A total of eight crossbred wethers equipped with rumen fistulas were used in two experiments to complete a physiological study of acidosis in sheep. Experiment I was designed to test an experimental antibiotic at .25% of the amount of wheat given, two levels of NaHCO₃, 2 and 4%, expressed as a percent of the wheat given, and a combination of the antibiotic (.25%) plus 2% NaHCO₃. Acidosis was induced in the four sheep (controls) by feeding cracked soft white wheat (International Reference Number 4-05-337) at 50 g/kgBW^{0.75} divided among three feedings given over an 8-hr period on the third day of the experiment. Additional wheat was given depending on rumen pH values at 0800 of the fourth day. All control treatments were given wheat until a rumen pH below 4.5.
was obtained. Ruminal pH, rumen lactic and volatile fatty acids were monitored four times daily for 96 hr post engorgement.

The 2 and 4% NaHCO$_3$ and the antibiotic plus 2% NaHCO$_3$ treatments were all effective in maintaining a higher pH than the control treatment (P<.01). The antibiotic alone was effective in maintaining a higher pH but to a lesser degree (P<.05). Low levels of rumen lactic acid (<30 mM/liter) were detected on the antibiotic treatment. All treatments were effective in preventing acute acidosis, and none of the animals went off feed. The 4% NaHCO$_3$ treatment resulted in higher acetic acid concentrations than the treatments containing Thiopeptin (P<.05). Treatments containing Thiopeptin resulted in increases in propionic acid (P<.01) and reduced acetic:propionic ratios compared to the buffer treatments. Differences in total volatile fatty acids or gross energy calculated from volatile fatty acids were not significant between the treatments. Experiment II was designed to study the changes in rumen pH, motility and the synthesis of volatile fatty acids and lactic acid during the onset and recovery of induced lactic acidosis. Acidosis was induced by the addition of sucrose (15 g/kg BW) in 700 ml of water through the fistula. Mean maximum lactic acid concentration was near or in excess of 100 mM/liter of rumen fluid during the first
48 hr post-inducement. The minimum pH values observed for each sheep were 4.53, 4.13, 4.29 and 4.48. Propionic and butyric acids were absent from rumen samples by 14 hr post-inducement. Acetic acid concentrations decreased to <5 mM/liter during the first 24 hr following the addition of the sugar. Rumen motility decreased in frequency and amplitude during the first 4 hr. Normal rumen motility was observed in one sheep by 52 hr but not in another, until 84 hr post-inducement. Two of the four sheep were "off feed" by 4 hr post-inducement while the remaining two did not become anorexic until 7 hr. Animals began to consume small amounts of alfalfa when rumen pH values increased to >5.0 but consumption similar to pre-inducement intake was not observed until the rumen pH was >6.0 and lactic acid could no longer be detected in the rumen fluid.
A Physiological Study of Acidosis in Ruminants

by

Wesley W. Kezar

A THESIS

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Typed by Alanna K. Johnson for Wesley W. Kezar
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To my wife, Pat, and our children Jeff, Darin, Rick, Ryan and Kodi. We completed the degree as a family and grew in character and our love for each other because each had to sacrifice in order to accomplish our goal. We all wish to thank the people of Corvallis who became our close friends and made our time in Corvallis among the best years of our lives.
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CHAPTER I

Effect of Thiopeptin and Sodium Bicarbonate on the Prevention of Lactic Acidosis Induced in Sheep\textsuperscript{1,2}

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Key Words: Acidosis, Antibiotic, Sodium Bicarbonate, pH, Sheep, Lactic.

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\textsuperscript{2}Research reported was supported in part by Merck & Co., Rahway, NJ and by Church & Dwight Co., Inc., New York, NY.
\textsuperscript{3}Present address: Agricultural Industries & Services Div., Univ. of Minnesota Technical College, Waseca, MN 56093.
\textsuperscript{4}Department of Animal Science.
SUMMARY

Four crossbred wethers were utilized in a randomized block design to test Thiopeptin, an experimental antibiotic, at .25% of the amount of wheat given, two levels of NaHCO$_3$, 2 and 4%, expressed as a percent of the wheat given, and a combination of the antibiotic (.25%) plus 2% NaHCO$_3$. Acidosis was induced in the four sheep (controls) by feeding cracked soft white wheat (International Reference Number 4-05-337) at 50 g/kgBW$^{.75}$ divided among three feedings given over an 8-hr period on the third day of the experiment. Additional wheat was given depending on rumen pH values at 0800 of the fourth day. All control treatments were given wheat until a rumen pH below 4.5 was obtained. Ruminal pH, rumen lactic and volatile fatty acids were monitored four times daily for 96 hr post engorgement.

The 2 and 4% NaHCO$_3$ and the antibiotic plus 2% NaHCO$_3$ treatments were all effective in maintaining a higher pH than the control treatment (P<.01). The antibiotic alone was effective in maintaining a higher pH but to a lesser degree (P<.05). Low levels of rumen lactic acid (<30 mM/liter) were detected on the antibiotic treatment. All treatments were effective in preventing acute acidosis, and none of the animals went off feed. The 4% NaHCO$_3$ treatment resulted in higher acetic acid concentrations than
the treatments containing Thiopeptin (P<.05). Treatments containing Thiopeptin resulted in increases in propionic acid (P<.01) and reduced acetic:propionic ratios compared to the buffer treatments. Differences in total volatile fatty acids or gross energy calculated from volatile fatty acids were not significant between the treatments.
INTRODUCTION

When cattle or sheep are abruptly changed from a roughage to a concentrate diet, lactic acidosis may result causing the animal to go off feed for several days or resulting in death (Ryan, 1964; Vestweber et al., 1974; Allison et al., 1975, Dougherty et al., 1975a; Slyter, 1976). Two useful treatments in the search for additives to prevent lactic acidosis are specific antibiotics to help control rumen microbial populations and buffers to prevent the rapid decrease in rumen pH.

