reproductive variables are determinately affected in heat-stressed dry cows (even if they were intensely cooled during lactation; Wiersma and Armstrong 1989). Consequently, actively cooling dry cows should be an important part of a farm's heat stress abatement strategy.

Theoretical Reasons for Altered Metabolism

Well-fed ruminants primarily oxidize acetate (a rumen produced VFA) as a principal energy source. During NEBAL, cattle increased their energy dependency on NEFA. However, despite the fact that heat-stressed cows have marked reductions in feed intake and are losing considerable amounts of body weight, they do not mobilize adipose tissue (Rhoads et al., 2009; Wheelock et al., 2010). Therefore, it appears that heat stressed cattle experience altered post-absorptive metabolism compared to thermal neutral counterparts, even though they are in a similar negative energetic state. The unusual lack of NEFA response in heat-stressed cows is probably in part explained by increased circulating insulin levels (O'Brien et al., 2010; Wheelock et al., 2010), as insulin is a potent anti-lipolytic hormone. Increased circulating insulin during heat stress is unusual as malnourished animals are in a catabolic state and experience decreased insulin levels. The increase in insulin action may also explain why heat-stressed animals have increased rates of glucose disposal (Wheelock et al., 2010). Therefore, during heat stress, preventing or blocking adipose mobilization/breakdown and increasing glucose "burning" is presumably a strategy to minimize metabolic heat production (Baumgard and Rhoads, 2007).

The increase in extra-mammary glucose utilization during heat stress creates a nutrient trafficking problem with regards to milk yield. The mammary gland requires glucose to synthesize milk lactose, and lactose is the primary osmoregulator, thus determines overall milk volume. However, in an attempt to generate less metabolic heat, the body (presumably skeletal muscle) appears to utilize glucose at an increased rate. Therefore, the mammary gland may not receive adequate amounts of glucose, and as a result, mammary lactose production and subsequently 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 Stress and Immunity

The metabolic profile of heat-stressed cattle shares some similarities to animals with a stimulated immune system, and this is primarily characterized by hyperinsulinemia (Baumgard and Rhoads, 2013). The increased circulating insulin during heat stress is unusual because reduced feed intake, negative energy balance ,and body weight loss (hallmarks of heat stress) are typically associated with hypoinsulinemia. Incidentally, lactating cows with an activated immune system also have increased circulating insulin concentrations, despite reduced feed intake (Waldron et al., 2006). Reasons for the hyperinsulinemia are not clear but may include lipopolysaccharide **(LPS)**, an endotoxin produced by gram-negative bacteria. We have demonstrated that cattle IV infused with LPS have marked (>50 fold) hyperinsulinemia 2 hr after LPS administration (Baumgard and Rhoads, 2013; Figure 3). Interestingly, the severe increase in insulin following LPS injection only causes minor hypoglycemia, and this likely means that LPS causes insulin resistance. Heat-stressed rodents, poultry, pigs, and humans have increased levels of circulating LPS because of intestinal integrity issues, and presumably, heat-stressed cattle do as well. Insulin's role during the immune response and during heat acclimation is not clear, but proper insulin action is necessary in order to up-regulate heat shock proteins.

Heat Stress Abatement

Heat abatement strategies are often employed as a means to ameliorate the negative

effects of heat stress on production during the warm summer months (VanBaale et al., 2005). Cooling cows with shade and evaporative cooling with soakers and fans is a relatively cheap strategy to help minimize economic losses during an increased heat load (Collier et al., 2006). However, despite new barn construction and heat abatement systems, milk yield and other production parameters continue to be adversely affected by heat stress (Burgos et al., 2007).

Feedstuffs have varying heat increments **(HI)**, largely due to efficiency of nutrient utilization or digestive end products (VanSoest et al., 1991). Fiber digestion results in a higher HI (sum of heat produced from rumen fermentation and nutrient metabolism) than digestion of fat or non-fiber carbohydrates **(NFC)**. The major end-product of fiber fermentation (acetate) is utilized less efficiently compared to the major end-product of NFC digestion (propionate; Baldwin et al., 1980).

