Effects of Monensin Administation on Mammary Function in Late Lactating Crossbred Holstein Cattle**

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ABSTRACT : An experiment was carried out to study the effect of monensin administration on mammary functions in crossbred Holstein cattle. Fourteen non-pregnant late lactating crossbred Holstein cattle, approximately 270 days postpartum, were selected for the experiment. They were divided into two groups of 7 animals each. Seven animals in the treated group were given sodium monensin orally in a slow-release capsule. Animals in both control and treated groups were fed the similar diet to maintain milk production and body score at 2.5. Rice straw was fed as a source of dietary fiber throughout the experimental period. After monensin administration, a significant increase in the molar percent of ruminal propionate ($p<0.05$) and a significant decrease in the molar percent of ruminal acetate ($p<0.05$) were apparent in comparison to the pretreated period. The ratio of acetate to propionate concentration decreased significantly after monensin administration ($p<0.05$), while it was maintained at the similar level throughout the period of experiment in the control group. Monensin did not affect the molar percent of ruminal butyrate and valerate. The concentration of milk allantoin between the control group and monensin treated group was not different. An excretion rate of allantoin in milk decreased in animals treated with monensin ($p<0.05$). Mammary blood flow did not show significant difference between control and monensin treated groups. The plasma glucose concentration, arteriovenous concentration difference and mammary gland uptake of glucose remained constant in both groups. Milk yield of the later stage of lactation in the control group declined during lactation advance while a tendency to increase in the milk yield was apparent after 21 days monensin administration. Milk compositions for concentration of lactose, fat and protein in both control group and monensin treated group did not change throughout the experimental periods. From these results, it can be concluded that the action of monensin could affect the ruminal fermentation pattern. Monensin could not increase milk yield in the late lactating period. *(Asian-Aust. J. Anim. Sci. 2001. Vol 14, No. 12 : 1712-1718)*

Key Words : Crossbred Holstein Cattle, Monensin, Mammary Gland, Lactation

INTRODUCTION

Monensin, the carboxylic polyether ionophore synthesing from bacteria (Haney and Hoehn, 1967), has been used in dairy cattle for improving feed efficiency. Monensin is also used as an antiketogenic and antibloat agent by its inhibitory effects on certain microorganisms in the rumen (Bergen and Bates, 1984). The presence of monensin in the rumen can affect both volatile fatty acid (VFA) and nitrogen productions (Schelling, 1984). The dairy cattle treated with monensin and feeding on either high fiber dietary or high concentrated dietary sources has been shown a tendency to increase the net portal flux and portal-arterial difference of glucose concentration whereas the net hepatic and the total splanchnic flux of glucose, L-lactate, β-hydroxybutyrate, acetate, propionate and butyrate are not affected (Harmon and Avery, 1987; Harmon et al., 1993). The arterial plasma glucose concentration in the cow treated with monensin has been shown either no alteration (Harmon and Avery, 1987; Sauer et al., 1989; Harmon et al., 1993; Hayes et al., 1996; Ramanzin et al., 1997) or increase (Abe et al., 1994). The concentration of other arterial plasma metabolites are not consistently affected except the decrease in the level of plasma β-hydroxybutyrate (Harmon and Avery, 1987; Sauer et al., 1989; Abe et al., 1994). An increase in milk yield has been noted after treated with monensin in pastured cows (Lowe et al., 1990; Lean et al., 1994; Hayes et al., 1996), but it was not apparent in the feedlot cow (Baile et al., 1982; Sauer et al., 1989; Abe et al., 1994; Ramanzin et al., 1997). A number of studies have shown that an increase in milk production per amount of feed intake is thought to concern to an absolute decrease in feed intake during monensin treatment.

It is known that large quantities of glucose are removed by the mammary glands for synthesis of milk lactose (Annison and Linzell, 1964). An increase in the plasma glucose concentration during monensin supplement should be a possible route to supply more glucose to the mammary cell. The plasma glucose concentration depends directly on the gluconeogenic capacity of the liver and the utilization of glucose by many organs. Since the main glucogenic substrate in the ruminant is propionate, an increase in the ruminal propionate concentration by the action of monensin would increase glucose availability to the mammary gland. This physiological mechanism has been used to explain the

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effect of monensin on milk production (Lowe et al., 1990; Lean et al., 1994; Hayes et al., 1996). Thus, the present study was conducted to obtain more information on whether monensin administration in crossbred dairy cattle feeding on rice straw during late lactation should be a potential dietary supplement for milk production.