Klatte and Thomas (1967), Prins and Mulder (1969) and Streeter et al. (1974) have suggested that selective antibiotics should be useful in preventing lactic acidosis. When using in vitro methods, Beede and Farlin (1977) found four antibiotics to be effective in decreasing lactate production and three additional antibiotics moderately effective.

Shelton et al. (1969) showed improvement in the feedlot performance of lambs fed high concentrate rations when a 1:1 combination of sodium and potassium carbonates were fed as 2% of the ration. Several other workers (Embry et al., 1968; Brethour and Duitsman, 1972, 1973; Saville et al., 1973; Ralston and Patton, 1976) have shown the use of buffers to be beneficial when used with
high concentrate rations. Herod *et al.* (1977) tested the buffering ability of 23 combinations of compounds *in vitro* and reported carbonates and bicarbonates, in proper combination, were the most promising buffers tested.

The purpose of this experiment was to test the effectiveness of NaHCO$_3$ used at two levels, Thiopeptin, an experimental antibiotic, and a combination of the two in preventing lactic acidosis in sheep abruptly switched from an all roughage diet to one containing 75% cracked wheat.

**EXPERIMENTAL PROCEDURES**

**Animals and Engorgement Feeding**

Four crossbred sheep ranging in weight from 41 to 60 kg were surgically prepared with rumen cannulas and then maintained on a grass hay-alfalfa diet. At the beginning of the experiment, the sheep were placed in wooden crates and given cracked white wheat at a rate of 30 g/kgBW$^{.75}$ divided between two feedings 8 hr apart. This feeding regime was repeated on day two of the experiment. During the first two days, chopped alfalfa was fed as 35% of the diet. On the third day of the experiment, each sheep was fed 50 g/kgBW$^{.75}$ of cracked
wheat divided between three feedings 4 hr apart. Beginning on the third day the amount of chopped alfalfa was reduced to 25% of the total diet. Any feed not consumed within a 30-min period was added through the rumen cannula. The feeding regime begun on day three was continued until each animal had a rumen pH between 4.0 and 4.5. When this pH was maintained for a few hours, the animal was given 50 g of sodium bicarbonate in a one-liter solution, added through the cannula. After 30 min fluid was pumped out through the cannula until no further could be obtained and an equal amount to that removed was added from a donor animal maintained on a grass hay diet. This procedure was carried out to prevent prolonged low pH values in the rumen which might result in damage to the rumen tissues. At the time the donor fluid was placed in the rumen of the acidotic sheep, they were offered grass hay-alfalfa diets. Water and trace mineralized salt were available ad libitum at all times.

Treatments

After the feeding regimes were determined that would result in lactic acidosis for each sheep (controls) and the animals had been back on the grass hay-alfalfa diets for at least 30 days, the sheep were returned to the crates and one of the following treatments was tested. Sodium
bicarbonate added to the diet at the level of 2% of the amount of wheat given (2%), NaHCO₃ at 4% of wheat given (4%), Thiopeptin, the experimental antibiotic⁵, at the level of .25%⁶ of wheat given (A) and a combination of the antibiotic, .25%, plus 2% NaHCO₃ (A + 2%). The exact feeding schedule that produced acidosis was followed during each of these treatments. On days four, five and six, when the control animals had been off feed, the animals on the treatments received wheat which was fed at the level of 50 g/kgBW⁷ divided between three feedings over an 8-hr period. A three week period between each of the treatments was allowed so the animals could re-adapt back to grass hay-alfalfa diets. The sheep were on the various treatments in pairs and the sequence of the four treatments was varied between the two pairs in order to discount any possible effects due to the order in which the treatments were tested.

Sampling

Four rumen samples were taken each day during each treatment. Samples were collected prior to feeding at 700, 1100 and 1500 hr and again at 1900 hours. Samples

⁵Produced by Merck & Co., Inc., Rahway, NJ.
⁶Antibiotic at .25% represents the premix weight. The premix contained 2% activity of the antibiotic.
were obtained through the rumen cannula with the aid of a suction pump. The pH of each sample was determined within one minute of collection. A 5 ml sample was placed in a centrifuge tube containing 1 ml of 25% metaphosphoric acid and refrigerated for at least 4 hr before being centrifuged at 12,500 rpm for 20 minutes. The supernatant fluid was decanted and frozen for future volatile fatty acid and lactic acid analysis. During the control treatment, lactic acid analysis was done on the same day the samples were collected to insure the sheep had developed lactic acidosis.

Analysis

Volatile fatty acids (VFA) and lactic acid were determined using a gas chromatograph following procedures outlined by Carlsson (1973). Gross energies produced from VFA were calculated using values reported by Blaxter (1962).

Statistical

It was desired to analyse means by least squares analysis of variance procedures under a randomized block design, with sheep acting as blocks. Due to the time-sequential nature of the observations on the same treatment, it was possible that each set of observations had
a serial correlation pattern (Neter and Wasserman, 1974). Observations closer together in time were suspected of being more correlated than observations further apart. Since each set of means was comprised of averages of varying numbers of observations, the individual variance of the averages became a function of both the number of observations and the particular value of the serial correlation coefficient between observations. As the exact correlation coefficients were not known, an analysis was done on each set of data for a range of correlation coefficients between zero and one. Treatment differences that were significant for all coefficients looked at, have been reported as being significant.

