CHAPTER V

DISCUSSION

In the present experiment, measurements of ruminal VFA composition, mammary gland activities and milk production were performed in order to clarify the action of monensin supplementation in late lactating crossbred Holstein cattle feeding on rice straw.

Monensin administration in cattle has been shown to affect both the concentration of VFA and microbial activities in the rumen. An increase in the concentration of propionate in the rumen has been reported, while the concentrations of both acetate and butyrate decrease. The lower the ratio of ruminal acetate to propionate concentration would show in the treated cow when compared to that of the untreated cow (Schelling, 1984). In the present results, changes in ruminal VFA concentrations in animals treated with monensin showed the similarity of the typical changes as those observed in animals feeding with monensin (Schelling, 1984). However, the concentration of butyrate in the rumen did not alter after monensin treatment in the present experiment. This phenomenon also noted from several previous studies (Zinn et al., 1994; Ramanzin, 1997; Richardson et al., 1976). This may be related to the type of dietary intake. It has been shown that the main microbial population in the rumen in cows feeeding on the high forage ration are acetate, butyrate and lactate producing bacteria, while propionate producing bacteria is the predominant microbial population living in the rumen of cow feeding on the high concentrated diet (Phillipson, 1970). In the present study, rice straw was given to animals as the main source of dietary fiber. The proportion of VFA produced in all crossbred Holstein cattle in the present study were consistent with the earlier report in

cow fed with high fiber diet ration. The less action of monensin in cow fed with high forage diet than that fed with low forage diet would be due to the differences in microbial populations, since the mode of action of monensin is more specific to the gram positive bacteria, including acetate, butyrate and lactate producing bacteria (Bergen and Bates, 1984). However, no data are available on the mode of action of monensin relating to different levels of dietary fiber and microbial population. In the present experiment, the ratio of acetate to propionate also decreased in animals treated with monensin while it was not apparent in the control group (Figure 2). These results would support the relationship between monensin action and dietary composition, which monensin involves in the viewpoint of the ruminal fermentation pattern (Schelling, 1984).

The milk allantoin concentration has been used as a parameter representing the ruminal microbial activities (Giesecke et al., 1994). The effect of monensin on microbial activities in the rumen depends on the host adaptability on microbial growth (Schelling, 1984). Monensin could decrease bacterial nitrogen reaching the abomasum and increase in dietary protein to the lower gut (Poos et al., 1979). The present experiment in both the control and treated groups, the milk allantoin concentrations seemed remained constant throughout periods of experiment. However, both of the concentration of milk allantoin and the allantoin excretion at week 8 was lower than that of the first week after monensin administration (Figure 3). It did not occur in the control group. This may indicate the lower microbial activity after monensin administration and the alteration in the protein degradation pattern may be evidence. However, 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. This effect would be due to the higher milk yield in the group treated with monensin than those of the

control group. 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 treatment (Dewhurst and Webster, 1992). The host adaptibility after monensin treatment may be subjected to species differences.

In the present experiment, daily milk yield from the control animals declined as lactation advance to the later stage of lactation. In the animals treated with monensin, milk yield slightly increased at the first 4 weeks and then declined through the last 4 weeks of the experiment. In comparison between the control and the treated groups, the percentage of changes of milk yield were not different. Milk yield in the group treated with monensin increased 3.3% and 2.6% at week 2 and week 3 of treatment, respectively (Figure 4). The percent change in milk yield of the control group declined in the similar pattern of the late lactation. The small change in the milk volume observed in the present experiment may come from the effect of monensin on feed efficiency and protein utilization. The potential of monensin action especially in the later stage of lactating cow was apparent, although few data on monensin treatment during the late lactation are available. According to Clark (1974), post-ruminal supplementation of protein increased milk production. The higher in milk yield in animals treated with monensin in the present experiment may relate to the difference in the nitrogen substrate delivered to the mammary gland.

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 reported (Sauer et al., 1989; Lowe et al., 1990; Abe et al., 1994), which is related to the lower concentration of ruminal butyrate (Schelling, 1984). The milk fat concentration was not affected in the

monensin treated group in the present experiment, which was probably related to the nonsignificant change of the ruminal butyrate concentration. From these results in the present study, it seems that monensin had the positive effect on mammary function although it failed to increase the milk volume per day.

