MILK PRODUCTION RESPONSE TO FEEDING RUMENSIN

R. K. McGuffey

McGuffey Dairy Consulting, Indianapolis, IN

INTRODUCTION

Rumensin (monensin sodium; Elanco Animal Health) was first approved in 1975 by FDA for use in feedlot cattle. The claim, “For Improved Feed Efficiency” quickly led to widespread use of Rumensin throughout the feedlot industry. Once in the marketplace, cattlemen, nutritionists and veterinarians observed “ancillary benefits” of reduced death losses due to coccidiosis and digestive diseases such as bloat and sudden death. Additional claims for Rumensin followed including:

• “For improved feed efficiency” in mature reproducing beef cattle;
• “For increased rate of gain” in growing cattle on pasture or in dry lot (stocker, feeder calves and beef and dairy replacement heifers)
• “For prevention and control of coccidiosis caused by Eimeria spp” in feedlot cattle, mature reproducing cattle, growing cattle on pasture or in dry lot, and calves (excluding veal calves) and prevention in goats maintained in confinement.

Rumensin gained approval from FDA in November of 2004 for use in dairy cattle. The claim, “For increased milk production efficiency” applied to dairy cattle (lactating and dry) fed a total mixed ration containing 11 to 22 grams Rumensin per ton on a dry matter basis. In December of 2005 the claim and dosage rate were expanded to include component-feeding systems. Dosage rates in a minimum of one pound of feed allowed a range from 185 to 660 mg Rumensin per head per day to lactating cows or 115 to 410 mg/head/day Rumensin to dry cows.

Rumensin delivers two types of benefits to cattle—one allowed in the official claim and the aforementioned ancillary benefits. Both provide significant economic returns to producers. The objective of this paper is to discuss the mechanism of action of Rumensin in the gastrointestinal tract to provide a background and explanation of benefits derived from use of Rumensin. While the emphasis will be on dairy cattle, data from beef cattle will be used where potential application exists for dairy cattle.

THE MODE OF ACTION OF RUMENSIN

Rumensin belongs to a chemical class of compounds called ionophores. Biological actions of Rumensin are directed at the microbial inhabitants of the gastrointestinal tract of
ruminants. These actions select for certain bacteria, generally Gram-negative, at the expense of others. In general, metabolism of the selected microorganisms favors the host animal. Resistance of Gram-negative bacteria to ionophores appears to be conferred by the presence of an outer membrane which is impermeable to large molecules. Changes in metabolism of cattle fed Rumensin are a result in the changes in the pattern of end-products of digestion and not a systemic effect.

Ionophores share a common mode of action that begins with attachment to the cell membrane of bacteria and protozoa (Fitzgerald and Mansfield, 1973, Chow et al., 1994). Attachment initiates a cascade of cations across cell membranes, exchanging a monovalent cation for a proton (Rumensin, laidlomycin propionate, salinomycin, lasalocid) or a divalent cation for two protons (lasalocid) (Pressman, 1976). Cation gradients and affinities drive the resulting primary and secondary ion exchanges at the rate of >1000 events per second (Pressman, 1976). As the greatest concentration gradient is the cytoplasmic K+ compared to extracellular K+, this drives the exchange of an intracellular K+ for an extracellular proton. Having deposited the complexed cation into the extracellular (rumen) fluid, the ionophore then re-complexes an extracellular cation, and reverses the process (Russell and Strobel, 1989).

The ionophore-induced reactions of the cell culminate in reduced intracellular K+ concentration, lower pH and greater intracellular Na+ concentrations (Russell and Strobel, 1989). Bacteria, typically Gram-positive, are forced to utilize cellular transport systems to dissipate the intracellular H+ and Na+. For Gram-positive bacteria, the proton-ATPase and sodium-ATPase pumps are elicited to expel H+ at the expense of one ATP per proton. Reduced energy reserves lower protein synthesis and cell division. Ultimately, the organism is incapable of sustaining a rate of cell division sufficient to maintain normal metabolic significance (Russell and Strobel, 1989).