Results and Discussion

The effect of the treatments on mean pH values recorded each day are shown in table 1. The lowest pH values recorded for each sheep and corresponding lactic acid concentrations are shown in table 2. The 2 and 4% NaHCO₃ and antibiotic + 2% NaHCO₃ treatments were effective in maintaining higher pH levels in the rumen than for controls (P<.01). The antibiotic treatment was also effective in maintaining higher pH values than controls, but to a lesser degree (P<.05).
All control treatments resulted in acute lactic acidosis with typical symptoms. The sheep became lethargic, anorexic and developed moderate diarrhea. Increased respiration rates were noted in all animals when the rumen pH fell below five. Maximum lactic concentrations ranged from 107.1 to 128.7 mM/liter for the four animals on the control treatment. These concentrations are in agreement with values reported by Allison et al. (1964), Uhart and Carroll (1967) and Dunlop (1970). While the increased wheat feeding began on day three, the lowest pH values and maximum lactic acid concentrations occurred during the fourth day. Individual susceptibility to acidosis was evidenced by the fact that the total amount of wheat required on days three and four to induce acidosis ranged from 59.3 to 82.2 g/kgBW$^{75}$.

A rumen pH of ca. five seems critical in that none of the sheep went off feed until the pH was near or below five. Lactic acid concentration increased rapidly when the rumen pH went below 4.8. Dougherty et al. (1975b) showed that this is the pH range where the contractions of the reticulo-rumen decrease in magnitude and frequency and the rumen becomes static. At pH <5 the rumen ingesta changed markedly, becoming yellowish-green in color and very watery in consistency, indicating
that the ingesta had become hypertonic to the plasma with a resulting flow of body fluids into the rumen.

Control animals were off feed for 28 to 40 hr. This represents the time lapse until moderate feed intake was resumed. Because the animals were treated to raise the rumen pH, the time the animals were off feed is probably not indicative of what one might observe under field conditions.

The quantities of wheat used to induce lactic acidosis in this study were less than that used in the study by Beede and Farlin (1977). Levels of lactate observed in their study were less than in the present study despite the feeding of additional wheat. Data on the effects of different types of wheat and the incidence of lactic acidosis are lacking and might provide some insight to dissimilarities observed in various reports. Some of our unpublished data would indicate that time is a factor as well as amount fed. Pellets containing 75% cracked wheat fed at 40 g/kgBW.75, divided equally between two feedings 8 hr apart, caused no problems when fed for a 10 day period but acidosis developed when the 40 g/kgBW.75 was fed at a single feeding on the 11th day. Additional animals developed acidosis when given a single feeding of the pellets at
the 40 g/kgBW\textsuperscript{75} level after being maintained on a grass hay-alfalfa diet.

It was assumed in this study that there was no permanent damage to the animals during the control treatments in which lactic acid was induced or that any tissue damage would not affect subsequent experiments. The damaging effect upon the epithelial surface of the rumen and other parts of the gastro-intestinal tract has been reported by Ahrens (1967), Kay et al. (1969), Thomson (1967) and Dunlop (1967). To substantiate this assumption, six crossbred lambs were induced with acute acidosis by administering a sucrose solution via a stomach tube. The initial dose of sucrose was 15 g/kgBW. The rumen pH was kept below five for varying lengths of time by additional doses of sucrose solution. When the sheep were slaughtered and the tissues examined, no gross tissue damage was found in any animal until the pH had been below five for more than 48 hr. The animals in this experiment were treated so that no animal had a rumen pH below five for more than 28 hr.

While all treatments were effective in preventing acute lactic acidosis from developing, the antibiotic treatment did result in the presence of rumen lactic acid $>20$ mM/liter in three of the four sheep (table 2). No adverse effects were observed and the animals continued
to eat all feed that was offered. Giesecke et al. (1977) reported that lactic acid levels of 10 mM/liter were tolerated by sheep but the present study would indicate higher levels can exist for short periods of time without any deleterious effects.

The effect of the treatments on the formation of volatile fatty acids (VFA) was quite marked. Concentrations of acetic (C$_2$) and propionic (C$_3$) acids are shown in figures 1 and 2, respectively. The addition of either 2 or 4% NaHCO$_3$ resulted in an increase in C$_2$ (P<.01) compared to the control animals. Treatments containing the antibiotic (A or A + 2% NaHCO$_3$) did not show any significant increase. These results are explained by the fact that the C$_2$ levels in the control animals decreased due to the occurrence of acidosis while the treatments containing the antibiotic also showed a decrease in C$_2$ concentrations. The 4% treatment resulted in more C$_2$ (P<.05) that did the A or A + 2% treatments. All treatments showed increases (P<.01) in C$_3$ when compared to control treatments. These differences are largely due to the fact that C$_3$ and butyric (C$_4$) acids decreased rapidly as the lactic acid concentration increased. Similar results were reported by Reid et al. (1957). Treatments containing the antibiotic resulted in
increases ($P < .01$) in $C_3$ when compared to the 2 or 4% NaHCO$_3$ treatments. The combination of A + 2% resulted in still a further increase in $C_3$ ($P < .05$) when compared to the antibiotic used alone. It appears that the treatments containing the antibiotic are altering the microbial population of the rumen towards one capable of producing increased amounts of $C_3$ and lesser amounts of $C_2$ (table 3). Such a shift is recognized as beneficial under feedlot situations. No statistical differences were noted for any treatments concerning the production of butyric acid in the rumen.

