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NATURALLY SIMULATED HEAT STRESS IN LACTATING DAIRY COWS : HEAT PRODUCTION, HEAT LOSSES, AND THERMOREGULATORY FUNCTIONS

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ABSTRACT

An experiment was designed to study the effects of naturally simulated cyclic laboratory heat on heat production, heat loss and thermoregulatory functions in lactating dairy cows. Six multiparous lactating Holstein cows, 90 to 150 d postpartum with a milk production level of 25 kg/d or greater, were used in the experiment. The cows were assigned into two groups of environmental conditions (thermoneutral [TN] and hot). Each group of experimental cows was exposed to respective environmental conditions for 10 d with single reversal design. Prior to the experiment, the cows were adjusted for 7 d to cyclic hot (22 to 35 °C and 30 to 50 % relative humidity). In the period 1, group 1 was exposed to thermoneutral conditions (18 to 22 °C and 50 to 65 % relative humidity) for 10 d. Both groups were adjusted to cyclic hot (22 to 35 °C and 30 to 50 % relative humidity) for 4 d before the period 2. In the period 2, the environmental conditions were switched for each group. The results of the experiment showed that heat stress increased rectal temperature, skin temperature and respiratory rates ($P<0.01$). The increased body temperature due to heat stress triggered elevation of vaporization heat losses through skin and respiratory tract ($P<0.01$) without a significant reduction in heat production. The depression of gross energy intake and milk energy output ($P< 0.01$) during heat stress did not result in parallel decrease in heat production. It was concluded that in the degree of heat stress used in this experiment, dairy cows still produced the same amount of heat as compared to TN to support more work required for thermoregulatory mechanism (maintenance energy expenditure). To reduce the effects of heat stress on lactating dairy cows in the tropics, it was suggested to devise an appropriate technology to increase dissipation of heat without exposing additional metabolic energy expenditure to dissipate heat from the body.

Keyword : Holstein cows, lactating, heat production, heat loss, thermoregulation.

It is known that dairy cow has a range of ambient temperature for optimum production called thermoneutral zone. Brody (1948) stated that when dairy cows maintained in the environment with ambient temperature above or below the thermoneutral zone, the mechanism of thermoregulation was triggered to regulate and maintain body temperature relatively constant.

It was well documented that in the case of heat stress, the animal body gained heat from the environment that caused increase in body temperature activated thermoregulatory mechanism such as increasing evaporative heat loss (Kibler and Brody, 1950; Thompson *et al.*, 1951), decreasing energy intake (Johnson, 1980) and metabolic rate (Kibler and Brody, 1949; Johnson, 1980), to restore body temperature back to normal. However, most of the heat stress studies reported so far used constant temperature and humidity during the whole experimental period. The results of such experiments are definitely different from the effects of natural heat stress which change over 24 h period (Kibler and Brody, 1956). The objectives of this current study were to measure thermoregulatory mechanism in the lactating heat-stressed cows maintained in the laboratory-controlled environment simulated similar to natural heat stress during the summer.

MATERIALS AND METHODS

Experimental Design and Protocol

Six multiparous lactating Holstein cows, 90 to 150 d postpartum with a milk production level of 25 kg/d or greater, were selected from the University of Missouri Holstein Farm (Columbia, Missouri, USA). The use of cows for this heat stress experiment was approved by the University of Missouri Animal Care Use Committee. In the Brody Animal Climatology Laboratory chambers, the cows were assigned into two groups of environmental conditions for 10 d with single reversal design. Prior to the experiment, the cows were adjusted for 7 d to cyclic hot (22 to 35 °C and 30 to 50 % relative humidity). In the period 1, group 1 was exposed to thermoneutral conditions (18 to 22 °C and 50 to 65 % relative humidity) and group 2 to cyclic hot (24 to 35 °C and 55 to 65 % relative humidity) for 10 d. Both groups were adjusted to cyclic hot (22 to 35 °C and 30 to 50 % relative humidity) for 4 d before the period 2. In the period 2, the environmental conditions were switched for each group.

The cyclic environmental conditions used simulated average outside summer condition using computer to control temperature, humidity, and air flows. The temperature was gradually increased from a minimum in the morning to reach a peak that was maintained between 11:00 and 16:00 hours and gradually decreased to reach the lowest point at midnight. The relative humidity was cycled opposite to the temperature, as normally occurs in a daily cyclic, gradually decreasing during the day to the lowest level between 11:00 to 16:00 hours, and then gradually increasing to the highest level at midnight. During the experiment, photoperiod in the chamber was

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maintained for 14 h (05:00 to 19:00 hours) using an automatic timer (Inter-matic Timers Inc., Chicago, Ill.).