In the present experiment, the plasma glucose concentration did not alter throughout the experimental period in either control animals or animals treated with monensin. An increase in the plasma glucose concentration has been shown to be a positive result and an important point which could be used to explain the action of monensin on milk production (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, which appears mostly to be channeled towards glucose synthesis, the increased systemic plasma glucose concentration does not depend on the propionate availability. The complex of the metabolic regulation of glucose synthesis in the liver may be the major limiting factor, which determines systemic glucose concentration. Postruminal addition of propionate to the level similarity to that of the production of propionate in the rumen when cows are fed a high-grain, low fiber diet has no significant effect on systemic plasma glucose, insulin, milk yield and milk composition (Frobish and Davis, 1976). Studies on the effect of increasing ruminal propionate by monensin have been reported, which could spare gut glucose utilization and tends to increase the plasma concentration of glucose in the portal circulation, but does not affect the plasma glucose in systemic circulation (Harmon and Avery, 1987; Harmon et al., 1993). The variation in the amount of propionate increased in order to raise plasma glucose level has been reported by Frobish and Davis (1976) for 100%, 5.7% by Harmon et al. (1993) and 10% by the present experiment. It could be concluded that monensin supplementation at the level 320 mg/day/cow could not raise systemic plasma glucose. An increase in the ruminal propionate production by the

action of monensin in the present study was agree to those reported during feeding on monensin in ruminant (Brown and Hogue, 1985; Sauer et al., 1989; Ramanzin et al., 1997).

The mammary blood flow, mammary arteriovenous concentration difference and the extraction ratio of glucose in the present study were not affected by monensin supplementation throughout the experimental period. Glucose uptakes by the mammary gland were not different between control group and the group treated with monensin. It is known that increasing glucose available to the mammary gland should increase milk yield by the gland because the level of systemic glucose is the main substrate for lactose synthesis which 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 from the concentration of glucose in the systemic circulation but also by the gland activity. However, plasma glucose is not the main factor determining the glucose transport across membrane of the mammary cells. 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) and glucose uptake by the mammary cell has been shown to correlate to the other nutrients, especially the acetate (Miller et al., 1991a,b). In the present study, monensin administration could not raise the uptake of glucose by mammary gland either raising systemic plasma glucose concentration or altering the uptake capacity. 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, 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 could not raise mammary glucose uptake. A slight increase in milk yield after monensin treatment in this experiment may indicate that monensin improve milk yield in late lactation period. It may imply that the capacity of the mammary cell itself increased during monensin administration. According to the study by 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 decrease in the total DNA content of mammary parenchyma, representing a net fall in cell number. The mode of action of monensin may come from an increase in the utilizations of the other substrates such as amino acids and acetate other than glucose for lactose synthesis.

In conclusion, the effects of monensin on mammary gland function at the late lactating period of crossbred Holstein cattle would involve the pattern of changes in the ruminal fermentation. An increase in the concentration of ruminal propionate did not affect the systemic plasma glucose concentration and the uptake capacity by the mammary gland.