MATERIALS AND METHODS

Animal and feed management

Fourteen non-pregnant late lactating crossbred Holstein cattle, approximately 270 days postpartum, were used in the experiments. They were divided into two groups of seven animals each. Animals were housed in tie stall type shades, having a solid floor and open sides. An ambient temperature in the shed was recorded daily and averaged weekly during the experiments. The maximum temperature in the shed at noon and the minimum at night were 34±1°C and 26±1°C, respectively. The relative humidity was 68 ± 12%. All cows were fed twice a day with concentrate and rice straw as the main source of roughage (table 1). The animals individually received the concentrate mixture (7-10 kg/day) to maintain a moderate body score condition (2.5 scale = 1 to 5) in combination with roughage *ad libitum*. Water and mineral block were available for *ad libitum* intake. The composition of minerals in 1 kg lick block consists of Na, 136 gm; Ca, 140 gm; P, 60 gm; Mg, 20 gm; K, 25 gm; S, 12 gm; Fe, 1,000 mg; Zn, 800 mg; Mn, 350 mg; Cu, 300 mg; Co, 80 mg; I, 245 mg; and Se, 20 mg. Cows were milked twice a day at 06:00 and 16:00 h and milk yields were recorded by weight. Body weight of individual animal was recorded weekly through the period of the experiment.

Experimental procedures

Two consecutive periods of both pretreatment and experimental periods were assigned in both control and the treated groups. The period of study in each group consisted of 14 days of the pretreated period and 56 days of the experimental period. In the pretreated period, animals in both groups were allowed to adjust to the type of diet. In the experimental period, animals in the control group were given similar diets throughout the period of experiment. Animals in the treated group individually received orally with a slow-released intraruminal monensin capsule (Rumensin®; Elanco Animal Health, Wiri, Auckland, New Zealand), containing 32 gm of sodium monensin. The slow-released form had 100 days duration of action and it released approximately 320 mg of monensin per day. Each capsule number was recorded and the animal was monitored for 20 minutes after treatment to ensure that the animal was successfully treated. Mammary blood flow measurements, milk yield and ruminal fluid collections were performed on days 14, 21, 28, 42 and 56 after monensin administration.

Table 1. Chemical composition of experimental diet and nutrient analysis as a percentage of dry matter

	Rice straw	Concentrate
Dry matter	85.9	89.4
Crude protein	2.26	17.8
Acid detergent fibre	61.2	21.5
Neutral detergent fibre	67.2	28.8
Lignin	8.8	7.0
Ash	16.8	5.6

Concentrate formation: ingredients by fresh weight (100 kg) consisted of soy bean meal (30 kg), cotton seed (25 kg), cassava (25 kg), rice bran (15 kg), dicalcium phosphate (2 kg), sodium bicarbonate (1.7 kg), potassium chloride (0.7 kg) and premix (0.6 kg).

Measurements of mammary blood flow, mammary arteriovenous difference, mammary extraction ratio and uptake of glucose

On the day of the experiment, mammary blood flow was determined in duplicate. Mammary blood flow for half of the udder was determined by the dye T-1824 dilution technique as described by Chaiyabutr et al. (1997). The whole mammary blood flow was calculated by doubling the half-udder mammary blood flow since the yields of the two half of the udder were similar.

The plasma glucose concentration from both coccygeal artery and milk vein were determined by colorimetric method using the enzymatic oxidation in the presence of glucose oxidase (Human GmBH, Germany). Arteriovenous concentration difference and mammary extraction ratio of glucose were calculated from the plasma glucose concentration in blood from both coccygeal artery and milk vein. Mammary uptake of glucose was calculated using mammary plasma flow and glucose arteriovenous difference of the gland.