Table 1 illustrates heat increments of several common feedstuffs. The heat increment value expressed as Kcal/Mcal, net energy lactation **(NE_L)** was derived for total digestible nutrient **(TDN)** values of 40 to 100% and fitted to a multiple linear regression model: $y = a+bx+cx^2$, where y = Kcal HI/Mcal NE_L; x = TDN; and solved constants are a = 1350.812, b = -17.1496, and c = 0.091517 (Chandler, 1994).

Nutritional Strategies of Heat Stress

There are several nutritional strategies to consider during heat stress. A common strategy is to increase the energy and nutrient densities (reduced fiber, increased concentrates, and supplemental fat) of the diet, as feed intake is markedly decreased during heat stress. In addition to the energy balance concern, reducing the fiber content of the diet is thought to improve the cow's thermal balance and may reduce body temperature. However, increasing ration concentrates should be considered with care as heat-stressed cows are highly prone to rumen acidosis.

Fiber

Fiber is necessary for proper rumen function; current recommendations state a minimum dietary neutral detergent fiber **(NDF)** of 25% with the proportion of NDF from roughages equaling 75% of total NDF (NRC, 2001). However, its digestion and metabolism create more heat than compared to concentrates (Van Soest et al., 1991). One common nutritional strategy involves reducing dietary fiber during an increased heat-load. However, adequate fiber in the diet is essential to maintain rumen health, and high quality forage helps to maintain feed intake. Grant (1997) demonstrated that a roughage NDF value of 60% still provides sufficient fiber for production of fat-corrected milk. On the other hand, Kanjanapruthipong and Thaboot (2006) speculated that the minimum dietary NDF of 23% DM and roughage NDF proportion of 55% dietary NDF have sufficient effective NDF for dairy cows in the tropics.

Protein

It is thought that because of reduced feed intake, dietary protein levels may need to be increased during heat stress (West, 1999). However, there are inconsistencies within the literature as benefits and negative consequences of increased protein and altered protein solubility have both been reported (Huber et al., 1993, 1994). The negative effects of increased dietary protein agrees with recent recommendations which suggest that addition of dietary CP, more specifically rumen un-

degradable protein, is not helpful during heat stress (Arieli et al., 2006). A possible reason why highly degradable protein diets appear to be deleterious during heat stress is that both rumen motility and rate of passage decline. This allows for a longer residence time and thus more extensive protein degradation (Linn, 1997). We have demonstrated that blood urea nitrogen is elevated in heat-stressed cows compared to pair-fed controls (Wheelock et al., 2010), although it is not clear whether this originates from excess rumen ammonia production or from skeletal muscle breakdown. Regardless, excess ammonia needs to be eliminated and this removal has an energy cost (7.2 kcal/g of nitrogen; and this increases heat production) as it is metabolized to urea and excreted in the urine (Tyrell et al., 1970). How heat stress affects dietary protein requirements is ill-defined and more research is needed in order to generate more appropriate recommendations.

Fat

Increasing the amount of dietary fat has been a widely accepted strategy within the industry in order to reduce basal metabolic heat production. As stated above, the heat increment of fat is over 50% less than typical forages (Table 1), so it is seemingly a rational decision to supplement additional lipid and reduce fiber content of the diet. However, there are surprisingly few experiments specifically designed to evaluate how supplemental dietary fat affects body temperature indices or even production parameters (Table 2). Most experiments report little or no differences in rectal temperatures (Moody et al., 1967; Knapp and Grummer 1991; Chan et al., 1997; Drackely et al., 2003) and only one paper demonstrated a slight reduction at a specific time of day but not at the other times (Wang et al., 2010). In fact, one report indicated that cows fed additional fat actually had increased rectal temperatures (Moallem et al., 2010), and these same authors and a recent report (Wang et al., 2010) indicate that additional fat-fed cows had increased respiration rates. A reason why feeding fat does not seemingly improve the thermal balance of heat-stressed cows is difficult to rationalize. It could be that small decreases in a thermal load would be difficult to detect at specific but limited time points, but that these minor changes would accumulate over time into a significant improvement. It would be of interest to evaluate body temperatures in heat-stressed cows fed additional fat utilizing a continuous thermometer system (i.e., HOBOs or eye-button technology).

