Relationship of Thermal Status to Productivity in Heat-Stressed Dairy Cows Given Recombinant Bovine Somatotropin¹

R. S. Settivari,* J. N. Spain,* M. R. Ellersieck,† J. C. Byatt,‡ R. J. Collier,§ and D. E. Spiers*²

*Division of Animal Sciences, and

†Agricultural Experiment Station, University of Missouri-Columbia, Columbia 65211

‡Department of Animal Sciences, University of Florida, Gainesville 32611

§Department of Animal Sciences, University of Arizona, Tucson 85721

ABSTRACT

The responses of lactating Holstein cows to daily administration of bovine somatotropin (bST) were measured at thermoneutrality (T_n) and under both constant and cycled heat-stress conditions to determine the relationship between thermal status and bST-induced shifts in milk production. All tests included a 5-d acclimation period at T_n (18°C), followed by a 2-d increase in ambient temperature to 28.5°C. After d 3, ambient temperature was cycled between 28.5 (day) and 25.5°C (night) for 4 d. Daily injections with either 31 mg of bST or saline began on d 1 of the experiment. Milk production, feed intake, and respiratory rate (RR) were measured daily. Intraperitoneal, telemetric temperature transmitters were used for a continuous measure of core body temperature (T_{core}). Blood samples were collected during each phase to evaluate the changes in serum chemistry in response to bST and heat stress. Following a 15-d recovery, cows were switched across injection treatments and the study was repeated. Milk production decreased by ~18.4% below the initial yield at T_n by the end of 7 d of heat challenge. Although a reduction in milk production occurred during heat stress in both groups, milk production was higher in bST-treated cows compared with control cows during periods of constant and cyclic heat. Likewise, bST treatment during the entire period increased the milk-to-feed ratio over the control level by ~11.3%. Plasma insulin-like growth factor 1 and serum nonesterified fatty acids accompanied the increased growth hormone level with bST treatment (~122.0 and 88.8%, respectively), whereas plasma urea nitrogen was reduced by ~13.3% to reflect the shift to lipid metabolism. There was no difference in T_{core} of the treatment and control groups at T_n. Both bST and control cows increased RR and

T_{core} above the T_n level by ~94.8 and 2.9%, respectively, during constant heat, with a greater increase in T_{core} of bST-treated compared with control cows (~0.6%). The increase in RR during heat stress preceded T_{core} by 1 d for both groups. During cyclic heat, T_{core} decreased by ~0.4% compared with constant heat in both the control and bST-treated groups. Bovine somatotropin treatment increased milk production similarly during the T_n and heat-stress periods, ~8.3% over the control; however, the bST-induced increase in milk-to-feed ratio was greatest during the continuous and cyclic heat-stress phases, ~16.2%. This increase occurred together with the elevation in T_{core}.

Key words: body temperature, growth hormone, heat, milk production

INTRODUCTION

Exposure to severe heat-stress conditions decreases the thermal gradient between an animal and the environment, which minimizes nonevaporative heat dissipation. Exposure of lactating dairy cows to heat stress ultimately produces a collective reduction in the rates of metabolism, feed intake, and milk production, and an increase in the respiratory rate (RR) and core body temperature (T_{core}) that results in a significant loss of productivity during the summer months (Armstrong, 1994).

Recombinant bST is a synthetically derived hormone that is virtually identical to naturally occurring bovine growth hormone (GH). Exogenous administration of bST enhanced the galactopoietic performance of dairy cows in laboratory and field environments, with a greater peak milk yield and increased persistency of yield over the lactation cycle (Etherton and Bauman, 1998). Nevertheless, high-producing and multiparous cows are known to be extremely susceptible to heat stress (Armstrong, 1994), and bST-injected animals might therefore be more sensitive to the deleterious effects of heat stress than nontreated animals.

Information on the shift in thermoregulatory ability of bST-injected cows during heat stress is limited. Ma-

Received July 8, 2006.

Accepted October 3, 2006.

¹Contribution from a USDA-BARD Cooperative Agreement and the Missouri Agricultural Experiment Station.

²Corresponding author: spiersd@missouri.edu

nal et al. (1991) reported that bST increased evaporative heat loss in lactating dairy cows, which helped to dissipate the increased heat load associated with increased milk yield. Nonetheless, many of the previous studies were performed using uncontrolled or constant heat-stress conditions, with limited data collected as representative measurements for the entire study. A detailed analysis of the blood chemistry, productivity, and thermoregulatory ability of dairy cows would help determine the beneficial effects of bST under thermoneutral (**T_n**) and heat-stress conditions.

The effect of heat stress on bST-injected cows was determined in the present study under **T_n** (18°C) and heat-stress (28.5°C) conditions, which included both constant and cycling phases. A reduction in nighttime temperature is known to increase the animal-to-ambient thermal gradient to augment heat dissipation. In addition, summer heat stress in many regions, especially in the Midwestern United States, is a series of repeating sessions of constant heat (1 to 2 wk) and cooler periods of the same duration. For these reasons, the effect of bST treatment in lactating dairy cows was examined during exposure to constant and cycling heat conditions, along with early recovery. Changes in blood chemistry were determined at regular intervals to identify the series of hormonal and metabolic shifts that accompany bST treatment under these environmental conditions.

MATERIALS AND METHODS

Animals

Eight lactating Holstein cows (611.6 ± 44.5 kg of BW; 138.9 ± 6.4 DIM) from Foremost Dairy Farm at the University of Missouri-Columbia were placed in chambers in the Brody Environmental Center at the University. They were individually maintained in stanchions and fed a daily diet ad libitum (Table 1), offered over 2 feedings at 0800 and 2000 h. Refused feed was weighed and subtracted from the amount fed the previous day to derive the daily feed intake. Water was also provided ad libitum. Animal use in this study was approved by the University of Missouri-Columbia Animal Care and Use Committee.