Total volatile fatty acids (TVFA) and gross energy (GE) from volatile fatty acids are presented in table 4. While all treatments were higher ($P < .01$) in TVFA when compared to the control treatment, these differences are due to the decrease in VFA in the control treatment as acidosis progressed. No statistical differences for TVFA were observed between the NaHCO$_3$ or antibiotic treatments. This illustrates the point that while the buffer treatments were higher in $C_2$, the antibiotic treatments produced more $C_3$ with the result that total concentrations of VFA were not altered.

There were no statistical differences between any of the buffer or antibiotic treatments for GE content
of TVFA. The fact that C\textsubscript{3} acid is more efficiently produced in the rumen than C\textsubscript{2} is not accounted for in these data; thus while GE data look very similar, the A and A + 2% treatments which result in the production of more C\textsubscript{3} may be more beneficial in a fattening ration than would be indicated by this experiment.

The fact that Thiopeptin was successful in preventing acidosis as well as increasing the proportion of propionic acid produced, certainly would make it a potentially useful feed additive for protection against acidosis. The combination of the antibiotic plus 2% NaHCO\textsubscript{3} resulted in an additive effect and, based on the data presented in this study, would be the preferred treatment in a fattening ration. It gave the beneficial effect of a higher rumen pH plus a higher percentage of propionic acid.
<table>
<thead>
<tr>
<th>Day</th>
<th>Control</th>
<th>Wheat</th>
<th>2% NaHCO&lt;sub&gt;3&lt;/sub&gt;</th>
<th>4% NaHCO&lt;sub&gt;3&lt;/sub&gt;</th>
<th>Wheat</th>
<th>Antibiotic</th>
<th>Wheat</th>
<th>Antibiotic + 2% NaHCO&lt;sub&gt;3&lt;/sub&gt;</th>
<th>Wheat</th>
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<tbody>
<tr>
<td>Day 1</td>
<td>6.12</td>
<td>564</td>
<td>6.16</td>
<td>564</td>
<td>6.27</td>
<td>564</td>
<td>5.70</td>
<td>564</td>
<td>5.78</td>
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<tr>
<td>Day 2</td>
<td>5.81</td>
<td>564</td>
<td>6.05</td>
<td>564</td>
<td>5.95</td>
<td>564</td>
<td>5.78</td>
<td>564</td>
<td>5.88</td>
</tr>
<tr>
<td>Day 3</td>
<td>4.92</td>
<td>940</td>
<td>5.52</td>
<td>940</td>
<td>5.68</td>
<td>940</td>
<td>5.41</td>
<td>940</td>
<td>5.41</td>
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<td>Day 4</td>
<td>4.37</td>
<td>814</td>
<td>5.75</td>
<td>940</td>
<td>5.55</td>
<td>940</td>
<td>5.38</td>
<td>940</td>
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<td>Day 5</td>
<td>4.61</td>
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<td>5.76</td>
<td>940</td>
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<td>940</td>
<td>5.25</td>
<td>940</td>
<td>5.54</td>
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<tr>
<td>Day 6</td>
<td>----</td>
<td>----</td>
<td>5.56</td>
<td>940</td>
<td>5.79</td>
<td>940</td>
<td>5.22</td>
<td>940</td>
<td>5.64</td>
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*The pH values were not obtained after rumen fluid from donor animal was added.*
TABLE 2. LOWEST RUMEN pH VALUES OBTAINED IN EACH TREATMENT AND CORRESPONDING LACTIC ACID CONCENTRATION (mM/liter)

<table>
<thead>
<tr>
<th>Sheep</th>
<th>Control pH</th>
<th>Lactic pH</th>
<th>2% NaHCO₃ pH</th>
<th>Lactic pH</th>
<th>Antibiotic pH</th>
<th>Lactic pH</th>
<th>Antibiotic + 2% NaHCO₃ pH</th>
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<tbody>
<tr>
<td>A</td>
<td>4.52</td>
<td>128.72</td>
<td>5.46</td>
<td>---</td>
<td>5.42</td>
<td>---</td>
<td>5.34</td>
</tr>
<tr>
<td>B</td>
<td>4.34</td>
<td>115.75</td>
<td>5.39</td>
<td>---</td>
<td>5.28</td>
<td>---</td>
<td>5.06</td>
</tr>
<tr>
<td>C</td>
<td>4.25</td>
<td>121.60</td>
<td>5.68</td>
<td>---</td>
<td>5.63</td>
<td>---</td>
<td>5.03</td>
</tr>
<tr>
<td>D</td>
<td>4.36</td>
<td>107.06</td>
<td>5.84</td>
<td>---</td>
<td>5.89</td>
<td>---</td>
<td>5.34</td>
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</tbody>
</table>
TABLE 3. MEAN ACETIC:PROPIONIC ACID RATIOS AS AFFECTED BY TREATMENT

<table>
<thead>
<tr>
<th>Sheep</th>
<th>Control</th>
<th>2% NaHCO₃</th>
<th>4% NaHCO₃</th>
<th>Antibiotic</th>
<th>Antibiotic + 2% NaHCO₃</th>
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</thead>
<tbody>
<tr>
<td>A</td>
<td>2.34</td>
<td>2.09</td>
<td>2.22</td>
<td>.99</td>
<td>1.06</td>
</tr>
<tr>
<td>B</td>
<td>6.76ᵃ</td>
<td>1.97</td>
<td>2.38</td>
<td>1.28</td>
<td>1.06</td>
</tr>
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<td>2.33</td>
<td>1.81</td>
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<td>.90</td>
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<tr>
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<td>2.09</td>
<td>2.07</td>
<td>.86</td>
<td>1.18</td>
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</tbody>
</table>