Determination of milk compositions

Milk sample collected by hand milking was divided into two portions, the fresh milk sample and formalinized milk sample (1 ml of 40% formalin in 60 ml of fresh milk). All milk samples were kept at -20°C. Formalinized milk samples were analysed for protein, lactose and fat concentrations as followed. Milk protein concentration was analyzed using the micro-Kjeldhal method. Milk lactose concentration was analyzed by the colorimetric method (Teles et al., 1978). Milk fat concentration was measured using microcapillary method as described by Chaiyabutr (1994). The concentration of milk allantoin was analyzed from fat and protein-free milk sample by the method modified from Young and Conway (1942).

Determinations of ruminal volatile fatty (VFA) acid concentrations

The ruminal content was collected by stomuch tube after 2.5 h of morning feeding, since the total VFAs reached a maximum concentration approximately 2.5 h after each meal (Whitelaw et al., 1970). After intubation, the ruminal content was collected by suction force using the air pump. The ruminal content was filtrated immediately using two layers of cheeseclot, and the ruminal fluid was collected and preserved by adding 3 ml of 6 N hydrochloric acid to 60 ml of ruminal fluid. The preserved ruminal fluid sample was freezed at -20°C until ruminal VFA concentrations were analyzed.

Ruminal VFA concentrations were analyzed by the method modified from Erwin (1961). Frozen ruminal fluid was thawed and centrifuged at 3,000 rpm for 10 minutes. The supernatant (0.7 ml) was added with 0.4 ml of the internal standard mixture, 1 volume of 1.6% of 4-methyl-n-valeric acid in 4 volume of 25% metaphosphoric acid. The concentration of VFA was measured by gas chromatography equipped with a hydrogen flame ionization detector (Shimadzu, Model 7 AG, Japan). A 3.3 mm (id) 2 m column containing FFAP, was operated at 160°C with a nitrogen flow rate of 40 ml/min. The composition of individual VFA was expressed as mole/100 mole of the total VFA concentration.

Statistics

All data were reported as the mean values ± S.D. The paired t-test was used to estimate the significant difference of the values within group. The unpaired t-test was used to estimate the significant difference of values between groups (Glantz, 1992).

RESULTS AND DISSCUSSION

The molar percent of ruminal VFA during monensin administration

An increase in ruminal VFA concentration in late lactation crossbred dairy cattle treated with monensin in the present experiment show the similarity of the typical changes as those observed in animals feeding with monensin (Schelling, 1984). The molar percent of ruminal propionate increased, while the molar percent of ruminal acetate decreased in animals treated with monensin (table 2). These would lead to lower the ratio of ruminal acetate to propionate in animals treated with monensin. The ratio of acetate to propionate reduced at day 14 and 28 ($p<0.05$) in comparison to day 0. However, the molar percent of ruminal butyrate did not alter after monensin administration. This phenomenon has been noted from several previous studies (Zinn et al., 1994; Ramanzin, 1997; Richardson et al., 1976). It is known that the main ruminal microbial populations in cows feeding on the high forage ration are

Table 2. The ruminal VFA compositions, the total concentration and the ratio of acetate to propionate in the control group and the group treated with monensin

Monensin treatment	Individual VFA(mol/100 mol)								Total VFA(mM):		C2/C3 ratio:	
	Acetate:		Propionate:		Butyrate:		Valerate:					
	Control	Treated	Control	Treated	Control	Treated	Control	Treated	Control	Treated	Control	Treated
Pretreatment period	71.6±2.5	69.4±3.6	18.4±1.7	19.1±2.9	9.3±1.0	10.7±1.7	0.71±0.16	0.73±0.19	104.15±25.0	111.56±21.0	3.94±0.5	3.71±0.7
14 day after treatment	70.8±3.0	67.3±3.4**	17.7±1.7	20.4±3.1*	10.8±2.3	11.5±1.9	0.72±0.17	0.78±0.16	111.30±20.9	143.77±43.2	4.04±0.5	3.38±0.6*†
21 day after treatment	69.2±3.0**	67.9±4.2*	18.4±2.3	20.2±2.9	11.6±2.2*	11.2±3.2	0.80±0.19	0.77±0.27	106.30±24.3	136.17±27.4†	3.82±0.6	3.44±0.63
28 day after treatment	71.3±3.5	67.0±3.5†	16.9±1.6	21.0±2.1*††	11.0±2.8	11.1±2.4	0.77±0.18	0.91±0.29	110.66±28.3	150.12±38.8†	4.25±0.5	3.25±0.4*†
42 day after treatment	70.9±1.9	68.8±4.0	18.4±1.6	20.0±3.5	9.9±1.4	10.5±1.9	0.80±0.17	0.70±0.38	127.69±23.3	128.27±24.5	3.87±0.4	3.60±0.8
56 day after treatment	71.8±1.2	68.0±4.3	18.1±1.5	21.4±2.3*†	9.4±1.3	10.0±2.3	0.74±0.12	0.75±0.39	112.10±18.0	141.95±46.7	4.00±0.4	3.36±0.6†

Values are means ± SD (n=7).