Experimental Design

The treatment schedule consisted of 2 trials, with a crossover design, in which bST-treated animals in trial 1 were switched to control animals in trial 2, and vice versa for the control group in trial 1. Animals were initially implanted intraperitoneally with telemetric temperature transmitters (model VHF-T-1; Mini-Mitter, Sunriver, OR) to provide T_{core} . Following a 7-d re-

Table 1. Ingredients and chemical composition of the diet fed to dairy cows

Item	Amount, %
Feed ingredient	
Corn silage	32.0
Alfalfa silage	11.0
Chopped alfalfa hay	16.2
High-moisture shelled corn	12.8
Soybean meal, 48%	5.0
Soybean hulls	6.0
Corn gluten feed	9.6
Fish meal	0.5
Hydrolyzed animal fat ¹	0.7
Ground corn	5.1
Dicalcium phosphate	0.3
Salt	0.1
Dynamate ²	0.2
Limestone	0.3
Trace minerals ³	0.1
Vitamins ADE ⁴	0.06
Vitamin E ⁵	0.06
Chemical composition, ⁶ %	
OM	92.1
CP	16.6
ADF	24.5
NDF	40.0

¹Manufactured by Southwest By-Products (Springfield, MO).

²Trade name for a mineral supplement produced by IMC-Agrico (Mundelein, IL) containing a guaranteed minimum analysis of 22% sulfur, 18% potassium, and 11% magnesium.

³Mineral mixes were formulated to contain 12% Ca, 10% Fe, 8% Mn, 8% Zn, 2% CuSO₄, 200 mg/kg of Co, and 10,000 mg/kg of Se.

⁴Contains 88,200,000 IU/kg of vitamin A, 1,764,000 IU/kg of vitamin D₃, and 2,646 IU/kg of vitamin E.

⁵Contains 44,100 IU/kg of vitamin E.

⁶Mean dietary DM content was $56.4 \pm 2.8\%$.

covery period from surgery, cows were provided a 5-d acclimation to 18°C (**T_n**). During the treatment period, ambient temperature (**T_a**) was maintained at **T_n** for the first 3 d (Figure 1), followed by a 2-d increase to 28.5°C. Cows were maintained at this **T_a** for 3 d (d 7 to 9). Following the constant heat period, **T_a** was cycled for a 3.0°C daily reduction from the day to night level for 4 d (d 10 to 13). This allowed for determination of the benefit of a slight nighttime reduction in **T_a**. The **T_a** used in the present study was not severe. Starting on d 13, the **T_a** was gradually lowered over 2 d to 18°C and maintained at this level for 2 d (d 16 and 17) to measure the recovery response. Trial 2 was started following 15 d of recovery, when the **T_a** was maintained at 18°C. Chamber percent relative humidity was fixed at 50 to 60% during the entire experiment. Chamber humidity and **T_a** were measured every 30 min by using a XR330 pocket logger (Pace Scientific, Inc., Charlotte, NC) and were used to calculate the temperature-humidity index (**THI**; Thom, 1959).

Figure 1. The temperature-humidity index [THI, representing average air temperature ($^{\circ}\text{C}$) and relative humidity (%)] across chambers and periods, was estimated to identify the degree of heat stress. The dashed line (THI = 72) separates an animal's comfort zone (THI ≤ 72) from the stress zone (THI > 72). Tn = thermoneutrality.

Treatments

The 2 daily treatments were the control (1.0 mL of sterile water) and 31 mg of recombinant bST (V406-001), which was supplied by Monsanto (St. Louis, MO). The bST dose was split to observe the effects on performance during the day and night periods. Prior to injection, bST was reconstituted in 75 mM NaHCO_3 . A 3-mL syringe, with a 2.5-cm 20-gauge needle, was used to deliver 1.0 mL of solution. Storage was at -20°C . The contents of the syringe were thawed and injected subcutaneously in the cows twice daily (0930 and 2000 h) on alternative sides in the postscapular region, beginning on d 1 and continuing for 17 d.

Measurements

Core body temperature was measured every 10 min throughout the entire period. The RR was determined daily by counting flank movements for 60 s at 0800, 1200, and 2000 h. Both measures were used as indicators of the thermal status of the dairy cows, with RR being the more sensitive end point, especially during acute heat stress (Brown-Brandl et al., 2005). Cows

were milked twice daily, at 12-h intervals (0500 and 1700 h), using milking procedures outlined by the National Mastitis Council (<http://www.nmconline.org/milkprd.htm>). Milk was collected into a sanitized, stainless-steel milk can and weighed to measure daily milk production.

Blood Sampling and Analysis

Blood samples (18 mL) were collected by tail venipuncture on d -1 (pretreatment Tn period), 4 (preheat Tn period), 7 (constant heat period), 10 (early cycling heat period), 13 (late cycling heat period), and 15, 16, and 17 (postheat Tn period) at 0900 to 1000 h to analyze changes in metabolic status associated with bST and heat-stress treatments. Serum and plasma were separated and frozen to determine GH, prolactin, IGF-I, insulin, plasma urea nitrogen (PUN), NEFA, and glucose. Serum GH was measured by RIA using an antiserum raised in rabbits, as described earlier (Byatt et al., 1992). Insulin and IGF-I were measured as described by Vicini et al. (1988). Serum prolactin was measured as described previously (Byatt et al., 1994). The serum

NEFA concentration was measured with a NEFA-C kit (Wako Chemical Co., Dallas, TX). Both PUN and plasma glucose concentrations were measured using a Dimension Clinical Chemistry Analyzer (E.I. du Pont Nemours Co., Wilmington, DE; Byatt et al., 1992). Intraassay coefficients of variation for glucose, NEFA, PUN, prolactin, insulin, GH, and Igh-1 assays were 0.3, 1.8, 1.6, 6.6, 8.8, 9.0, and 9.3%, respectively, and interassay coefficients of variation were 0.7, 1.0, 3.3, 4.7, 8.0, 10.3, and 8.3%, respectively. Blood variable values during the postheat T_n period were averaged because mean values did not differ across the 3-d period.

Statistical Analysis

Mean group values were calculated for all measured variables as a function of time. Hourly average values were used for T_{core} analysis and daily averages were used for RR, feed intake, and milk production. All variables were analyzed as repeated-measures ANOVA using the MIXED procedure of SAS (SAS Institute, Inc., Cary, NC). Cow was considered random and all other model effects were considered fixed. Blood variables were analyzed for the effects of bST and heat-stress treatments and were compared among different phases. Significance levels for the main effects and their interactions were set at $P < 0.05$ for all analyses.