ᵃ,ᵇ High ratios are due largely to low concentrations of propionic acid during lactic acidosis.
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<th>Control GE</th>
<th>27% NaHCO₃ TVFA</th>
<th>27% NaHCO₃ GE</th>
<th>Antibiotic TVFA</th>
<th>Antibiotic GE</th>
<th>Antibiotic + 27% NaHCO₃ TVFA</th>
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<tr>
<td>B</td>
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<td>4.07</td>
<td>95.71</td>
<td>29.71</td>
<td>104.86</td>
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<td>84.80</td>
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<tr>
<td>C</td>
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<td>65.78</td>
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<td>22.77</td>
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</table>
Figure 1. Acetic acid concentration as affected by treatments.
Figure 2. Propionic acid concentration as affected by treatments.


CHAPTER II

Ruminal Changes During the Onset and Recovery of Induced Lactic Acidosis in Sheep

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Key Words: Acidosis, Lactic Acid, pH Motility, Anorexia, Volatile Fatty Acids.

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\textsuperscript{3}Department of Animal Science.
SUMMARY

Four crossbred wethers equipped with rumen fistulas were used to study changes in rumen pH, motility and the synthesis of volatile fatty acids and lactic acid during the onset and recovery of induced lactic acidosis. Acidosis was induced by the addition of sucrose (15 g/kg BW) in 700 ml of water through the fistula. Mean maximum lactic acid concentration was near or in excess of 100 mM/liter of rumen fluid during the first 48 hr post-inducement. The minimum pH values observed for each sheep were 4.53, 4.13, 4.29 and 4.48. Propionic and butyric acids were absent from rumen samples by 14 hr post-inducement. Acetic acid concentrations decreased to <5 mM/liter during the first 24 hr following the addition of the sugar. Rumen motility decreased in frequency and amplitude during the first 4 hr. Normal rumen motility was observed in one sheep by 52 hr but not in another, until 84 hr post-inducement. Two of the four sheep were "off feed" by 4 hr post-inducement while the remaining two did not become anorexic until 7 hr. Animals began to consume small amounts of alfalfa when rumen pH values increased to >5.0 but consumption similar to pre-inducement intake was not observed until
the rumen pH was >6.0 and lactic acid could no longer be detected in the rumen fluid.
INTRODUCTION

Over a period of years many workers have presented excellent research with respect to lactic acidosis and the effects the disorder has on the reticulo-rumen (Hun-gate et al., 1952; Dunlop and Hammond, 1965; Allison et al., 1964; Huber, 1971; Ryan, 1964;Uhart and Carroll, 1967). Changes that occur with respect to rumen pH, volatile fatty acids, formation of lactic acid and rumen motility as well as many others have been documented and reviewed (Elam, 1976; Huber, 1976; Slyter, 1976; Brent, 1976).

The purpose of the present research was to induce lactic acidosis in sheep equipped with rumen fistulas and monitor rumen changes such as motility, pH, volatile fatty acids and lactic acid not only in the developmental stages of the disorder but also during the recovery period. Many of the studies involving the inducement of acidosis have resulted in the death of the animals and as a result, data on recovery periods are lacking. The objectives were to observe the conditions existing at the time the sheep went "off feed" as well as those when they began to consume feed again. We hoped to gain a better understanding of the conditions existing when rumen stasis occurs as well as those when rumen motility resumes and thus to gain some insight into how the
conditions in the rumen, the motility patterns of the rumen, and the desire to consume feed might be related.

MATERIALS AND METHODS

Animals

Four crossbred wethers (A, B, C, D) weighing 48 to 55 kg were prepared with rumen fistulas following procedures outlined by McCann et al. (1973). Animals were given a minimum of 60 days recovery time before rumen motility studies were conducted. The sheep were maintained on grass hay-alfalfa diets prior to the studies. Water and trace mineralized salt were available ad libitum at all times.

Inducement of Acidosis

The sheep were given sucrose at the level of 15 g/kg BW in a 700 ml solution of water warmed to 39 C. The sucrose solution was placed in the rumen through the fistula at 0800 immediately following rumen motility tracings and rumen pH determination.

Rumen Motility Recordings

The sheep were placed in separate metabolism crates three days prior to any recordings. Pre-inducement
recordings were carried out prior to the morning feedings and at 4, 8 and 12 hr post-feeding on two consecutive days. Diets consisted of chopped alfalfa offered once daily, and the animals were allowed to eat ad libitum for a 4 hr period. Post-inducement recordings were carried out prior to the addition of sucrose and 2, 4, 7, 10, 14, 24, 28, 32, 36, 48, 52, 60, 72, 76, 84, 96 and 108 hr post-inducement.

The recording apparatus consisted of a thick wall balloon (finger cot) mounted over a one hole No. 2 rubber stopper which was connected to a 61 cm section of semi-rigid .64 cm O.D. aluminum tubing. A small catheter was run down through the tubing, flared and sealed with wax to prevent any air leakage from the balloon once it was inflated. The balloon was securely fastened to the stopper by wrapping with suture material and covering with waterproof tape. The catheter from the balloon was connected to a pressure transducer with the aid of Leur-Lock fittings. A second catheter tube led from the dome of the pressure transducer to a 50 ml syringe with a Leur-Lock holder. Thirty ml of air were injected into the balloon. Back pressure on the syringe was eliminated with

4Statham P 23 transducer, Statham Industries, Inc., Oxnard, CA 93030.
the aid of a three way stopcock. A Gilson Polygraph\textsuperscript{5} connected to the electrical transducer was used to record the movements of the reticulo-rumen.