RUMINAL METABOLISM

Optimizing ruminal fermentation through enhanced organic matter digestion, primarily carbohydrates, is a primary goal in feeding cattle for productive purposes. For fattening cattle, optimizing starch digestion is foremost. Optimizing fiber digestion is the favored strategy of lactating and growing cattle. These strategies are directed at controlled fermentation of starch, reduced methane production, reduced degradation of plant protein, and/or reduced biohydrogenation of unsaturated fatty acids (Van Nevel and Demeyer). These processes are not mutually exclusive. Rumensin affects each of these processes of ruminal fermentation.

Carbohydrate Metabolism

Rumensin’s effect on carbohydrate fermentation is an increase in the production of propionic acid. Enhanced rumen propionate production is partially explained by the
replacement of Gram-positive bacteria with Gram-negative bac-teria (Dawson and Boling, 1984; Russell, 1987). However, a significant portion of the propionate effect results from the ionophore-mediated alterations in metabolism occurring in the Gram-negative population (Bergen and Bates, 1984, Stahl et al., 1988; Morehead and Dawson, 1992).

Anaerobic bacteria in the rumen derive energy from substrate oxidation by the transfer of electrons (and hydrogen) to acceptors other than oxygen primarily volatile fatty acids (VFA) and methane. As propionate production in the rumen increases, fermentation balance requires a decrease in methane production (Chalupa, 1977; Hungate, 1966). Up to 12% of the gross energy of feeds can be lost as eructated methane. Diverting hydrogen to other end-products, e.g. propionate, captures more digestible energy from fermented organic matter, resulting in more efficient use of feed energy and lessens the contribution of atmospheric methane from cattle.

Richardson et al., (1978) reported a 36 percent and 54 percent increase in the molar proportion of ruminal propionate in pasture- and concentrate-fed cattle, respectively, when supplemented with 200 mg/d of Rumensin. Richardson et al., (1976) fed Rumensin to cattle for 148 days and obtained rumen samples by stomach tube bi-weekly or at termination of the study. The relative molar concentration of propionate in total VFA increased consistently from 0.32 in the controls to 0.41 and 0.44 for animals fed 100 and 500 mg Rumensin per day, respectively.

Propionate production rate was increased by 49% and 76% in steers fed forage and concentrate rations, respectively (Van Maanan et al., 1978). Similarly, Rumensin fed with hay and concentrate diet increased propionate production rate by 44% (Prange et al.,1978) and with corn silage and concentrate diet by 65% (Rogers and Davis, 1982). Improved energetics of rumen fermentation caused by Rumensin is illustrated by the work of Rogers and Davis. Steers were fed a basal diet of 50 percent corn silage and 50% concentrate either with or without Rumensin (33 mg/kg dry matter). Dry matter intake and production rates of acetic, propionic and butyric acids were measured. Feeding of Rumensin increased the daily ruminal production of acetic, propionic and total acids per kg of dry matter consumed by 29, 64 and 35 percent, respectively. Total VFA energy produced in the rumen per kg of dry matter consumed was increased from 0.852 Mcal/kg dry matter for control steers to 1.137 Mcal/kg dry matter for steers fed Rumensin, a 33 percent increase in ruminal digestible energy.

Rumensin has little or no effect on methanogens; rather methanogenesis is reduced by decreased availability of hydrogen and formate, the ruminal substrates for methane production. Many bacteria that produce these substrates are sensitive to Rumensin. Van Nevel and Demeyer (1977) provided additional evidence for this mechanism when in the presence of Rumensin, methane production by mixed cultures of rumen microbes was increased by addition of hydrogen gas.