RESULTS

T_{core}

Determination of T_{core} began on the first day of bST treatment, and T_{core} from d 1 was therefore considered as the pretreatment period because the bST treatment did not have sufficient time to affect the T_{core}. The average T_{core} on d 1 did not differ ($P = 0.89$) between control ($38.65 \pm 0.10^\circ\text{C}$) and bST-treated ($38.66 \pm 0.10^\circ\text{C}$) cows (Figure 2A). Similarly, the T_{core} did not change ($P < 0.05$) between bST-treated and control cows during the preheat and postheat treatment periods, as well as during the transition periods. Heat stress increased ($P < 0.01$) the T_{core} in bST-treated and control cows during the constant and cyclic heat-stress periods compared with the preheat treatment period (Figure 2A). The first indications of a significant increase ($P < 0.01$) from the preheat treatment (d 3, last day of the preheat treatment period) appeared on d 6 in both groups and remained so until d 13. The T_{core} during constant heat ranged from 39.46 (d 7) to $39.77 \pm 0.12^\circ\text{C}$ (d 9) in control cows, whereas it was 39.74 (d 7) and $40.07 \pm 0.12^\circ\text{C}$ (d 9) in bST-treated cows. The nocturnal reduction in Ta lowered the T_{core} in both control and bST-treated cows from a constant heat level, with a T_{core} range of 39.59 (d 10) to $39.30 \pm 0.12^\circ\text{C}$ (d 13) in control cows and 39.87

(d 10) to $39.54 \pm 0.12^\circ\text{C}$ (d 13) in bST-treated cows. A gradual reduction in Ta during the postheat treatment reduced ($P < 0.01$) the T_{core} in both groups below the heat-stress level. During the postheat treatment, both bST-treated and control cows had lower T_{core} ($P < 0.04$) on d 15, 16, and 17 compared with during the preheat treatment (d 3), suggesting that heat-stress adaptation had altered the T_n response. Although the bST treatment increased the T_{core} ($P < 0.01$; $39.17 \pm 0.09^\circ\text{C}$) above the control level ($39.00 \pm 0.09^\circ\text{C}$) during the 17-d treatment period, bST and phase-interactive effects on T_{core} were absent ($P = 0.74$).

RR

The RR was not different ($P = 0.26$) between control (41.4 ± 2.1 breaths/min) and bST-treated (43.5 ± 2.1 breaths/min) cows during the pretreatment period (Figure 2B). Similarly, the RR did not change ($P > 0.05$) between these groups during the preheat and postheat periods, as well as during the transition periods. Heat stress increased ($P < 0.01$) the RR in both groups compared with the preheat period (d 3), with the first indication of a significant increase ($P < 0.05$) on d 5 and continuing until d 14 and 16 in the control and bST-treated groups, respectively. The RR was greater in bST-treated compared with control cows on d 9 ($P < 0.01$; constant heat stress) and d 11 ($P = 0.03$; cyclic heat-stress period). During constant heat, the RR ranged from 82.4 (d 7) to 83.3 ± 2.8 breaths/min (d 9) in control cows and 86.6 (d 7) to 92.4 ± 2.9 breaths/min (d 9) in bST-treated cows. The nighttime reduction in Ta stabilized the RR in both groups, from 86.3 (d 10) to 71.3 ± 2.9 breaths/min (d 13) in control cows, and 92.3 (d 10) to 74.3 ± 2.9 breaths/min (d 13) in bST-treated cows. A gradual reduction in RR was observed in both groups during the postheat period as Ta was decreased. As observed for T_{core}, bST treatment increased ($P < 0.01$) the RR in bST-treated (66.2 ± 1.6 breaths/min) compared with control (63.3 ± 1.6 breaths/min) cows during the 17-d treatment period; however, an interaction effect on RR between heat stress and bST was absent ($P = 0.65$).

Feed Intake

Feed intake values were not different ($P = 0.89$) between control (24.0 ± 0.8 kg/d) and bST-treated (24.0 ± 0.8 kg/d) cows during the pretreatment period. Similarly, feed intake did not change ($P > 0.05$) between bST-treated and control cows during the preheat, heat stress, and postheat periods (Figure 3A). Heat stress reduced ($P < 0.01$) feed intake in bST-treated and control cows during the constant and cyclic heat periods

Figure 2. (A) Average core body temperature of bST-treated and control cows during treatment as a function of time. The asterisk reflects a difference ($P < 0.05$) in the average core body temperature between bST-treated and control dairy cows for the day. (B) Average respiration rate of bST-treated and control cows during treatment as a function of time. Error bars on the mean values indicate $\pm$ SE. The asterisk shows a difference ($P < 0.05$) in the average respiration rate between bST-treated and control cows for the day. Tn = thermoneutrality.

Figure 3. (A) Average daily feed intake of bST-treated and control cows during treatment as a function of time. Error bars on the mean values indicate $\pm$ SE. (B) Average change in milk production of bST-treated and control cows during treatment as a function of time. Error bars on the mean values indicate $\pm$ SE. The asterisk shows a difference ($P < 0.05$) in average milk production between bST-treated and control cows for the day. Tn = thermoneutrality.

Figure 4. (A) Average change in a.m. milk production of bST-treated and control cows during treatment as a function of time. Error bars on the mean values indicate $\pm$ SE. The asterisk shows a difference ($P < 0.05$) in the average a.m. milk production between bST-treated and control cows for the day. (B) Average change in p.m. milk production of bST-treated and control cows during treatment as a function of time. Error bars on the mean values indicate $\pm$ SE. The asterisk shows a difference ($P < 0.05$) in average p.m. milk production between bST-treated and control cows for the day. Tn = thermoneutrality.

Figure 5. Average milk-to-feed ratio of bST-treated and control cows during treatment as a function of time. Average milk production efficiency was calculated as the ratio of kilograms of milk produced per kilogram of feed intake. Error bars on the mean values indicate $\pm$ SE. The asterisk shows a difference ($P < 0.05$) in average milk production efficiency between bST-treated and control cows for the day. Tn = thermoneutrality.

compared with the preheat period. The first indication of a significant reduction ($P < 0.01$) in feed intake from the preheat period (d 3) was on d 7 in both groups and remained until d 14, when heat stress was reduced. A large reduction in feed intake occurred in both groups during constant heat, ranging from 22.3 (d 7) to 18.5 ± 1.1 kg/d (d 9) in control cows and 19.2 (d 7) to 17.2 ± 1.1 kg/d (d 9) in bST-treated cows. The nighttime reduction in T_a (cyclic heat) prevented a further drop in feed intake during heat stress and allowed for partial recovery. Feed intake during cyclic heat ranged from 19.0 (d 10) to 20.4 ± 1.0 kg/d (d 13) in control cows, and 18.0 (d 10) to 19.3 ± 1.0 kg/d (d 13) in bST cows. A gradual increase ($P < 0.01$) in intake was noticed in both groups during the postheat period as T_a decreased. In contrast to the T_{core} and RR, feed intake was not affected ($P = 0.68$) in bST-treated (22.1 ± 0.8 kg/d) compared with control (22.4 ± 0.8 kg/d) cows during the 17-d treatment period. Similarly, there was no interaction between heat stress and bST treatment ($P = 0.39$).