Additional fat feeding can sometimes decrease DMI in thermal neutral cows (Chillard, 1993), but reduced nutrient intake is typically not observed in heat-stressed cows fed supplemental fat (Moody et al., 1967; Skaar et al., 1989; Knapp and Grummer, 1991; Drackley et al., 2003; Warntjes et al., 2008; Wang et al., 2010). Milk yield responses to additional fat are variable and some authors report no diet effect (Moody et al., 1967; Knapp and Grummer, 1991; Chan et al., 1997; Moallem et al., 2010), while others report an increase in milk yield (Skaar et al., 1989; Drackley et al., 2003; Warntjes et al., 2008; Wang et al., 2010). Similar to body temperature indices and milk yield data, the effects of dietary fat on milk composition during heat stress also vary and no clear consensus has been reached (Table 2). Overall, results from a limited number of experiments vary, but little or no apparent benefit was typically observed when supplemental dietary fat was included. Reasons for the discrepancies are unclear, but this could be due to the type of fats used (saturated vs. unsaturated), rate of inclusion, type of "protection" (i.e. calcium salt vs. prill), environmental factors (i.e. severity of heat stress), or other dietary interactions.

Regardless, the dairy industry (nutritionists) needs additional controlled experiments (besides theoretical heat calculations) in order to make intelligent ration balancing decisions regarding the inclusion of supplemental fat.

Ionophores

We propose enhanced extra-mammary tissue glucose utilization may be a key mechanism explaining the decrease in milk yield during heat stress. Two glucose molecules are the substrate for lactose (the primary osmotic regulator of milk yield) synthesis and on a molar basis, lactose is nearly equivalent (95%) to two moles of glucose. Heat-stressed cows secrete about 370 g less lactose (Rhoads et al., 2009) or have approximately twice as much of a decrease in milk lactose yield as pair-fed thermal neutral controls (Wheelock et al., 2010). Therefore, heat-stressed cows in our previous experiments are secreting almost 400 g/day less glucose than thermal neutral counterparts on a similar plane of nutrition. Monensin is a well-described rumen modifier that increases the production of propionate, which is the predominate gluconeogenic precursor in ruminants. The increase in carbon conservation during fermentation is a key mechanism in how monensin increases feed efficiency in growing and lactating ruminants. We have now demonstrated that monensin increases the gluconeogenic rates (on a DMI basis), and utilizing monensin is a key strategy to improve the glucose status of heat-stressed cows (Baumgard et al., 2011).

Water

Water intake is vital for milk production (milk is ~87% water), but it is also essential for thermal homeostasis. This stresses how important water availability and waterer/tank cleanliness becomes during the summer months. Keeping water tanks clear of feed debris and algae is a simple and cheap strategy to help cows remain cool (Baumgard and Rhoads, 2007).

*Dietary cation-anion difference **(DCAD)***

Having a negative DCAD during the dry period and a positive DCAD during lactation is a good strategy to maintain health and maximize production (Block, 1994). It appears that keeping the DCAD at a healthy lactating level (~+20 to +30 meq/100 g DM) remains a good strategy during the warm summer months (Wildman et al., 2007).

Minerals

Unlike humans, bovines utilize potassium **(K+)** as their primary osmotic regulator of water secretion from sweat glands. As a consequence, K+ requirements are increased (1.4 to 1.6% of DM) during the summer, and this should be adjusted for in the diet. In addition, dietary levels of sodium (Na+) and magnesium **(Mg+)** should be increased as they compete with K+ for intestinal absorption (West, 2002).

Summary

Heat stress negatively impacts economic parameters associated with profitable milk production. Implementing heat stress abatement strategies is crucial to minimize fiscal losses. In addition to physical barn management, nutritional strategies can be implemented to help ameliorate summer-induced losses. Maintaining rumen health is of primary importance as heat-stressed cows are more prone (for a variety of reasons) to rumen acidosis. Another widely held dogma is that supplementing dietary fat is an effective tactic during heat stress, and this stems from

theoretical calculations indicating that the heat increment of feeding is much lower for lipids (especially compared to roughages). However, a review of the limited literature fails to corroborate the arithmetic heat savings or ultimately demonstrate a consistent effect on production parameters. The dairy industry needs definitive research on whether or not to include supplemental fat during the warm summer months.