Feeding of Rumensin changes the site of digestion of dietary carbohydrate fractions. Ruminal digestion of starch may be decreased but post-ruminally, starch digestion is increased to the extent that total tract digestibility is unchanged (Muntifering et al., 1981; Funk et al., 1986). Fiber digestion is largely unaffected by Rumensin (Allen and Harrison,
Increased numbers of ionophore-resistant fibrolytic bacteria, such as Fibrobacter succinogenes, may offset reduced numbers of ionophore-sensitive ruminococci. Additionally, a longer rumen retention time caused by Rumensin may contribute to maintenance of normal fiber digestion (Lemenager et al., 1978).

Feeding Rumensin to lactating cows decreased ruminal digestion of organic matter, acid detergent fiber and starch (Ali Haimoud et al., 1995). Total tract digestibility of these components was not affected. Plazier et al., (1999) reported that apparent digestibility of gross energy was increased in early post-partum dairy cows that had received a Rumensin controlled release capsule three weeks prior to calving. The improvement in energy digestibility was a consequence of improved fiber digestibility.

**Nitrogen Metabolism**

Russell et al., (1988) identified bacteria in the rumen that use amino acids as their sole source of energy. These bacteria contain enzyme systems with high specific activities for ammonia production. These amino acid fermenters are sensitive to Rumensin (Chen and Russell, 1991). When nonlactating cows were fed hay supplemented with soybean meal, there was a 30 percent decrease in rumen NH3 concentration and a similar reduction on the specific activity of ammonia production. Rumensin caused an approximate ten-fold decrease in the most probable number of bacteria in the rumen that fermented amino acids and peptides as an energy source (Yang and Russell, 1993).

A reduction in rumen NH3 concentration was observed frequently in early studies with Rumensin. Poos et al., (1979) reported a decrease in the proportion of bacterial protein and an increase in plant protein entering the small intestines when cattle were fed Rumensin with either brewers dried grains or urea as the primary nitrogen source. Using the Rumensin controlled release capsule, Plazier et al., (2000) reported decrease in rumen ammonia and an improvement in nitrogen digestibility that resulted in an improved nitrogen balance of dairy cattle immediately post partum. Spears (1990) reported that Rumensin improved nitrogen digestibility in cattle. More research on the effects of Rumensin on nitrogen metabolism is needed for lactating dairy cattle.

**Fat Metabolism**

Rumensin inhibits hydrolysis of triglycerides and biohydrogenation of unsaturated fatty acids in the rumen (Van Nevel and Demeyer, 1995). Biohydrogenation of linoleic acid was reduced and production of trans-octadecenoic acid isomers was increased by Rumensin in continuous ruminal fermenters (Fellner et al., 1997). Jenkins et al., reported that Rumensin and soybean oil produced higher concentrations of trans-10 C18:1 in ruminal contents when barley compared to corn was the starch source. This finding suggested that higher rates of ruminal degradation of starch (as with barley) in the presence of Rumensin and soybean oil resulted in more incomplete biohydrogenation of linoleic acid. Under this scenario, Rumen-
sin would increase the risk of milk fat depression. Grinaari et al., (1998) proposed that milk fat depression in cattle required two ruminal the presence of dietary linoleic acid and an altered ruminal environment.

FEEDING OF RUMENSIN TO DAIRY COWS—
POTENTIAL ANCILLARY BENEFITS

The transition period is the most critical time frame of the lactation cycle for the occurrence of health conditions that compromise the success of lactation. Health conditions associated with calving and the nutritional and metabolic changes associated with the initiation of lactation are largely a failure of the nutrition and management of the cow. Occurrence of one health condition increases the risk of occurrence of other health conditions. Most health conditions that occur during the transition period with the exception of twins or abnormal presentation of the calf at delivery can be prevented with sound nutrition, timely feeding, adequate facilities and observation. Rumensin can play a critical role in reducing the risk of most health conditions associated with calving.

Rumensin increases the absorption at the rumen of divalent cations, Ca, Mg, Cu, Mn, Se and Zn. The trace minerals are positioned in many proteins associated with the immune system. A functional immune system decreases the occurrence of mastitis and retained fetal membranes following calving. Heuer et al., (2001) reported a reduced incidence of mastitis at calving in first calf heifers fed Rumensin prior to calving. However, similar findings were not observed in the nine-trial North American Rumensin dose-titration study.