Milk Production

Milk production was different ($P = 0.05$) between groups during the pretreatment period (i.e., bST: 28.1 ± 0.6 kg/d; control: 30.2 ± 0.6 kg/d; Figure 3B). For this reason, pretreatment values (i.e., d -3 to -1) were averaged for each animal and subtracted from each animal's daily value during treatment to determine the change from baseline. Heat stress reduced ($P < 0.01$) milk production in control cows during the constant and cyclic heat periods compared with the preheat period. The first indication of a significant reduction ($P < 0.01$) in milk production from the preheat treatment (d 3, last day of the preheat treatment period) occurred on d 8 and remained in place until the end of the 17-d treatment. In bST-treated cows, the reduction ($P < 0.05$) in milk production occurred on d 7 compared with the preheat treatment period (d 3) and remained so until d 16. The average milk production in control cows changed from -1.9 (d 7) to -6.7 ± 1.0 kg/d (d 9), whereas

in bST-treated cows it moved from 1.2 (d 7) to -3.2 ± 1.0 kg/d (d 9). A reduction in nighttime Ta during cyclic heat stabilized the drop from -6.9 (d 10) to -6.6 ± 0.9 kg/d (d 13) in control cows, and from -2.7 ± 0.9 (d 10) to -2.4 ± 0.9 kg/d (d 13) in bST-treated cows. A gradual reduction in Ta during the postheat treatment period increased milk production in both groups (compared with d 13), with greater improvement in bST-treated (4.0 kg) vs. control cows (2.6 kg). By the end of the treatment (d 17), milk production in the control cows was still lower ($P < 0.01$) than the values on d 1, whereas bST-treated cows had a higher level of milk production ($P = 0.05$) than on d 1. Treatment with bST stabilized ($P < 0.01$) milk production during heat stress (-0.2 kg), whereas control cows lost production (-3.9 kg) during the 17-d treatment. Milk production was greater in bST-treated cows compared with control cows at all Ta (Tn, cyclic, and constant heat stress; $P < 0.02$) except on d 8. The average difference in milk yield between control and bST-treated cows was 3.7 kg. There were no phase and bST treatment interaction effects ($P = 0.17$) on milk production during the treatment. Control and bST-treated cows exhibited greater ($P < 0.01$) milk yield at the evening milking compared with the morning milking during the 17-d treatment period (Figure 4). Milk production at the a.m. reading was -2.4 ± 0.3 and 1.0 ± 0.3 kg/d ($P < 0.01$) for control and bST cows, respectively, during the 17-d treatment period ($P < 0.01$). Similarly, milk production during the p.m. period was -1.5 ± 0.3 and 0.8 ± 0.3 kg/d ($P < 0.01$) for control and bST-treated cows, respectively (Figure 4).

The average milk-to-feed ratio (MFR) was calculated by analyzing the ratio of milk produced per kilogram of DM intake (Figure 5). The MFR was not different ($P > 0.05$) for bST-treated and control cows during the pretreatment, preheat, transition, and postheat periods (except on d 17; $P = 0.02$). Heat stress increased ($P < 0.01$) the MFR in bST-treated and control cows during constant and cyclic heat stress compared with the preheat treatment period. The first indication of a significant increase ($P < 0.01$) in the MFR from the preheat treatment period (d 3) was on d 7 in bST-treated cows, and remained so until d 12. In contrast, control cows increased the MFR above the preheat treatment level ($P < 0.01$) only on d 8. The MFR was reduced ($P < 0.01$) in both groups during the postheat period. Cows treated with bST exhibited a greater MFR (1.37 ± 0.05 kg/kg; $P = 0.01$) than control cows (1.23 ± 0.05 kg/kg) during the 17-d treatment period. A significant interaction ($P = 0.03$) existed between the heat phase and bST treatment.

Blood Measures

The plasma glucose level did not change ($P = 0.90$) between control and bST-treated cows during the pre-

heat period (Table 2). However, heat stress reduced ($P < 0.01$) the glucose level in both groups below the Tn level. The reduction was more evident during the early and late cyclic heat-stress periods in both groups, compared with the Tn and constant heat periods. The plasma glucose level was lower ($P < 0.05$) in both groups during the postheat period compared with the preheat period. However, the glucose level in bST-treated cows was not different from control cows ($P = 0.89$) during the 17-d period. An interaction between the heat phase and bST treatment was absent for the plasma glucose level ($P = 0.81$).

Serum GH was similar ($P = 0.64$) between control and bST-treated cows during the pretreatment period (Figure 6A). An increase ($P < 0.01$) in serum GH was observed in bST-treated dairy cows compared with control cows during the preheat treatment period. Heat stress increased serum GH above the Tn level ($P < 0.01$) during cyclic heat; however, constant heat reduced ($P < 0.01$) serum GH in bST-treated cows compared with the preheat and cyclic heat periods. Heat stress did not change ($P > 0.05$) the serum GH of control cows from the pretreatment level. Serum GH was not different ($P = 0.82$) between the pre- and post-heat-stress treatment periods. As expected, serum GH was greater in bST-treated cows compared with control cows ($P < 0.01$) during the 17-d treatment. In addition, there was a significant interaction ($P = 0.05$) between heat phase and bST treatment during the entire treatment period.

Plasma IGF-I did not change ($P = 0.50$) between control and bST-treated cows during the pretreatment period (Figure 6B). The bST treatment increased ($P < 0.01$) plasma IGF-I above that of control cows during most of the treatment period, with no interaction effect between heat phase and bST treatment ($P = 0.10$) on the plasma IGF-I level.