P-value by paired *t*-test with respect to the Pretreatment period in the same group, (** $p<0.01$, * $p<0.05$).

P-value by unpaired *t*-test with respect to the similar period of experiment between control and monensin treated groups, (†† $p<0.01$, † $p<0.05$).

acetate, butyrate and lactate producing bacteria, whereas propionate producing bacteria is the predominant microbial population living in the rumen of cow feeding on the high concentrated diet (Phillipson, 1970). The action of monensin is more specific to the gram-positive bacteria, including acetate, butyrate and lactate producing bacteria (Bergen and Bates, 1984). The effect of monensin in the present experiment may be related to the type of dietary intake, the microbial population and activities and the host adaptability (Schelling, 1984).

The plasma glucose concentration, mammary blood flow and mammary glucose uptake

In the present experiment, plasma glucose concentration did not alter throughout the experimental period in both control animals and animals treated with monensin, although an increase in the plasma glucose concentration has been shown to be an important point for explain the action of monensin on milk production (table 3; Abe et al., 1994; Heyes et al., 1996; Ramanzin et al., 1997). Since propionate can be converted to cytosolic oxaloacetate via succinyl-CoA and mitochondrial malate, this complex of the metabolic regulation of glucose synthesis in the liver may be the major limiting and determination factors for systemic glucose concentration. An increasing in ruminal propionate during monensin administration could spare gut glucose utilization and tended to increase the plasma concentration of glucose in the portal circulation, but would not affect the plasma glucose in systemic circulation (Harmon and Avery, 1987; Harmon et al., 1993). However, it has been shown that postruminal addition of propionate to the level similar to that of the production of propionate in the rumen when cows are fed a high-grain, low fiber diet did not show significant effect on systemic plasma glucose, insulin, milk yield and milk composition (Frobish and Davis, 1976). An increase in the amount of ruminal propionate has been reported to vary from 100% (Frobish and Davis, 1976) to 5.7% (Harmon et al., 1993). In the present experiment, ruminal propionate increases 12% after monensin administration. It could be suggested that monensin supplementation at the level 320 mg/day/cow could not raise systemic plasma glucose.

Mammary blood flow, mammary arteriovenous concentration difference, the extraction ratio of glucose and glucose uptakes by the mammary gland in the present study were not affected by monensin supplementation throughout the experimental period. It is known that increasing glucose available to the mammary gland should increase milk yield because glucose is the main substrate for lactose synthesis and lactose provides the main osmotic property of the milk (Linzel and Peaker, 1971). The utilization of glucose by the mammary gland is determined by its uptake. Glucose uptake by the gland is affected not only by the concentration of glucose in the systemic circulation but also by the activity of mammary gland. However, plasma glucose is not the main factor determining the glucose

Table 3. Arterial plasma concentration of glucose, plasma glucose A-V difference, mammary extraction ratio, mammary glucose uptake, mammary blood flow and haematocrit in the control group and the group treated with monensin