*Aspects of this manuscript were adapted from the 2011 4-State Nutrition Conference proceedings paper (Nayeri et al., 2011).

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Table 1. Heat increment (HI) of common feed ingredients.[1]

Feed Ingredient	DM (%)	NDF (% of DM)	TDN (% of DM)	NE_L (Kcal/Kg)	HI/NE_L (Kcal/Mcal)
Haylage	35.0	53.0	59.0	1,326	658
Corn Silage	38.3	48.0	66.1	1,500	617
Grass Hay	88.0	53.0	55.0	1,228	684
Alfalfa Hay	89.9	47.5	60.0	1,350	651
Whole Cottonseed	93.0	49.0	87.0	2,453	386
Corn	87.0	10.0	88.0	2,035	550
Soybean Meal, 48% CP	90.0	14.0	81.0	1,866	562
Palm Oil (fatty acids)	100.0	0.0	170.1	5,676	214
Prill (fatty acids)	100.0	0.0	170.1	6,776	214
Tallow	99.0	0.0	191.3	6,402	214

[1]Adapted from Chandler, 1994.

Table 2. Effects of supplemental dietary fat on production parameters in lactating cows.

Reference	Fat Type	RT	RR	DMI	FE	MY	MF	MP	Metabolites
1	SFA/UFA	↑	↑	↓	↑	↔	↑	↔	↑ NEFA
2	SFA	↓	↔	↔	↑	↑	↑	↑	↓NEFA
3	SFA	NM	NM	↔	↔	↑	↓	↑	NM
4	LCFA	↔	↔	↔	↑	↑	↔	↓	↓NEFA
5	SFA	↔	↔	↔	↔	↔	↔	↔	NM
6	LCFA/Tallow	↔	↔	↔	↔	↔	↔	↔	NM
7	SFA	NM	NM	↔	↔	↑	↔	↔	↔
8	SFA/UFA	↔	↔	↔	↔	↔	↔	↔	↔

NM: Not Measured
↑: Increase
↓: Decrease
↔: No Change
SFA: Saturated Fatty Acids
UFA: Unsaturated Fatty Acids
LCFA: Long-Chain Fatty Acids

RT: Rectal Temperature
RR: Respiratory Rate
DMI: Dry matter intake
FE: Feed Efficiency
MY: Milk Yield
MF: Milk Fat
MP: Milk Protein
NEFA: Non-Esterified Fatty Acids

1 Moallem et al., 2010
2 Wang et al., 2010
3 Warntjes et al., 2008
4 Drackley et al., 2003
5 Chan et al., 1997
6 Knapp and Grummer, 1991
7 Skaar et al., 1989
8 Moody et al., 1967