No difference in serum NEFA was observed in control and bST-treated cows during the pretreatment period (Figure 7A). Serum NEFA were greater ($P < 0.01$) in bST-treated cows compared with control cows during the preheat treatment period. Heat stress increased ($P < 0.01$) NEFA in bST-treated cows above the Tn level, with no significant change ($P > 0.05$) in control cows compared with pretreatment values. Higher serum NEFA levels were observed during early and late cyclic heat stress compared with the Tn period. During the postheat treatment, NEFA level was lower ($P < 0.01$) in both control and bST-treated cows compared with the cyclic heat period. Serum NEFA levels were increased by bST treatment above those of control cows ($P < 0.01$) during the 17-d treatment, suggesting a negative energy balance. Similarly, a significant interaction effect was observed between heat phase and bST treatment on NEFA.

Table 2. Mean values of serum biochemistry profile from cows treated with saline or bST during thermoneutral, constant, and cyclic heat-stress conditions¹

Variable	Pretreatment, phase 1	Preheat treatment, phase 2	Constant heat, phase 3	Early cyclic heat, phase 4	Late cyclic heat, phase 5	Postheat, phase 6	SEM
Glucose ²	78.9	83.0	77.7	69.5	59.3	75.2	1.8
Insulin ³	15.6	17.8	18.8	16.6	13.1	17.8	1.3
Prolactin ⁴	11.6	10.8	19.1	19.9	20.9	14.6	1.2

¹No bST treatment effect and no interaction effect between bST treatment and phase. The phase effect was measured using all 6 phases in the model. The phase effect was significant at the $P \leq 0.01$ level.

²A difference was found between phases 1 and 4, 1 and 5, 2 and 3, 2 and 4, 2 and 5, 2 and 6, 3 and 4, 3 and 5, 4 and 5, 4 and 6, and 5 and 6.

³A difference was found between phases 2 and 5, 3 and 5, and 5 and 6.

⁴A difference was found between phases 1 and 4, 1 and 5, 2 and 3, 2 and 4, 2 and 5, 2 and 6, 3 and 6, 4 and 6, and 5 and 6.

Plasma urea nitrogen did not change in control and bST-treated cows during the pretreatment period. During the preheat treatment, PUN decreased ($P < 0.01$) in bST-treated cows compared with control cows (Figure 7B). An increase ($P < 0.01$) in PUN above the Tn level was observed during constant heat and in the initial stage of cyclic heat. In contrast, a reduction ($P < 0.01$) occurred in control and bST-treated PUN during late cyclic heat compared with early cyclic heat. Likewise, bST treatment decreased ($P < 0.01$) PUN below the control level during the entire 17-d treatment. Plasma urea nitrogen was lowered ($P < 0.01$) during the postheat period compared with the preheat treatment period. An interaction between heat phase and bST treatment was not seen for PUN ($P = 0.26$).

Serum insulin did not change ($P > 0.05$) between bST-treated and control cows during the pretreatment and preheat periods (Table 2). Heat stress increased ($P = 0.04$) serum insulin during the constant heat period compared with the Tn period. Nevertheless, the nighttime reduction in Ta reduced ($P < 0.05$) this level, especially during the later part of the cyclic heat period, compared with the pretreatment, preheat, and constant heat periods. A significant increase ($P = 0.03$) in serum insulin occurred during the postheat period compared with the cyclic heat-stress period. Serum insulin levels did not differ ($P = 0.96$) between the preheat and postheat periods. Likewise, the serum insulin level in bST-treated cows was not different from that of control cows ($P = 0.17$) during the 17-d period (Table 2). There was no interactive effect of heat phase and bST treatment on serum insulin ($P = 0.64$).

Serum prolactin did not change between control and bST-treated cows during the pretreatment ($P = 0.41$) and preheat periods ($P = 0.65$; Table 2). Serum prolactin in both control and bST-treated cows was above the Tn level during all stages of heat stress ($P < 0.01$), with a greater increase occurring during the later part of the

cyclic heat period (Table 2). Likewise, this level was greater ($P = 0.02$) during the postheat period compared with the preheat treatment period. Bovine somatotropin treatment did not ($P = 0.63$) affect the serum prolactin level in bST-treated cows compared with control cows, suggesting an alternate pathway for the bST-induced increase in milk production. There was no interaction between phase and bST treatment.

DISCUSSION

The present study determined the simultaneous impact of heat stress and bST treatment on productivity and evaluated the beneficial effect of cycling Ta. Few environmentally controlled studies have simulated the natural hot summer day, when Ta drops by 2 to 3°C.

T_{core} and RR

Exposure of dairy cows to heat stress in the present study resulted in a rapid increase in both RR and T_{core} . Moreover, the increase in RR preceded the onset of hyperthermia by 1 d (d 5 vs. d 6) for both the bST-treated and control groups (Figure 2). Likewise, the RR remained elevated for the entire heat period (Figure 2B), with a decline occurring only by the end of the cyclic heat period. Differences in the RR and T_{core} responses suggest that RR is more sensitive than T_{core} to heat stress, supporting the view by Brown-Brandl et al. (2005) that RR may be the superior indicator.

In the present study, bST treatment during heat stress elevated the T_{core} , with only a slight increase in RR to dissipate excess heat (Figure 2A and 2B). The highest T_{core} and RR were recorded in the bST groups during constant heat. Therefore, the elevated T_{core} in bST-treated cows during constant heat could be partially due to a reduced ability to dissipate the higher metabolic heat associated with increased milk production. Although T_{core} decreased in both groups of cows

Figure 7. Average (A) NEFA and (B) plasma urea nitrogen (PUN) levels of bST-treated and control cows during treatment. Error bars on the mean values indicate $\pm$ SE. The asterisk shows a difference ($P < 0.05$) between bST-treated and control cows for the day. Tn = thermoneutrality.

during cyclic heat compared with constant heat, the bST-treated group still exhibited higher T_{core} compared with control cows owing to a potentially greater metabolic heat production (Figure 3B). This would suggest that a bST-induced elevation in T_{core} would be seen under either scenario in the field. Earlier studies reported higher milk production and body heat in bST-treated dairy cattle exposed to T_n (15 to 22°C), hot (25 to 35°C), or cold (−5 to 5°C) cyclic thermal environments (Johnson et al., 1991; Manalu et al., 1991). In contrast, Cole and Hansen (1993) noted increases in rectal temperature and RR in dairy cows treated with bST under heat conditions (increased solar radiation), but no difference during T_n periods. These results agree with the current study showing no difference between treatment groups once there is a sufficient animal-to-ambient thermal gradient for heat dissipation.