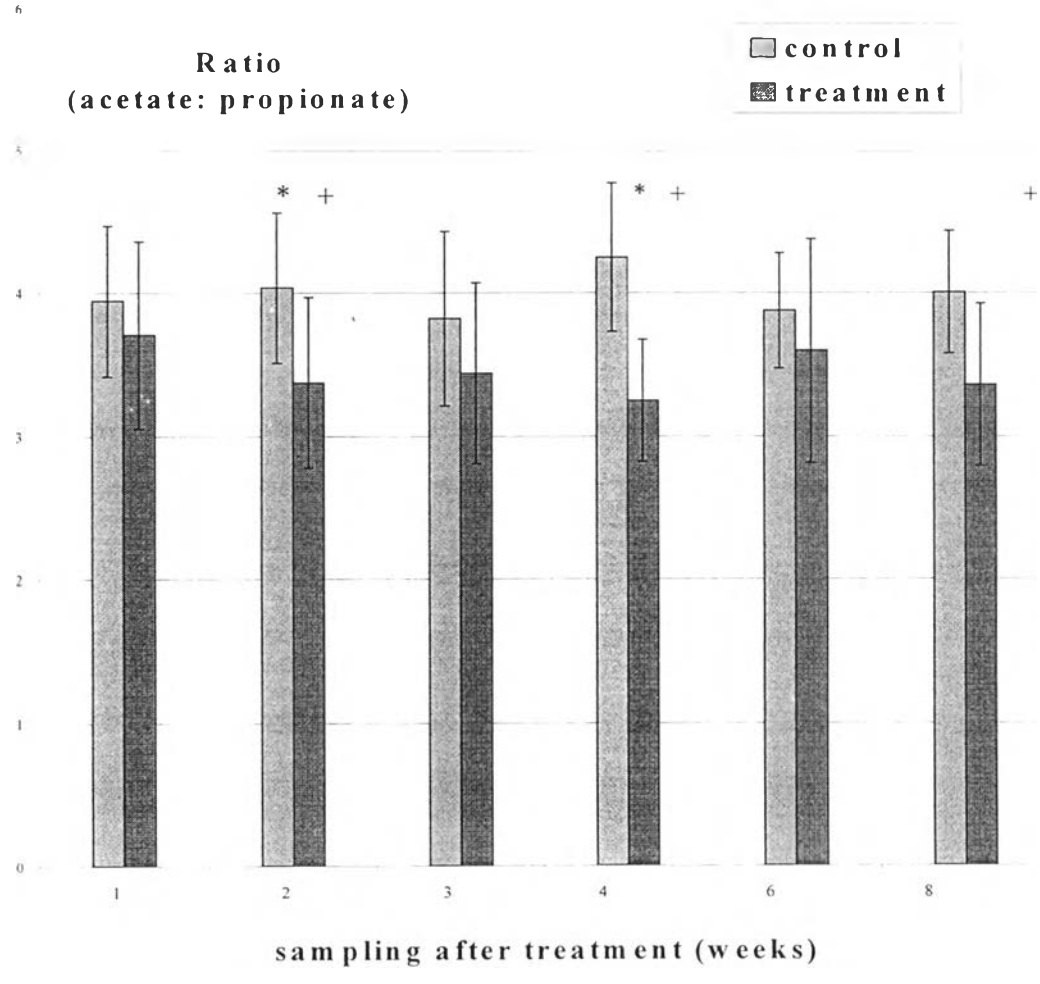

Figure 2 The ratio of acetate to propionate concentrations in the ruminal fluid in control group and the group treated with monensin.

P-value by paired *t*-test from the first week in the same group, (* P<0.05)

P-value by unpaired *t*-test between control and monensin treated groups,(+P<0.05)

