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Effects of cysteamine compound on milk production and hormonal responses of lactating cow during heat stress*

Z. Shen[1] and R. Zhang

Laboratory of Animal Physiology and Biochemistry, Nanjing Agricultural University
Nanjing 210095, China

ABSTRACT

In heat-stressed cow received cysteamine compound (Lactonin) the rectal temperature decreased ($P<0.05$), milk yield, fat-corrected milk, milk fat and feed conversion rate (FCR) increased ($P<0.05$). These changes were companied with trendy ($0.05<P<0.15$) of higher milk protein and lower somatic cell count. Additionally, plasma insulin enhanced ($P<0.01$) significantly in Lactonin herd ($n=49$), whereas T_3 and T_4 tended ($0.05<P<0.15$) to decline. The data show that Lactonin helps heat-stressed cow to maintain a more normal metabolism in hot summer, this positive effect of Lactonin on cow performance was associated with Lactonin-dependent alteration of plasma insulin, T_3 and T_4.

KEY WORDS: cysteamine compound, heat stress, milk production, thyroid hormone, insulin, cow

INTRODUCTION

In hot summer lactating cow reduced milk yield and feed intake and caused hormones changes in an attempt to attain homeorthermy. Estimated milk yield reduction was 0.2 kg per unit increase in temperature-humidity index (THI, Ravagnolo and Misztal, 2000), and milk yield and TDN intake declined by 1.8 and 1.4 kg for each 0.55°C increase in rectal temperature (Johnson et al., 1963). Bovine somatotropin (bST) improved milk yield and 3.5% FCM production in cow during the summer, and changed plasma hormone status (Igono et al., 1992). However, the application of bST to heat-stressed cow is limited due to consumers demand for feed safety. Lactonin is a compound of cysteamine (CS), which is

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[1] Corresponding author: e-mail: zmshen@njau.edu.cn

a component of coenzyme A. in vivo CS is oxidized to taurine. Therefore it is an endogenous substance. CS neutralizes somatostatine, consequently increases levels of growth hormone and blood glucose. The purpose of this study was to investigate the effects of cysteamine compound (Lactonin) on milk production of heat-stressed dairy cow in hot summer.

MATERIAL AND METHODS

96 Holstein cows, with average lactating day of 210 d, were assigned to 4 groups (G1, G2, G3 and G4) on the basis of their daily milk yield (M) prior to the experiment: G1<24, 24<G2<28, 28<G3<32 and G4>32 kg/d. In each group the cows were divided randomly into two subgroups: 1. Lactonin treatment (LT, n=49) and, 2. Control (n=47).

Lactonin was mixed with maize meal and administered to cow of LT at dose of 3000 U/d·head through the experimental period. In Control the equative maize meal was given without supplement of Lactonin. Experiment was carried out in Shanghai Bright Dairy Farm for 50 d (since July 11 through August 29, 2002), during this period the temperature and humidity in the cowshed was measured tree times per day, milk yield and milk composition was measured every second day and 10 days, respectively. Blood was collected through tail vein at d 10 and d 50 of the experiment.

THI = 0.72 (T_d+T_w) + 40.6 THI: temperature-humidity index; T_d: dry bulb temperature; $T_{w:}$ wet bulb temperature.

3.5% FCM = (0.4324 × milk yield) + (16.218 × milk fat)

The data was expressed as mean ± SEM. Differences were analysed by *F* test (Software SPSS) with P<0.05 considered as significant.

RESULTS

Lactonin improved cow performances. In LT of G1 the rectal temperature decreased (P<0.05), but milk yield, FCM, and milk protein increased by 21.7, 24.6 and 25.6%, respectively (P<0.05), feed conversion rate enhanced by 20.5% (P<0.05). Somatic cell count tended to decline (0.05<P<0.15).

Table 1. Effects of Lactonin on rectal temperature and respiratory rate

		G1	G2	G3	G4	Mean
RT	LT	38.77 ± 0.08*	39.47 ± 0.17	39.23 ± 0.15	39.22 ± 0.14	39.20 ± 0.08
	Cont	39.20 ± 0.13	39.01 ± 0.16	39.13 ± 0.20	38.87 ± 0.09	39.05 ± 0.08
RR	LT	76.1 ± 4.0	81.0 ± 5.8	78.0 ± 5.5	80.9 ± 3.7	81.2 ± 2.4
	Cont	72.3 ± 4.2	69.3 ± 3.5	76.0 ± 3.9	80.4 ± 4.0	74.5 ± 2.0
THI						79.2

RT - rectal temperature, RR - respiratory rate; *P<0.05

Mean value of milk fat (%) in LT groups increased (P<0.05) and milk protein (kg/d) tended to be higher than those in Control (0.05<P<0.15). Somatic cell count tended to be lower (0.05<P<0.15).