Recordings were conducted by inserting the balloon apparatus through the cannula and then inflating the balloon with the aid of the syringe. A one hole No. 5 rubber stopper which had been moved down over the aluminum tubing acted to hold the device in place by fitting tightly into the body tube of the rumen cannula (figure 1). Motility in different sections of the reticulo-rumen were recorded by bending the aluminum tubing towards the desired area. Further adjustment was possible by sliding the aluminum tubing in or out through the rubber stopper held in the tube of the cannula.

Rumen Samples

A sample of rumen contents was removed through the fistula with the aid of a suction pump prior to every recording. The pH of the sample was taken within one min of removal using a Orion digital pH meter.

\textsuperscript{5}Gilson polygraph, model M5P, Gilson Medical Electronics, Inc. Middletown, WI.
Analysis

Lactic and volatile fatty acid (VFA) analyses on rumen samples were done by gas chromatography using a modified procedure outlined by Carlsson (1973). Preparation of samples for gas chromatography involved taking a 5 ml sample of rumen fluid and placing it in a centrifuge tube containing 1 ml of 25% meta-phosphoric acid. These tubes were allowed to stand under refrigeration for a minimum of 4 hr before being centrifuged at 12,500 rpm for 20 min. Supernatants were removed and used for lactic and VFA analysis.

RESULTS

Acute acidosis developed in each of the four sheep (table 1) with typical symptoms being observed. All animals went off feed, became lethargic, had increased respiration rates and developed moderate diarrhea. Rumen fluid became very watery in consistency when the pH dropped below 5.0.

Animals A and B were anorexic by 4 hr post-inducement while animals C and D did not refuse feed until 7 hr after the addition of the sucrose. Lactic acid was detected in the rumen of each animal at the time the animals refused feed, with concentrations ranging between 20.3 and 54.1
mM/liter and corresponding pH values of 5.50 and 4.57, respectively. Maximum rumen concentrations of lactic acid were near or in excess of 100 mM/liter in each of the four sheep, but considerable variation was noted in the time when the maximum concentration occurred. Sheep B had its maximum concentration at 10 hr post-inducement while sheep C did not reach the maximum until 48 hr. Sheep C appeared to have begun recovery at 32 hr and had begun to eat alfalfa hay which was available to it. The feed consumed seemed to re-induce acidosis; the animal became anorexic, lethargic and by 48 hr had a rumen lactic acid concentration of nearly twice that detected in the 32-hr sample. Of the four sheep, this animal required the longest period to recover and consume pre-inducement quantities of feed. Complete anorexia continued in all animals until the rumen pH rose above five. Lactic acid concentrations in the rumen samples at the time first feed was consumed were between 55 and 70 mM/liter. Intake at this point was minimal and animals would not stand to eat for more than 15 min. at any one time. Feed intake did not approach pre-inducement levels until rumen pH values were >6 and lactic acid could no longer be detected in the rumen. For sheep D, these conditions were present by 48 hr while for sheep C, normal feed intake did not occur until 96 hr post-inducement.
The effects of induced acidosis on rumen motility are shown in figure 2. Nearly all motility in the reticulo-rumen was absent by 4 hr following the addition of the sucrose through the fistula. Considerable variation in motility was noted on the second and third days post-inducement among the four sheep. Sheep D was the only animal to have rumen motility at 28 hr post-inducement although the contractions were below pre-inducement values in both frequency and amplitude. Sheep C had motility present at 52 hr post-inducement but recordings showed the motility to be reduced in amplitude compared to pre-inducement with slight reductions in frequency being observed.

The relationship of individual volatile fatty acid (VFA) to the inducement of acidosis is shown in figure 4. Both C₃ and C₄ acids were reduced to concentrations of <2 mM/liter by 10 hr following the addition of sucrose to the rumen. By 14 hr neither acid was detected in the rumen fluid of any of the four sheep. With the exception of sheep D, the C₃ and C₄ acids were not detected again until 48 hr or later post-inducement. Sheep C, which was off feed the longest, did not have C₃ acid present in the rumen for a period of nearly 48 hr. Butyric acid in sheep C was absent for a period of 58 hr. Sheep D, which was off feed the shortest period of time, did not
have detectable levels of $C_3$ and $C_4$ acids for 12 and 20 hr, respectively. Acetic acid concentrations in the acidotic sheep followed similar patterns. Concentrations fell to 10 mM/liter or less by 14 hr post-inducement. While $C_2$ concentrations never reached zero, all four sheep did reach concentrations of $<5$ mM/liter during the first 24 hr after inducement.

Sheep D, the first to recover, had $C_2$ concentrations of $>20$ mM/liter by 36 hr while sheep C did not have similar values until 84 hr post-inducement. At the time each animal had returned to near pre-inducement feed intake levels, $C_2$, $C_3$ and $C_4$ acid concentrations were $>30$, $>11$ and $>5$ mM/liter, respectively.