Feed Intake

Dairy cows treated with bST in the present study did not show differences in DMI; however, they had greater energy output relative to energy intake compared with control cows. Tyrrell et al. (1988) demonstrated that energy expenditure for maintenance and the partial efficiency of milk synthesis were unchanged in bST-treated cows. Therefore, they concluded that productive efficiency gains with bST occurred because a large portion of the nutrients consumed were diverted for milk synthesis. Consistent with the results in the present study, Eppard et al. (1996) did not observe a difference in DMI in lactating dairy cows treated with bST compared with control cows under T_n conditions. Contrary to our findings, West et al. (1991) noted a reduction in the DMI of bST-treated dairy cattle exposed to heat stress. Again, this was not the case in the present study, which showed no effect of bST on feed intake for heat-stressed dairy cows, with both groups experiencing a 30% reduction in feed intake. Likewise, there was no parallel relationship between the effects of bST on feed intake and milk production. The difference between the results of this study and those reported by others might be due to the duration of treatment or the magnitude of heat stress. The relatively short exposure periods in the present study might have limited the time for a response to occur in treated animals for some of the physiological parameters such as DMI.

Milk Production

Heat stress alone in the present study did not produce a decrease in milk production until a THI of 76 was reached, and there was a significant increase in T_{core} . In contrast, Johnson et al. (1961) reported that milk

production in dairy cattle started decreasing as the mean daily THI exceeded 71. They reported that higher producing animals declined milk production more rapidly compared with lower producing dairy cows as the THI increased from 71 to 91, suggesting greater susceptibility of high-milk-producing animals to heat stress. This might explain the difference in critical THI values for the 2 studies. It is likely that the direct stimulus for a reduction in milk production is the T_{core} and not the THI. Therefore, a determinant of the thermal status of the animal may be a more valuable indicator of heat stress and a reduction in milk production.

In the present study, the beneficial effects of a nighttime reduction in T_a was evident even in total milk production, because the drop in daily milk production was stabilized during cycled heat. Igono et al. (1992) noted that milk production in lactating cows is enhanced during heat stress when the night T_a is reduced to 21°C for 3 to 6 h. In the current study, a 3°C reduction in T_a to only 25.5°C effectively stabilized the drop in milk production at a level below that at T_n (Figure 3B). This effect was likely due to the associated reduction in heat load, as indicated by the decrease in T_{core} under the cycling condition (Figure 2A) as well as to stabilization of the drop in intake.

Milk production in the present study was greater in bST-treated cows within the T_n environment, with no increase in T_{core} . However, Tyrrell et al. (1988) reported a greater release of heat energy in bST-treated dairy cows compared with control cows at T_n , which might lead to an increase in body temperature. In the present study, heat stress still reduced milk production in bST-treated cows. This reduction was parallel to that of control animals but occurred after the initial bST-induced increase. In both groups, this was possibly due to a reduced DMI and accumulated heat load. Schams et al. (1991) reported that induced galactopoiesis in bST-injected cattle was not due to feeding more concentrates but to other mechanisms. These can include the metabolism of nutrients, intracellular signal transduction systems, increased cellular rates of milk synthesis, and enhanced maintenance of secretory cells (Bauman et al., 1999). In the present study, the increased milk production in bST-injected cows compared with control cows during both T_n and moderate heat conditions were in accordance with previous work (Ominski et al., 2002).

MFR

The MFR of bST-treated cows in the present study increased by 10.9% compared with control cows, especially during cyclic heat. McCutcheon and Bauman (1986) observed a 16% increase in the MFR in lactating dairy cows injected at T_n with 25 IU of GH. Likewise,

Johnson et al. (1991) observed an increased efficiency of feed energy utilization to milk energy secretion in bST-treated cows during exposure to both Tn or heat conditions, without a change in BW or temperature. In the present study, the small increase in MFR that occurred in control cows during heat stress was a transient response attributable to a more rapid reduction in feed intake than milk production. This was expected because system input (i.e., feed intake) should precede output (i.e., milk production). Cows treated with bST during heat stress displayed a significantly higher MFR than control cows (Figure 5). This was likely due to a bST-induced diversion of a greater proportion of consumed nutrients to the mammary gland for milk synthesis. Bauman et al. (1999) connected much of the bST-related shift in nutrient utilization to insulin activity. Circulatory levels of insulin increase during feed intake, and bST treatment alters the insulin sensitivity of adipose tissue so that more nutrients are diverted from adipose tissue to the mammary glands. With reduced nutrient intake, such as during heat stress in the present study, there is greater mobilization of energy because of an increased lipolytic response to catecholamines (Bauman et al., 1999). Finally, they reported that bST-treated cows with reduced energy intake may exhibit a small increase in milk production without any evidence of a metabolic effect. Any combination of these mechanisms might have resulted in the increased MFR level noted in the present study.

Morning vs. Evening Milk

In the present study, bST-treated cows exhibited greater milk production compared with control cows during both the a.m. and p.m. milking periods (Figure 4). However, milk production was greater in bST-treated dairy cows during the p.m. compared with the a.m. milkings. This increase in milk production was greater during Tn and cyclic heat compared with constant heat. Throughout the study period, the lowest T_{core} was observed in both control and bST-treated cows at 0700 h, although in both groups the lowest T_{core} values differed during different phases, depending on the Ta. Even during cyclic heat, when the Ta decreased by 3°C, the lowest T_{core} in both groups occurred only at 0700 h, which was after the a.m. milking (0500 h). Therefore, an improvement in bST-treated dairy cow milk production was observed only during the p.m. milking, because the cows required a lag period to dissipate their excess body heat. Furthermore, the time period between the first injection of bST (0930 h) and the p.m. milking (1700 h) was shorter compared with the second bST injection (2000 h) and the a.m. milking (0500 h). McCutcheon and Bauman (1986) reported that

the lactational response to bST depends on the interval between injections, with the bST concentration being more important than the pattern of elevation. The diminished bST response at the a.m. vs. p.m. milkings in the present study might be due to differences in the blood level of bST.

Blood Measures

Concentrations of plasma glucose were not different in bST-treated and control cows prior to heat exposure (Table 2). However, heat stress reduced the plasma glucose levels in both groups, followed by partial recovery during the postheat period. Itoh et al. (1998) observed a similar reduction in plasma glucose levels in heat-stressed (28°C) dairy cows. The reduction in plasma glucose during heat stress could be attributed to a reduction in feed intake. The lack of a bST treatment effect on the plasma glucose level was reported previously (Bauman et al., 1988).