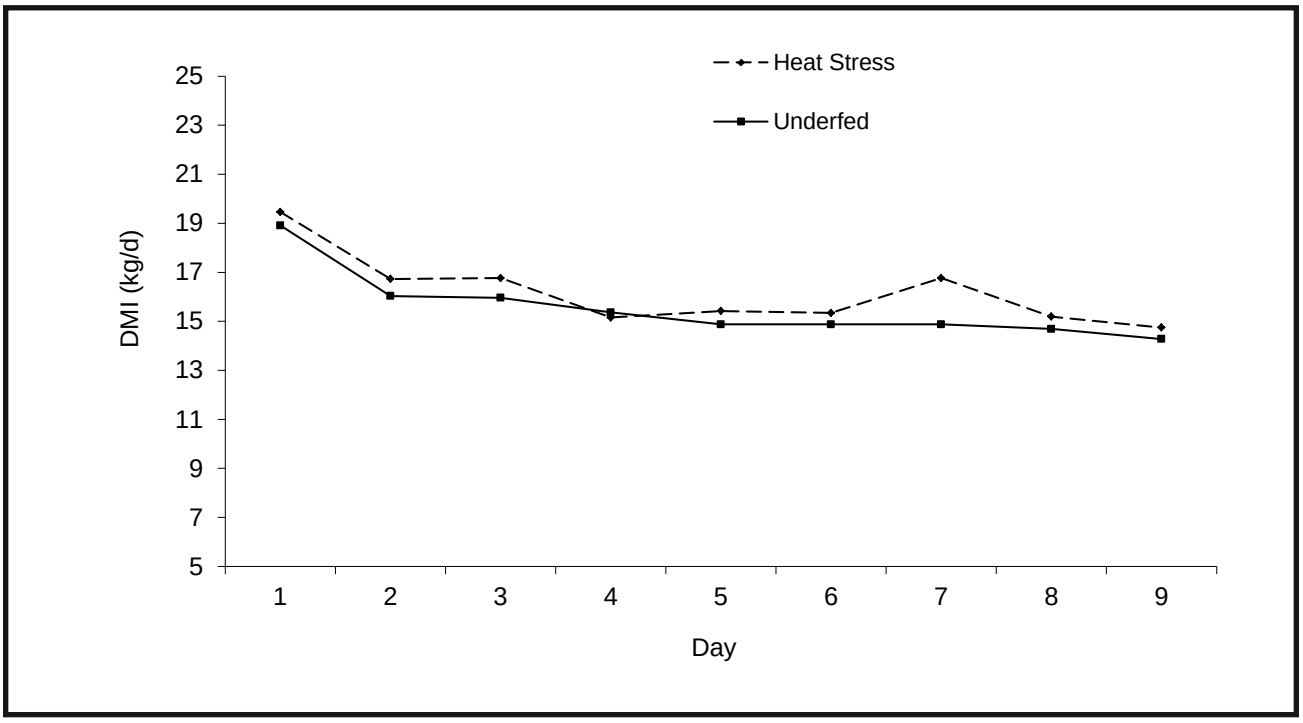

Figure 1. Effects of heat stress and underfeeding (pair-feeding) thermal-neutral lactating Holstein cows on dry matter intake (Rhoads et al., 2009).