Table 2. Effects of Lactonin on milk production of heat-stressed cow

		G1	G2	G3	G4	Mean
Milk yield, kg/d	LT	16.8 ± 0.8*	19.9 ± 0.6	21.6 ± 1.1	26.9 ± 1.0	21.4 ± 0.7
	Cont	13.8 ± 1.2	20.3 ± 0.6	21.5 ± 1.2	27.7 ± 0.9	20.7 ± 0.9
FCM, kg/d	LT	17.2 ± 0.9*	20.4 ± 0.7	22.7 ± 1.2	28.5 ± 1.0	22.3 ± 0.8
	Cont	13.8 ± 1.3	20.7 ± 0.9	21.6 ± 1.2	28.9 ± 0.9	21.1 ± 0.9
Milk fat, %	LT	3.67 ± 0.14	3.67 ± 0.06	3.83 ± 0.08*	3.88 ± 0.10	3.76 ± 0.05*
	Cont	3.40 ± 0.13	3.59 ± 0.11	3.52 ± 0.09	3.76 ± 0.07	3.56 ± 0.05
Milk protein, %	LT	3.25 ± 0.09	3.11 ± 0.06	3.39 ± 0.35	3.06 ± 0.10	3.20 ± 0.09#
	Cont	3.16 ± 0.07	3.08 ± 0.06	3.00 ± 0.08	2.9 ± 0.06	3.03 ± 0.04
Milk protein, kg/d	LT	0.54 ± 0.02*	0.62 ± 0.01	0.71 ± 0.04	0.82 ± 0.03	0.67 ± 0.02#
	Cont	0.43 ± 0.04	0.63 ± 0.03	0.64 ± 0.03	0.8 ± 0.02	0.62 ± 0.02
SMC, 103/ml	LT	398 ± 62#	486 ± 118	420 ± 76	259 ± 75	395 ± 44#
	Cont	659 ± 122	655 ± 163	541 ± 86	270 ± 87	529 ± 62
FCR	LT	0.47 ± 0.02*	0.56 ± 0.02	0.61 ± 0.03	0.76 ± 0.03	0.60 ± 0.02
	Cont	0.39 ± 0.04	0.57 ± 0.02	0.60 ± 0.03	0.78 ± 0.02	0.58 ± 0.05

*P<0.05, **P<0.01, # - 0.05<P<0.15; SMC - somatic cell; FCR - feed conversion rate

Table 3. Effects of lactonin on plasma hormones

		T_3, ng/ml	T_4, ng/ml	Insulin, μIU/ml
G1	LT	1.50 ± 0.07#	57.73 ± 4.88*	47.30 ± 4.16**
	Cont	1.64 ± 0.04	70.39 ± 2.26	30.32 ± 4.58
G2	LT	1.73 ± 0.04	68.61 ± 4.28	47.59 ± 4.83**
	Cont	1.67 ± 0.10	66.32 ± 5.26	25.84 ± 2.09
G3	LT	1.67 ± 0.07	76.22 ± 4.80	44.75 ± 5.58
	Cont	1.78 ± 0.05	77.16 ± 5.31	32.08 ± 7.16
G4	LT	1.65 ± 0.07#	64.15 ± 3.36	48.66 ± 4.10**
	Cont	1.80 ± 0.05	67.89 ± 4.50	26.10 ± 2.83
Mean	LT	1.65 ± 0.03#	66.80 ± 2.27	47.18 ± 2.21**
	Cont	1.72 ± 0.03	70.44 ± 2.27	28.26 ± 2.32

* P<0.05, ** P<0.01, # - 0.05<P<0.15

Lactonin altered plasma hormone status. Insulin concentration enhanced significantly in all LT groups (P<0.01), except G3. Whereas concentration of T_4 was significantly lower in LT of G1, and mean concentration of T_3 tended to be lower in LT groups.

DISCUSSION

The upper critical temperature for lactating cows is when the THI exceeds 72. The ambient THI imposed in this trial, exceeded 76 through whole experiment period, was sufficient to cause heat stress (HS) in the cows (Igono et al., 1992). Administration of Lactonin to cows exhibited resulted lower rectal temperature and higher respiratory rate, indicating cows were able to dissipate a possible greater heat production induced by Lactonin. Likely, Lactonin, being similar with bST, enabled cows to maintain normal body temperatures under HS (Johnson, 1991). Lactonin impacted effects on plasma hormone status. On the one hand Lactonin led to a lower concentrations of T_3 and T_4. This could avoid cow produce more heat in hot environments. On the other hand, Lactonin increased insulin concentration, which helped cow to maintain a more normal metabolism in hot summer and induced the expression of heat-shock protein gene hsp70 (Takeda et al., 2001). Taken together, Lactonin facilitates dairy cow adapted to hot environments and improves cow performance. In this trial the low yielding cows were more responsive to Lactoin than the high yielding cows, which was also observed in bST experiment.

Lactonin increased milk yield, FCM, milk fat, milk protein and feed conversion rate, but reduced somatic cells of heat-stressed cow during hot summer. These positive effects of Lactonin on cow performance were associated with Lactonin-dependent alteration of plasma insulin, T_3 and T_4.

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