**DISCUSSION**

The affect of added sucrose on the changes occurring in the reticulo-rumen of the experimental sheep was as expected. Previous experiments with graded levels of sucrose led us to the 15 g/kg BW level used. We wished to obtain pH values between 4 and 4.5 and lactic acid concentrations near 100 mM/liter in the rumen fluid. Other work at our station as well as that of other researchers made us well aware of the high risk of death to the animal if the rumen pH fell below 4.0. This is not to imply that the rumen pH is totally responsible
for the well being of the animal experiencing lactic acidosis.

Our lactic acid concentrations and pH values during the first 4 hr post-inducement (20.26, 5.50; 21.17, 5.17; 46.26, 5.14 and 32.16, 5.28) disagree with those of Briggs et al. (1957) who reported that lactic acid levels of >20 mM/liter were always associated with a rumen pH of below 5.0. We believe that the method of analysis used in this study is possibly more sensitive, resulting in higher levels of lactic acid earlier following inducement of acidosis.

The effect of lactic acid concentration on the animal going off feed is unclear based upon the results of this study. Sheep A, C and D were eating when rumen lactic acid concentrations were 11.49, 48.3 and 32.2 mM/liter, respectively. All three animals were anorexic 3 hr later although the lactic acid concentration was <5 mM/liter greater for two of the animals than when they were eating 3 hr earlier. Length of time following the appearance of lactic acid may be a factor but samples were not taken frequently enough during the first 8 hr to test this. Bueno (1975) infused DL-lactic acid into the duodenum of sheep and reduced the dry matter intake by 50%. This would imply that rumen lactic acid concentration may not be the only factor affecting feed intake.
The role of lactic acid on rumen motility remains unanswered. Juhász and Szegedi (1968) lowered the pH of the rumen with lactic acid below 5.0 but noted stasis of the rumen did not occur for 2 to 4 hr. Our work showed there was a decrease in the frequency and amplitude of contractions at 2 hr post-inducement, prior to the formation of detectable levels of lactic acid in the rumen fluid (figure 3). Although sheep C had a concentration of rumen lactic acid >100 mM/liter (52 hr), contractions were very evident (figure 3). These strong contractions, despite the high concentrations of lactic acid, cause one to further question the role of lactic acid in inhibiting rumen motility. Dougherty et al. (1975) showed strong rumen contractions in two of four sheep with pH values of 4.4 and 4.5, 72 hr after inducing acidosis in sheep using corn and oats. Corresponding lactic acid concentrations were not given for the study.

The near total absence of volatile fatty acids in the rumen fluid following the inducement of acidosis agrees with the work of Ryan (1964) for similar pH values and lactic acid concentrations. The amounts and percentages of VFA's present when pre-inducement feed intake levels were reached are in agreement with values reported by Uhart and Carroll (1967) for VFA's present at the time steers resumed eating after being off feed
when suddenly switched to a high grain diet.

Initial loss of rumen motility and anorexia seem to be closely related at least with respect to time. Similar results were reported by Lane (1968). The sheep in the present experiment developed motility with reduced frequency and amplitude while the animals were anorexic but pre-inducement feed intake and pre-inducement motility tended to occur at about the same time in individual animals. If the strong contractions of the reticulo-rumen induced normal appetite or if increased feed intake induced contractions similar to those pre-inducement remains to be answered.

One should consider other factors which occurred at the time normal levels of feed intake were being consumed following acidosis. Lactic acid in the rumen had declined to undetectable levels. The volatile fatty acid concentrations had risen to $>50 \text{ mM/liter}$ and the rumen pH was $>6$. All of these factors appear to be inter-related in the recovery process that occurs in animals which have developed acidosis. Additional factors such as histamine (Dougherty, 1942), endotoxins from bacteria (Mullinax et al., 1966) and secretin (Bruce and Huber, 1973) may very well be involved but were not studied in these experiments.
Certainly there are limitations which must be considered in a study of this type. Only four animals were used in the experiment. Because of this fact and because of the nature of the study, no statistical analysis was attempted.

The procedures for obtaining rumen motility data used in this study could lend themselves for use with any rumen fistulated animals with minor modifications. The use of a balloon apparatus for measuring rumen motility does not give rise to as continuous or as high quality recording as those using radiotelemetric methods. It does have the advantage of requiring much less time and expense in preparation. The most difficult problem in the procedure was the placement of the balloon into the rumen for each reading since exact positioning in the same location required some adjustments for proper recording. This became easier as the operator became more familiar with the animals and apparatus. Differences in activities (eating, resting or ruminating) can account for some of the differences observed in recordings in the same location at different times (Church, 1975).

While we assembled more than one balloon apparatus, we noted some variation between recordings when different units were used. We were fortunate in that the device proved to be very durable, allowing the same apparatus to collect recordings on all four sheep.
The literature available would indicate that many factors may be involved with rumen motility changes in acidosis and the present study was not designed to study all of those factors. We did hope to add to the present knowledge with respect to what changes occur as the animals recover from acute lactic acidosis and how these changes may affect the behavior of the animals. By learning more about what occurs in the recovery process, the day may come when the "off feed" period might be shortened considerably.
TABLE 1. EFFECT OF ADDED SUCROSE ON RUMEN pH AND LACTIC ACID CONCENTRATION (mM/liter)

<table>
<thead>
<tr>
<th>Time (hr)</th>
<th>Sheep A pH</th>
<th>Sheep A lactic</th>
<th>Sheep B pH</th>
<th>Sheep B lactic</th>
<th>Sheep C pH</th>
<th>Sheep C lactic</th>
<th>Sheep D pH</th>
<th>Sheep D lactic</th>
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<td>96.56</td>
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<td>6.72</td>
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<td>6.58</td>
<td>---</td>
<td>6.37</td>
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</tr>
</tbody>
</table>

\(^a\)Animal would no longer respond to feed.