The serum insulin of cows in the present study did not differ between treatment groups at any time (Table 2). The insulin concentration deviated from the Tn level only near the end of heat stress, when it was reduced. Itoh et al. (1998) observed an increase in serum insulin in dairy cows exposed to constant heat stress (28°C and 60% relative humidity) for 13 d. Sartin et al. (1985) noted lower insulin secretion rates in lactating cows at Tn. The partial increase in milk production during the cyclic heat and postheat periods could be due to lower serum insulin levels in both groups, which might allow essential nutrients to be directed toward the mammary gland. In the present study, no significant difference was observed in serum insulin levels between the bST-treated and control groups. Similar results were observed by Schams et al. (1991).

In the present study, heat stress significantly increased prolactin levels above the Tn level (Table 2). These observations are in accordance with previous studies (Giesecke, 1985). The beneficial role of prolactin in galactopoiesis is well documented, although it is less important for maintenance of lactation in ruminants compared with nonruminants (Yang et al., 2005). Prolactin levels did not differ between control and bST-treated dairy cows during treatment, suggesting a different mechanism of action for prolactin-induced lactation from that of bST. These observations agree with others (Miller et al., 1999) who reported that prolactin and bST interact, independently of the IGF-I pathway, to stimulate galactopoiesis.

Throughout the study, a significant increase in the blood circulatory concentrations of IGF-I was observed in bST-injected cows compared with control cows (Figure 6A). Others (West et al., 1991) observed a similar

increase in blood IGF-I in bST-treated cows that accompanied increased milk production. Infusion of the mammary gland with IGF-I, but not bST, stimulates milk production (Bauman et al., 1999), providing evidence that the galactopoietic effect of bST on mammary gland function is through IGF-I. It is possible that IGF-I has a direct effect on increased milk production by increasing either the alveolar cell number or activity (Schams et al., 1991). It is worth noting that despite large decreases in feed intake and energy balance, the GH-IGF-I axis did not dissociate. In fact, the IGF-I concentration increased during thermal stress. This may be associated with the increased insulin also detected in this study.

In the present study, exposure to heat stress alone increased the bST level after approximately 1 wk (Figure 6A). Johnson and Vanjonack (1976) reported that short- and long-term heat stress would initially increase GH, with a decrease after prolonged exposure (Manalu et al., 1991). A steady increase in the levels of blood GH was observed in bST-treated dairy cows compared with control cows in the present study. These results are in accordance with the results of others (Miller et al., 1999).

Exposure to heat stress in the present study increased serum NEFA levels only in bST-treated cows (Figure 7A). This is in contrast to the results of Itoh et al. (1998), who observed an increase in serum NEFA above the T_n level in dairy cattle exposed to heat stress (28°C) alone. Our results agree with other reports (Bauman et al., 1988; Schams et al., 1991). The elevated NEFA concentrations observed may indicate mobilization of body fat stores to provide energy for increased milk production. Bauman et al. (1999) reported that lipolysis would increase in bST-injected cows in negative energy balance. Mammary and peripheral tissues use NEFA as an energy source as an alternative to glucose (Tyrrell et al., 1988). Serum NEFA levels are negatively correlated with animal energy balance (Bauman et al., 1988); therefore, higher NEFA in bST-treated dairy cows indicate that they probably had a more negative energy balance. Concentrations of serum NEFA increased both in response to restricted feed consumption as well as in response to bST treatment (Capuco et al., 2001). Therefore, the increase in serum NEFA levels in bST-treated dairy cows could be due to reduced feed intake in response to heat stress and bST treatment. The shift in NEFA was more pronounced in bST-injected cows during heat-stress conditions. The animals could be more sensitive to the combined effects of bST and heat stress in the adipose tissue response to homeostatic signals that affect lipid metabolism.

Heat exposure in the present study increased PUN levels during the constant heat-stress period, with a tendency toward reduction during the cyclic heat period

(Figure 7B). Srikanthakumar and Johnson (2004) observed a similar increase in PUN in heat-stressed dairy cows. There was a significant decrease in blood PUN in bST-treated cows compared with control cows. This is similar to that seen in lactating cows (Bauman et al., 1989). Lowered PUN might indicate reduced AA oxidation by the mammary gland or increased milk protein synthesis. At a similar nitrogen intake, the PUN level decreases in bST-treated dairy cows, suggesting improved nitrogen utilization with higher milk protein production and lowered AA catabolism (Oldenbroek et al., 1993).

CONCLUSIONS

Daily treatment with recombinant bST increased milk production in dairy cows above control levels during both T_n and heat-stress conditions, with no effect on feed intake. Treatment with recombinant bST did not change the T_{core} at T_n but elevated it during heat stress. These results suggest that bST increases milk production in dairy cows during heat stress, but potentially increases susceptibility to heat stress. The proven benefit and efficacy of bST during hot climatic conditions should be coupled with procedures to reduce the magnitude of heat stress. The nighttime lowering of air temperature increases the rate of heat loss, thereby improving performance under this condition.

REFERENCES

- Armstrong, D. V. 1994. Heat stress interaction with shade and cooling. *J. Dairy Sci.* 77:2044–2050.
- Bauman, D. E., R. W. Everett, W. H. Weiland, and R. J. Collier. 1999. Production responses to bovine somatotropin in northeast dairy herds. *J. Dairy Sci.* 82:2564–2573.
- Bauman, D. E., D. L. Hard, B. A. Croaker, M. S. Patridge, K. Garrick, L. D. Sandles, H. N. Erb, S. E. Franson, G. P. Hartnell, and R. L. Hintz. 1989. Long-term evaluation of a prolonged-release formulation of N-methionyl bovine somatotropin in lactating dairy cows. *J. Dairy Sci.* 72:642–647.
- Bauman, D. E., C. J. Peel, W. D. Steinhour, P. J. Reynolds, H. F. Tyrrell, A. C. Brown, and G. L. Haaland. 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.
- Brown-Brandl, T. M., R. A. Eigenberg, G. L. Hahn, J. A. Nienaber, T. L. Mader, D. E. Spiers, and A. M. Parkhurst. 2005. Analyses of thermoregulatory responses of feeder cattle exposed to simulated heat waves. *Int. J. Biometeorol.* 49:285–296.
- Byatt, J. C., P. J. Eppard, L. Munyakaz, R. H. Sorbet, J. J. Veenhuizen, D. F. Curran, and R. J. Collier. 1992. Stimulation of milk yield and feed intake by bovine placental lactogen in the dairy cow. *J. Dairy Sci.* 75:1216–1223.
- Byatt, J. C., P. J. Eppard, J. J. Veenhuizen, T. L. Curran, D. F. Curran, M. F. McGrath, and R. J. Collier. 1994. Stimulation of mammary and lactogenesis by recombinant bovine placental lactogen in steroid primed dairy heifers. *J. Endocrinol.* 140:33–43.
- Capuco, A. V., D. L. Wood, T. H. Elsasser, S. Kahl, R. A. Erdman, C. P. Van Tassell, A. Lefcourt, and L. S. Piperova. 2001. Effect of somatotropin on thyroid hormones and cytokines in lactating dairy cows during ad libitum and restricted feed intake. *J. Dairy Sci.* 84:2430–2439.