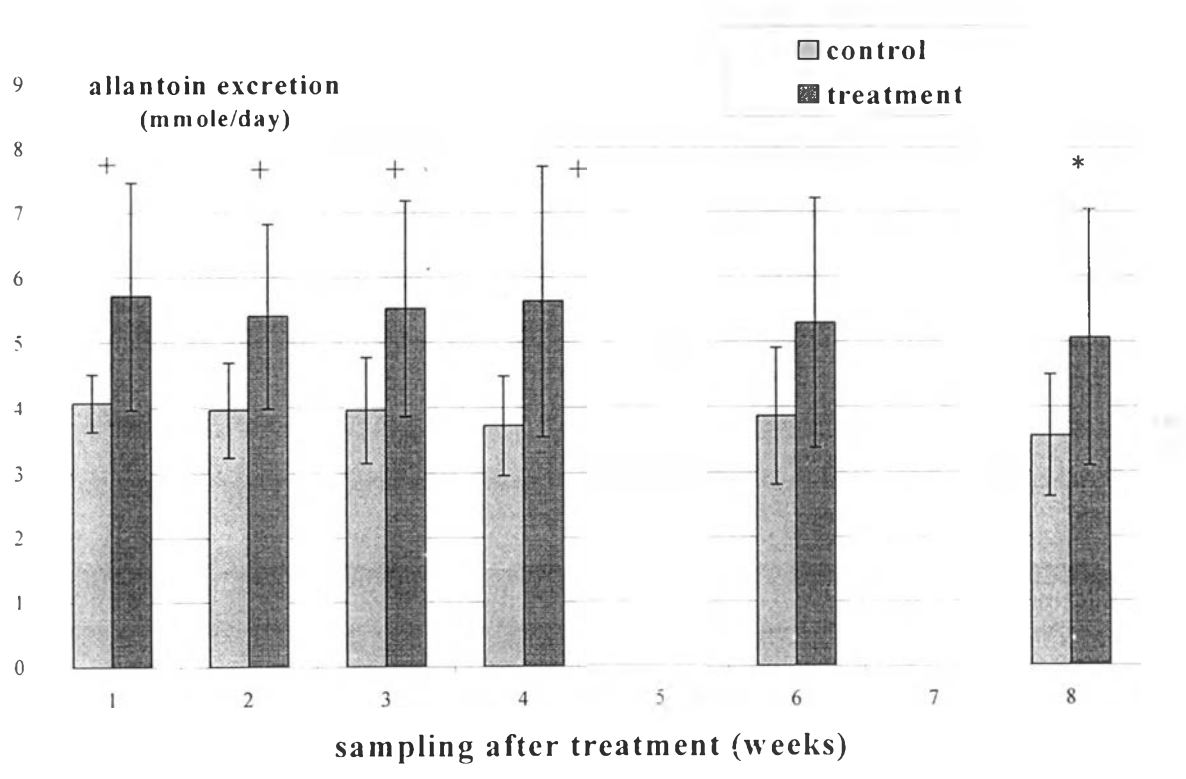

Figure 3. Milk allantoin excretion of control animals and animals treated with monensin.

P-value by unpaired *t*-test between control and monensin treated groups, *P < 0.05

P-value by unpaired *t*-test between control and monensin treated groups, (+ P<0.05).

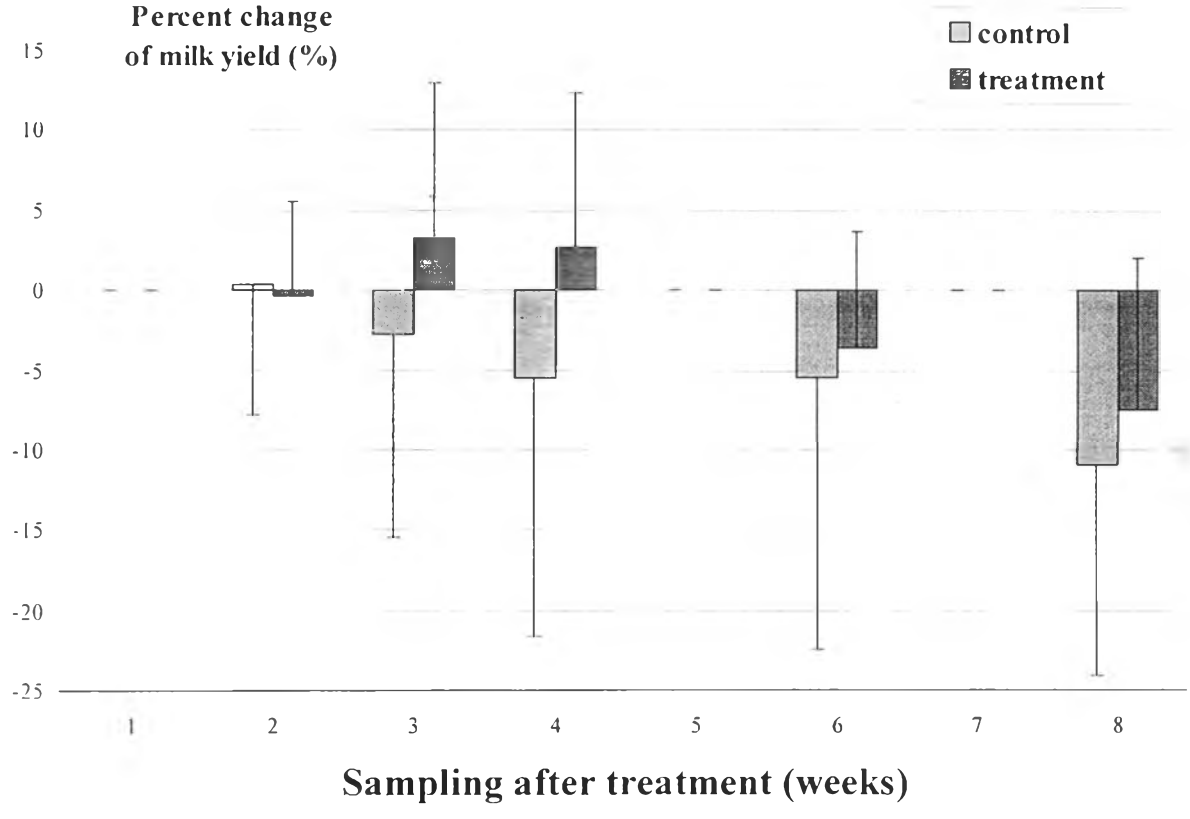

Figure 4 Changes in percentages of milk yield in the control group and the group treated with monensin.

P-value by unpaired *t*-test between control and monensin treated groups, *P < 0.05

P-value by unpaired *t*-test between control and monensin treated groups, ($^{+}$ P<0.05).