\(^b\)Animal began eating small amounts of feed.

\(^c\)Animal consuming full feed.
Figure 1. Cross section through body cavity showing placement of balloon apparatus in the ventral sac of the rumen.
2a. Caudal ventral blind sac motility in Sheep B prior to and following the inducement of acidosis.

2b. Reticulum motility recordings in Sheep B prior to and following the inducement of acidosis.
3a. Ventral sac motility in Sheep C prior to and following inducement of acidosis.

3b. Reticulum motility recordings in Sheep C prior to and following the inducement of acidosis.
Figure 4. Volatile fatty acid concentrations in the rumen fluid of sheep prior to and following the induction of acidosis.
LITERATURE CITED


APPENDIX
TABLE 1. EFFECT OF SUCROSE ON RUMEN pH, LACTIC ACID (mM/liter), FEED INTAKE AND RUMEN MOTILITY

<table>
<thead>
<tr>
<th>Time (hr)</th>
<th>Sheep A</th>
<th>Sheep B</th>
<th>Sheep C</th>
<th>Sheep D</th>
</tr>
</thead>
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<td>pH-7.20, lactic-0</td>
<td>pH-6.77, lactic-0</td>
<td>pH-6.70, lactic-0</td>
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<tr>
<td></td>
<td>gave 818 g sucrose, motility-normal</td>
<td>gave 717 g sucrose, motility-normal</td>
<td>gave 735 g sucrose, motility-normal</td>
<td>gave 800 g sucrose, motility-normal</td>
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<td>pH-5.71, lactic-11.49</td>
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<td>pH-5.45, lactic-0</td>
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<tr>
<td></td>
<td>motility-reduced frequency and amplitude</td>
<td>motility reduced frequency and amplitude</td>
<td>motility-reduced frequency and amplitude</td>
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<td>eating, anorexia at 7 hr, motility-near stasis</td>
<td>eating, anorexia at 7 hr, motility-reduced frequency and amplitude</td>
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<td>28</td>
<td>pH-4.54, lactic-84.30</td>
<td>pH-4.41, lactic-54.19</td>
<td>pH-4.55, lactic-81.81</td>
<td>pH-4.67, lactic-115.63</td>
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<td>anorexia, motility-stasis</td>
<td>anorexia, motility-stasis</td>
<td>anorexia, motility-stasis</td>
<td>anorexia, motility-reduced frequency and amplitude</td>
</tr>
<tr>
<td>48</td>
<td>pH-5.34, lactic-64.21</td>
<td>pH-5.56, lactic-54.50</td>
<td>pH-4.46, lactic-156.79</td>
<td>pH-6.10, lactic-0</td>
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<tr>
<td></td>
<td>ate small amount, motility-reduced frequency</td>
<td>ate small amount, motility-stasis</td>
<td>anorexia, motility-reduced amplitude</td>
<td>began eating at 56 hr, motility-reduced amplitude</td>
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<tr>
<td>52</td>
<td>pH-5.52, lactic-68.28</td>
<td>pH-5.05, lactic-60.11</td>
<td>pH-4.71, lactic-103.08</td>
<td>pH-6.20, lactic-0</td>
</tr>
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<td></td>
<td>anorexia, motility-reduced frequency</td>
<td>anorexia, motility-stasis</td>
<td>anorexia, motility-reduced amplitude</td>
<td>full feed, motility-near normal</td>
</tr>
<tr>
<td>60</td>
<td>pH-6.50, lactic-1.83</td>
<td>pH-6.37, lactic-28.39</td>
<td>pH-4.68, lactic-106.39</td>
<td>pH-6.09, lactic-0</td>
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<tr>
<td></td>
<td>eating moderate amount, motility-reduced frequency</td>
<td>eating moderate amount, motility-reduced frequency</td>
<td>anorexia, motility-reduced amplitude</td>
<td>full feed, motility-normal</td>
</tr>
<tr>
<td>76</td>
<td>pH-6.95, lactic-0</td>
<td>pH-7.16, lactic-0</td>
<td>pH-5.17, lactic-58.64</td>
<td>pH-6.42, lactic-0</td>
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<tr>
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<td>full feed, motility-normal</td>
<td>full feed, motility-normal</td>
<td>ate small amount, motility-reduced frequency</td>
<td>full feed, motility-normal</td>
</tr>
<tr>
<td>84</td>
<td>pH-6.49, lactic-0</td>
<td>pH-6.78, lactic-0</td>
<td>pH-5.88, lactic-0</td>
<td>pH-6.56, lactic-0</td>
</tr>
<tr>
<td></td>
<td>full feed, motility-normal</td>
<td>full feed, motility-normal</td>
<td>eating small amount, full feed at 96 hr, motility-near normal</td>
<td>full feed, motility-normal</td>
</tr>
</tbody>
</table>
Figure 1. Caudal ventral blind sac motility in sheep A prior to and following inducement of acidosis. Note normal amplitude at 60 hr but reduced frequency.
Figure 2. Reticulum motility recordings in sheep D prior to and following inducement of acidosis. Note the normal frequency of contractions at 48 hr but reduced amplitude.
Figure 3. Ventral sac motility in sheep A prior to and following the inducement of acidosis.
Figure 4. Ventral sac motility in sheep B prior to and following the inducement of acidosis.
Figure 5. Caudal ventral blind sac in sheep C prior to and following the inducement of acidosis.