Cow Management

In the climatic chambers, the cows were housed individually in stanchions provided with comfort rubber mats. Cows were milked at 05:00 and 17:00 hours, and were allowed free access to a complete diet. Water was freely available from individual water cups.

Measurements

Heat production was measured between 11:00 and 15:00 hours on days 1, 3, 6, 8, and 10, by open-circuit indirect calorimeter. This method was based on the O_2 consumption and CO_2 production with correction for CH_4 production. Skin vaporization (measured in the same days by using a capsule procedure) and respiratory vaporization (measured simultaneously with the indirect calorimeter) heat losses were measured by multiplying the amount of moisture vaporized through the skin and respiratory tract by 0.58 kcal/g (2.43 kJ/g) (heat vaporization of water). Cooling heat loss in this experiment refers to heat loss due to the cooling effect of water consumed as it passed through the body as milk water, water loss through the skin and respiratory tract, and urine and and fecal water. Cooling heat loss was calculated by multiplying the difference between drinking water and body temperatures with the total water intake. An increase of 1 ^{0}C in water temperature is equal to 4.184 kJ (1 kcal) of heat per kilogram water. The details of the measurements and calculations of the method used were explained elsewhere (Manalu *et al.*, 1991).

Milk energy was estimated by using 3.2 MJ/kg (749.6 kcal/kg) solids-corrected milk (Tyrrell and Reid, 1965). Rectal temperatures were measured at 08:00 and 16:00 hours daily with an electronic digital thermometer (Electronic Digital Thermometer, Fisher Scientific, Pittsburgh, PA). Respiratory rates were measured at 08:00 and 16:00 hours daily. Skin temperatures were measured between 11:00 and 15:00 hours on alternate days at three different locations with a copper-constantant thermometer (Digi-Sense Thermocouple Thermometer, Cole Palmer Instrument, Chicago, Ill.). Cows were weighed (Weigh-Tronix Scale, with digital readout) on day 1 and day 10 immediately after PM milking. Water intake was measured individually from in-line meters daily.

Statistical Analysis

Data were analyzed with General Linear Model procedure (SAS, 1985) using cow (period) as an error term. A statistical significant difference of the means was tested at $\alpha = 0.05$ and $\alpha = 0.01$.

RESULTS AND DISCUSSION

Body weight, heat production, gross energy intake and milk energy output data are presented in Table 1. Mean body weight, gross energy intake and milk energy output of the experimental cows are presented in this report since those parameters are critical in assessing heat production data. There was no difference in body weight of the cows maintained in the TN as compared to those in the hot environmental conditions. The intensity of heat stress applied in this experiment did not show a significant depression of heat production. However, gross energy intake in the heat-stressed cows decreased ($P<0.01$) as compared to those maintained in the TN conditions. Milk energy output also decreased ($P<0.05$) as gross energy intake decreased in the hot environmental conditions.

Table 1. Body weight, heat production, gross energy intake and milk energy output of the TN and heat-stressed cows.[1)]

Parameter	TN	Heat
Body weight, kg	560.5 ± 15.63	558.7 ± 14.97
Heat production, MJ/d	128.0 ± 3.20	126.6 ± 4.0
Gross energy intake, MJ/d	351.1 ± 20.50	271.9 ± 14.60**
Milk energy output, MJ/d	62.8 ± 3.61	53.0 ± 4.00*

1) Presented as means and SE of 6 cows. See text for the number of observations.
* Refers to significant difference at $\alpha = 0.05$
** Refers to significant difference at $\alpha = 0.01$

Heat loss data and thermoregulatory indices of the experimental cows are presented in Table 2. Heat stress conditions increased vaporization heat losses through skin and respiratory tract ($P < 0.01$). However, cooling heat loss did not show any significant difference even though numerically higher during heat stress. This result was probably due to the high variation in water intake, especially in the heat-stressed cows. Cows maintained in the hot environment had a higher rectal temperature, skin temperature and respiratory rates ($P < 0.01$).

Table 2. Heat loss parameters and thermoregulatory indices of the TN heat-stressed cows.[1)]

Parameters	TN	Heat
Skin Vaporization, MJ/d	31.3 ± 3.4	47.0 ± 3.1**
Respiratory vaporization, MJ/d	7.4 ± 0.5	10.5 ± 0.3**
Cooling heat loss, MJ/d	7.6 ± 1.1	9.9 ± 1.2
Rectal temperature, ^{0}C	38.3 ± 0.09	39.7 ± 0.24**
Skin temperature, ^{0}C	29.6 ± 0.1	35.2 ± 0.30**
Respiratory rates, *breaths/min.*	43.2 ± 4.3	86.3 ± 6.50**