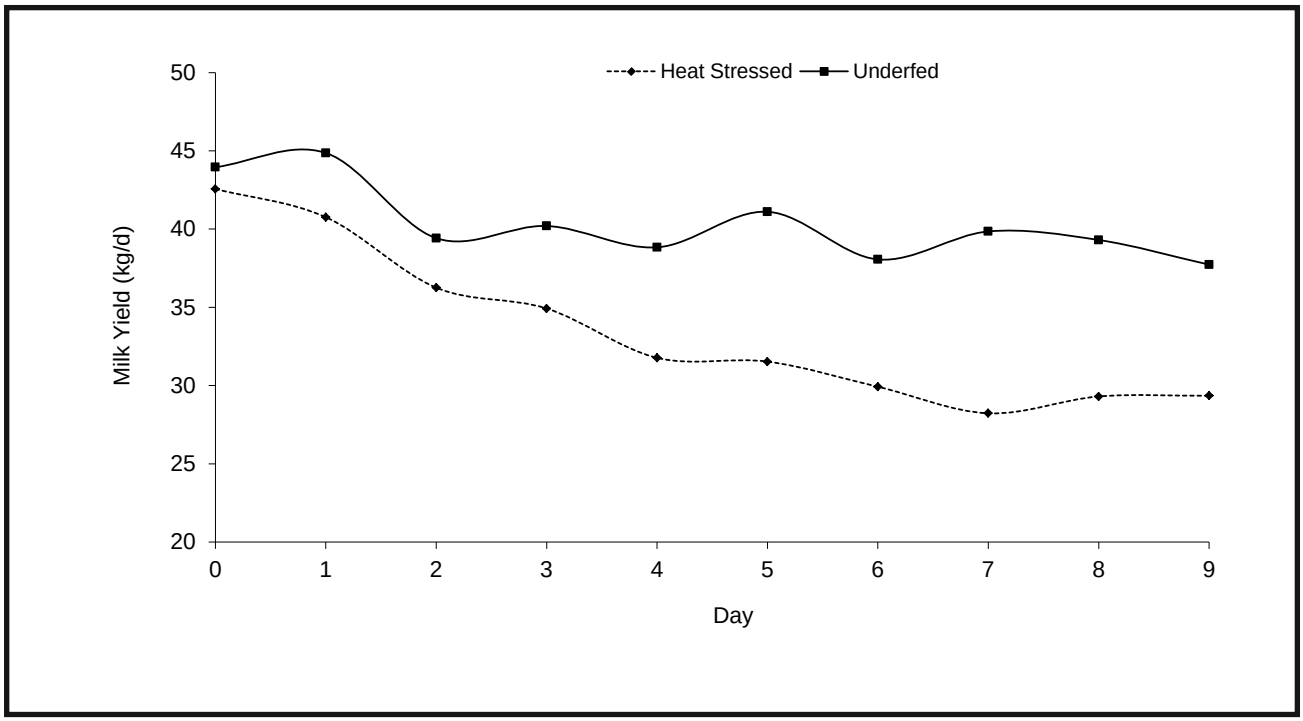

Figure 2. Effects of heat stress and underfeeding (pair-feeding) in thermal neutral conditions on milk yield in lactating Holstein cows (Rhoads et al., 2009).

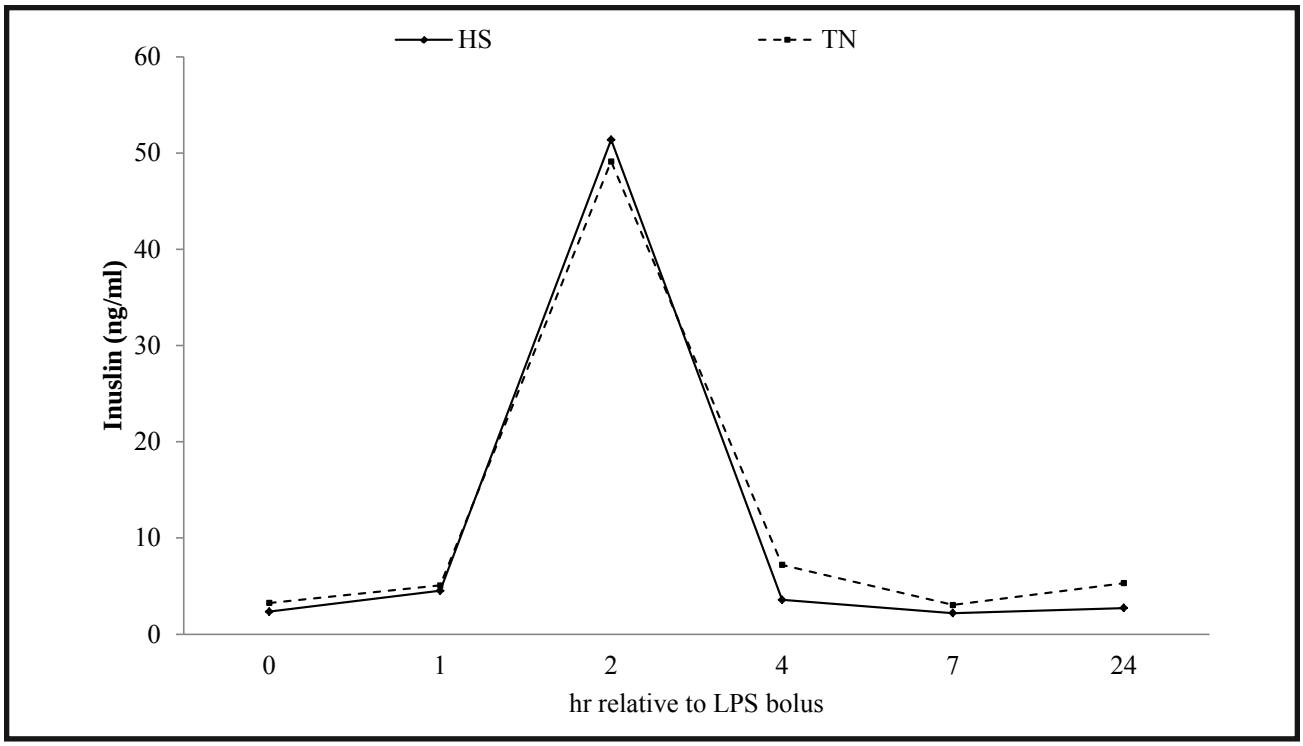

Figure 3. Effects of LPS infusion on blood insulin concentrations in growing Holstein calves either in heat-stressed (HS) or thermal neutral (TN) conditions (Baumgard and Rhoads, 2013).