Monensin treatment	MBF (ml/min):		Haematocrit (%):		$[glu]_a$ (mM):		A-V difference (mM):		Extraction ratio (%):		Glucose uptake (μmole/min):	
	Control	Treated	Control	Treated	Control	Treated	Control	Treated	Control	Treated	Control	Treated
Pretreatment period	3,201±1,046	4,191±950	28.3±2.3	31.7±1.6	3.54±0.19	3.27±0.54	0.90±0.37	0.82±0.23	25.4±10.2	25.2±6.1	1,991±7,736	2,287±627
14 day after treatment	3,227±1,043	4,344±1,269	29.6±3.2	31.1±1.6	3.24±0.24	3.09±0.44	0.93±0.49	0.62±0.26	28.3±13.3	20.4±8.8	2,200±1,792	1,930±1,099
21 day after treatment	4,547±2,011	4,796±2,401	29.1±3.1	30.4±2.3	3.30±0.33	3.33±0.28	0.74±0.31	0.75±0.17	22.5±10.0	22.3±4.7	2,244±1,303	2,500±1,614
28 day after treatment	3,772±1,643	4,711±1,859	29.1±3.0	30.4±2.0	3.29±0.31	3.18±0.33	0.53±0.22	0.62±0.30	16.0±7.0	19.2±8.4	1,471±1,021	2,023±1,168
42 day after treatment	3,559±1,203	4,049±657	29.5±3.0	30.6±1.6	3.36±0.24	3.16±0.30	0.82±0.19	0.77±0.23	24.6±6.0	24.6±7.4	2,054±901	2,149±649
56 day after treatment	3,605±720	4,069±2,160	29.1±2.7	29.5±2.9	3.38±0.25	3.20±0.42	0.82±0.31	0.67±0.20	24.4±9.6	20.7±4.9	2,173±1,019	1,865±1,071

Values are means ± SD (n=7).

Abbreviation: MBF, Mammary blood flow; $[glu]_a$, Arterial plasma glucose concentration; A-V dif, Arteriovenous difference of glucose.

Table 4. Milk allantoin concentration and excretion in the control group and the group treated with monensin

Monensin treatment	Allantoin (μmole/L):		Percent change (%):		Allantoin excretion (mmole/day):		Percent change (%):	
	Control	Treated	Control	Treated	Control	Treated	Control	Treated
Pretreatment period	551.15±88.80	578.86±70.63	-	-	4.07±0.44	5.71±1.75†	-	-
14 day after treatment	529.66±74.88	552.32±44.07	-3.48 ±6.5	-3.58 ±11.9	3.96±0.73	5.40±1.41†	-3.07±10.77	-3.55±16.21
21 day after treatment	546.07±69.65	538.07±41.68	-0.36±5.7	-5.91±12.5	3.96±0.81	5.52 ±1.66†	-3.12±13.96	-2.43±17.58
28 day after treatment	530.44±71.46	545.01±65.26	-3.27±6.3	-5.28±10.0	3.72±0.76	5.63±2.08†	-8.38±18.64	-2.76±12.67
42 day after treatment	542.06±64.12	540.64±59.30	-0.74 ±9.9	-5.54±13.7	3.85±1.05	5.29±1.92	-6.07±19.19	-8.43±17.34
56 day after treatment	529.44±56.19	537.34±42.40	-3.04±8.0	-6.36±9.5	3.55±0.95	5.06±1.97*	-13.21±17.34	-12.85±15.65

Values are means ± SD (n=7).

P-value by paired *t*-test with respect to the Pretreatment period in the same group, (* $p<0.05$).

P-value by unpaired *t*-test with respect to the similar period of experiment between control and monensin treated groups, († $p<0.05$).

transport across membrane of the mammary cells (Baldwin and Kim, 1993). In the physiological state, the mammary cells have a steep gradient of glucose across the plasma membrane, from 3.0 to 3.5 mM in plasma to 0.1 to 0.3 mM in the cell (Faulkner et al., 1981). The mechanisms, glucose transporter type I (GLUT1) which control glucose uptake across the plasma membrane is the main limiting factor for this high glucose concentration gradient. The uptake capacity of glucose by the mammary cell has been shown to relate to glucose transporter type 1 (GLUT1) (Zhao, 1996). GLUT1 regulation is a complex mechanism which operates in both acute and long term regulations including the regulation during stress and through hormonal status regulation (Fawcett et al., 1991). An inability to change the level of the hormone concentration e.g. growth hormone and insulin after monensin feeding has been noted (Duff et al., 1994). It would be the reason for the present results that monensin administration could not raise the uptake of glucose by mammary gland either raising systemic plasma glucose concentration or altering the uptake capacity.