1) Presented as means and SE of 6 cows. See text for the number of observations.
* Refers to significant difference at $\alpha = 0.05$
** Refers to significant difference at $\alpha = 0.01$

The results of this experiment showed that heat stress conditions caused increase in body temperature (skin and rectal temperature). The increased body temperature reflected an accumulation of heat in the body as a consequence of a greater heat gain than heat loss. Since homeothermic animals, including dairy cows, have a range of constant body temperature (Brody, 1948) any deviation from this normal range could trigger heat loss mechanism as an effort to bring the body temperature down to the normal level. In the heat stress conditions, the avenues of heat loss mechanism primarily via water vaporization through skin and respiratory tract (Brody, 1948). Dairy cows used both the skin and respiratory vaporization pathways to dissipate heat from the body. However, since dairy cows are panting species, they compensate their inability to sweat by panting, often protruding their tongues and blowing air rapidly over the moist surface, thereby accelerating vaporization rate (Brody, 1948).

Activities of sweat glands were regulated by the nervous system (Taneja, 1959) that was assumed to be controlled by the thermoregulatory center in the hypothalamus. The results of this experiment and other reports (Kibler *et al.*, 1949; Kibler and Brody, 1950; Thompson *et al.*, 1951; Johnson, 1980) showed that the increased heat gained from the environment triggered heat loss mechanism by increasing evaporation of water through skin and respiratory tract. However, given the increase in skin and respiratory vaporization, the magnitude of heat gained from the metabolic processes in the cells and from the environment were still higher than that could be dissipated by the heat loss mechanism, as were reflected in the higher rectal and skin temperatures.

Since dairy cows could not escape from the hot environmental conditions, the only way to lessen the heat load in the body was to reduce heat-generating processes in the body (Johnson, 1980). Oxidation of the nutrients in the cell for maintenance and production of energy expenditure is the primary source of heat in the body (Brody, 1948). To reduce this endogenous heat production, thermoregulatory center would activate satiety center in the hypothalamus to reduce feed intake (Collier, 1985; Johnson, 1986; 1987). This regulation was significantly reflected in the decreased gross energy intake in the heat-stressed cows (Ragsdale *et al.*, 1948; Johnson and Yeck, 1964; Johnson *et al.*, 1966).

Since the rate of milk synthesis and secretion depends on the availability of milk precursors (Collier, 1985), the depression in gross energy intake and the conditions of stressful environment could reduce the substrate flow into the mammary glands that resulted in decreased milk synthesis (Collier, 1985; Johnson, 1986). The reduction in the milk synthesis was reflected in the lower milk energy output in the heat-stressed cows as compared to the TN cows. However, the whole reduction of gross energy intake and milk synthesis did not cause a significant reduction of heat production as generally found in the heat-stressed animals (Kibler *et al.*, 1949; Johnson *et al.*, 1966; Kibler *et al.*, 1966; Johnson, 1980; 1986). The tendency of decreased heat production, however, was clear over the duration of the heat stress. Since thyroxin catalyzes the oxidation of nutrients to produce heat and ATP, reduction in feed intake automatically reduces heat production from fuel combustion. However, the increased work performed by the cows to dissipate heat would add an extra amount of heat production.

The cyclic environmental heat stress used in this study probably partly reduced the degree of heat stress as compared to the experiments conducted in the constant environment as was reported by Kibler and Brody (1956). It was assumed that the heat-stressed cows with lower gross energy intake and milk energy output still had the same level of heat production as those TN cows. Probably, it was partly due to the increased work performed to dissipate heat from the body.

The increased respiratory rates clearly involved a higher intensity of work performed by the respiratory muscle. This extra work could increase heat generated in the cells that ended up with no reduction in the heat production even though gross energy intake and milk energy output were reduced. The decreased milk energy output during the heat stress conditions could be attributed to the decreased gross energy intake (Johnson and Yeck, 1964; Johnson *et al.*, 1966; Kibler *et al.*, 1966) and to the increased energy expenditure for maintenance. The whole effects of heat stress on the lactating dairy cows were the increased energy expenditure for non productive processes (thermoregulatory mechanisms). As a result, the efficiency of energy utilization for milk synthesis became lower.

CONCLUSIONS

As a conclusion, heat-stressed cows consumed less energy, performed more thermoregulatory works to regulate body temperature with no change in heat production. Finally, heat-stressed cows produced less milk as a combinatorial result of lower energy intake and increased energy expenditure for maintenance of body temperature. To increase milk production in the tropics it is suggested to devise an appropriate technology to increase heat loss from the body without increasing energy expenditure for the maintenance work. As a feasible choice, using evaporative cooling mechanism with water sprays could increase heat loss without imposing more work performed by the respiratory muscle.

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