- Cole, J. A., and P. J. Hansen. 1993. Effects of administration of recombinant bovine somatotropin on the responses of lactating and nonlactating cows to heat stress. *J. Am. Vet. Med. Assoc.* 203:113–117.
- Eppard, P. J., J. J. Veenhuizen, W. J. Cole, P. G. Comens-Keller, G. F. Hartnell, R. L. Hintz, L. Munyakazi, P. K. Olsson, R. H. Sorbet, T. C. White, C. A. Baile, R. J. Collier, J. P. Goff, and R. L. Horst. 1996. Effect of bovine somatotropin administered to periparturient dairy cows on the incidence of metabolic disease. *J. Dairy Sci.* 79:2170–2181.
- Etherton, T. D., and D. E. Bauman. 1998. Biology of somatotropin in growth and lactation of domestic animals. *Physiol. Rev.* 78:745–761.
- Giesecke, W. H. 1985. The effect of stress on udder health of dairy cows. *Onderstepoort J. Vet. Res.* 52:175–193.
- Igono, M. O., G. Bjotvedt, and H. T. Sanford-Crane. 1992. Environmental profile and critical temperature effects on milk production of Holstein cows in desert climate. *Int. J. Biometeorol.* 36:77–85.
- Itoh, F., Y. Obara, M. T. Rose, H. Fuse, and H. Hashimoto. 1998. Insulin and glucagons secretion in lactating cows during heat exposure. *J. Anim. Sci.* 76:2182–2189.
- Johnson, H. D., R. Li, W. Manalu, K. J. Spencer-Johnson, B. A. Becker, R. J. Collier, and C. A. Baile. 1991. Effects of somatotropin on milk yield and physiological responses during summer farm and hot laboratory conditions. *J. Dairy Sci.* 74:1250–1255.
- Johnson, H. D., A. C. Ragsdale, E. L. Berry, and M. D. Shanklin. 1961. Effect of various temperature-humidity combinations on milk production of Holstein cattle. *Res. Bull. No. 791. Missouri Agric. Exp. Stn., Columbia, MO.*
- Johnson, H. D., and W. J. Vanjonack. 1976. Effects of environmental and other stressors on blood hormone patterns in lactating animals. *J. Dairy Sci.* 59:1603–1617.
- Manalu, W., H. D. Johnson, R. Li, B. A. Becker, and R. J. Collier. 1991. Assessment of thermal status of somatotropin-injected lactating Holstein cows maintained under controlled-laboratory thermoneutral, hot and cold environments. *J. Nutr.* 121:2006–2015.
- McCutcheon, S. N., and D. E. Bauman. 1986. Effects of pattern of administration of bovine growth hormone on lactational performance of dairy cows. *J. Dairy Sci.* 69:38–43.
- Miller, A. R., E. P. Stanisiewski, R. A. Erdman, L. W. Douglass, and G. E. Dahl. 1999. Effects of long daily photoperiod and bovine somatotropin (Trobect) on milk yield in cows. *J. Dairy Sci.* 82:1716–1722.
- Oldenbroek, J. K., G. J. Garssen, L. J. Jonker, and J. I. D. Wilkinson. 1993. Effects of treatment of dairy cows with recombinant bovine somatotropin over three of four lactations. *J. Dairy Sci.* 76:453–467.
- Ominski, K. H., A. D. Kennedy, K. M. Wittenberg, and S. A. Moshtaghi Nia. 2002. Physiological and production responses to feeding schedule in lactating dairy cows exposed to short-term, moderate heat stress. *J. Dairy Sci.* 85:730–737.
- Sartin, J. L., K. A. Cummins, R. J. Kemppainen, D. N. Marple, C. H. Rahe, and J. C. Williams. 1985. Glucagon, insulin and growth hormone responses to glucose infusion in lactating dairy cows. *Am. J. Physiol.* 248:108–114.
- Schams, D., F. Graf, J. Meyer, B. Graule, M. Mauthner, and C. Wollny. 1991. Changes in hormones, metabolites, and milk after treatment with sometribove (recombinant methionyl bST) in Deutsches Fleckvieh and German black and white cows. *J. Anim. Sci.* 69:1583–1592.
- Srikandakumar, A., and E. H. Johnson. 2004. Effect of heat stress on milk production, rectal temperature, respiratory rate and blood chemistry in Holstein, Jersey and Australian milking zebu cows. *Trop. Anim. Health Prod.* 36:685–692.
- Thom, E. C. 1959. The discomfort index. *Weatherwise* 12:57–59.
- Tyrrell, H. F., A. C. G. Brown, P. J. Reynolds, G. L. Haaland, D. E. Bauman, C. J. Peel, and W. D. Steinhour. 1988. Effect of ovine somatotropin on metabolism of lactating dairy cows: Energy and nitrogen utilization as determined by respiration calorimetry. *J. Nutr.* 118:1054–1061.
- Vicini, J. L., W. S. Cohick, J. H. Clark, S. N. McCutcheon, and D. E. Bauman. 1988. Effects of feed intake and sodium bicarbonate on milk production and concentrations of hormones and metabolites in plasma of cows. *J. Dairy Sci.* 71:1232–1238.
- West, J. W., B. G. Mullinix, and T. G. Sandifer. 1991. Effects of bovine somatotropin on physiologic responses of lactating Holstein and Jersey cows during hot, humid weather. *J. Dairy Sci.* 74:840–851.
- Yang, J., B. Zhao, V. E. Baracos, and J. J. Kennelly. 2005. Effects of bovine somatotropin on β -casein mRNA levels in mammary tissue of lactating cows. *J. Dairy Sci.* 88:2806–2812.