Milk allantoin concentration and allantoin excretion

Monensin could decrease bacterial nitrogen reaching the abomasum and increase in dietary protein to the lower gut (Poos et al., 1979). In the present experiment, both the concentration of milk allantoin and the allantoin excretion at day 56 were lower than that of the day 0 after monensin administration ($p<0.05$), which were not apparent in the control group. This result would also indicate the lower microbial activity (Giesecke et al., 1994). However, The present results for the milk allantoin excretion contradict with those demonstrated in the urinary allantoin-nitrogen excretion in sheep, which unaffected by monensin administration (Dewhurst and Webster, 1992). Host adaptibility after monensin treatment may be subjected to species differences (Schelling, 1984). In comparison between the control group and the group treated with monensin, the allantoin excretions in the group treated with monensin were higher than those of the control group throughout the experimental period (table 4). This effect would be in part due to the higher milk yield in the group treated with monensin than those of the control group (table 5).

Milk yields and milk compositions

Daily milk yield of the animals in the control group declined as lactation advance to the later stage of lactation. In animals treated with monensin, milk yield slightly increased at the first 4 weeks (≈2-3%) and then declined through the last 4 weeks of the experiment. A non-significant increase in milk yield in late lactation period after monensin treatment in this experiment may indicate that monensin increased the capacity of the mammary cell itself. According to the study of Wilde and Knight (1989), the capacity of the individual mammary cell does not decrease as milk yield falls after peak of lactation, while the progressive decline in milk yield is associated with a net fall in cell number. Increasing cells capacity as influenced by the monensin feeding may come from increasing feed efficiency and protein utilization (Goodrich et al., 1984). According to Clark (1974), post-ruminal supplementation of protein increased milk production. Milk compositions for lactose, protein and fat concentrations were not significantly different between the control group and the group treated with monensin in the present study. However, the lower in the percentage of milk fat during monensin treatment in dairy cattle has been noted (Sauer et al., 1989; Lowe et al.,

Table 5. Milk production and compositions in the control group and the group treated with monensin

Monensin treatment	Milk yield (kg/day):		% changed :		Milk composition					
					Protein(gm%):		Fat (gm%):		Lactose (gm%):	
	Control	Treated	Control	Treated	Control	Treated	Control	Treated	Control	Treated
Pretreatment period	7.57±1.6	9.95±3.0	-	-	3.46±0.43	3.54±0.25	4.54±1.43	4.09±1.51	4.19±0.36	4.37±0.36
14 day after treatment	7.64±1.9	9.91±3.0	0.36±8.12	-0.37±8.12	3.51±0.39	3.50±0.38	3.55±1.06	3.13±1.09	4.11±0.16	4.20±0.21
21 day after treatment	7.44±2.2	10.23±3.0	-2.79±12.6	3.30±9.69	3.54±0.39	3.49±0.26	3.64±1.33	3.61±1.53	4.19±0.50	4.22±0.34
23 day after treatment	7.15±1.8	10.18±3.0†	-5.52±16.1	2.70±9.65	3.47±0.48	3.44±0.33	4.56±1.42	3.52±1.84	4.09±0.25	4.05±0.71
42 day after treatment	7.34±2.8	9.69±3.3	-5.39±17.0	-3.56±7.27	3.45±0.41	3.44±0.23	4.63±1.67	3.42±1.43	3.96±0.51	4.18±4.13
56 day after treatment	6.91±2.5	9.35±3.4	-10.9±13.2	-7.44±9.51	3.41±0.43	3.75±0.80	5:53±1.14	4.27±2.12	3.95±0.40	4.26±3.33

Values are means ± SD (n=7).

P-value by unpaired *t*-test with respect to the similar period of experiment between control and monensin treated groups, (†$p<0.05$).

1990; Abe et al., 1994). It is known that lower milk fat is related to the lower concentration of ruminal butyrate (Schelling, 1984). In the present study, milk fat concentration was not affected by the monensin feeding, which was probably related to either the late stage of lactation or nonsignificant change of the ruminal butyrate concentration.

In conclusion, non-pregnant late lactating crossbred Holstein cattle were used in the present study and animals showed low milk yield in this period. The effect of hormones on mammary functions being established by pregnancy would be ruled out during monensin administration. The effects of monensin involve the pattern of the ruminal fermentation. An increase in the concentration of ruminal propionate in animals treated with monensin did not affect both the systemic plasma glucose concentration and the uptake capacity by the mammary gland.

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