A reduction in the occurrence and severity of ketosis has been reported in numerous studies with Rumensin premix or the Rumensin controlled release capsule (CRC). Sauer et al., (1989) fed Rumensin at 10 and 20 mg/kg DM to prepartum cows and observed a reduction in subclinical ketosis as measured by a reduction in the concentration of beta hydroxy butyrate in blood. Duffield et al., (1998) reported reductions in subclinical and clinical ketosis and other energy-associated conditions in the transition cow by administering a CRC to cows approximately 21 days prior to calving. Ketosis was reduced in cows fed 22 g/ton of Rumensin in the nine-trial North American Rumensin dose-titration study (Elanco, unpublished results). In some of these studies, occurrence of displaced abomasums often associated with ketosis was reduced in cows fed Rumensin.

Digestive Disorders

Lactic acid is another end-product of ruminal fermentation and, is approximately ten times stronger acid than any of VFA. The rumen contains bacteria that are lactate producers and others that are lactate users. The major lactate producing bacteria (Streptococcus and Lactobacillus) grow rapidly when diets rich in starch are fed. Both species, however, are sensitive to ionophores and fail to grow in its presence. Lactate utilizing bacteria, e.g. Megasphaera and Selenomonas, are not sensitive to Rumensin.
Non-infectious lameness is generally attributed to acute bouts of ruminal acidosis, primarily caused by overproduction of lactic acid. Sources of rapidly fermentable starch coupled with inadequate fiber intake are diets most likely responsible for setting off the cascade of events that lead to non-infectious lameness. The nutritional insult is most likely to occur in early lactation but the condition may not manifest itself for weeks or months. Heat stress may exacerbate the nutritional insult.

Feeding of Rumensin reduced incidence and duration of non-infectious lameness in European studies (Heuer et al., 2001) and the nine-trial North America study (Elanco, unpublished). The reduction in lameness may be attributed to a combination of two actions of Rumensin that affect lactic acid production. The first action is a reduction in the ruminal production of lactic acid by Streptococcus bovis.

The second mechanism relates to eating behavior of cattle consuming diets high in fermentable carbohydrates. In beef cattle, increased variation in dry matter intake occurs each time that the level of concentrate in the diet is increased (Burrin et al., 1988). Subacute acidosis increases day to day variation in DMI and decreases total DMI (Stock et al., 1990). Over-eating one day to compensate for the previous day’s under-eating can set the stage for acute acidosis.

Rumensin changes the eating behavior of beef cattle on high concentrate diets. Stock et al., (1995) evaluated Rumensin in individually fed cattle and observed reduced variance in feed intake especially when concentrate was 85 percent or more of the diet. In a similar study, steers adapted to an 85 percent concentrate diet containing Rumensin consumed smaller but more frequent meals than control steers. Rumensin tended to reduce the largest meal of the day (4.5 kg vs 5.4 kg), but total daily DMI was not different (Fanning et al., 1999). These changes in eating behavior presumably limit the risk of subacute acidosis.

**FEEDING OF RUMENSIN TO DAIRY COWS—MILK PRODUCTION RESPONSE**

Elanco Animal Health received approval of Rumensin for dairy cattle based upon the results of a study conducted at 9 sites in the United States (6 sites) and Canada (3 sites). A common protocol was used at each site with Holstein cattle both heifers and cows. Cattle were assigned to one of four levels of Rumensin (0, 7, 15 and 22 grams per ton on a dry matter basis) beginning approximately 21 days prior to calving. Cattle received the assigned level of Rumensin continuously through the following lactation, dry period and for up to 10 days into the next lactation. Milk and feed intake were determined daily, milk composition weekly and body weight and body condition score at scheduled times throughout the study. In addition, all reproductive events, health conditions including mastitis cases were monitored and treated according to an attending veterinarian’s care.

Diets were based on Nutrient Requirements for Dairy Cattle, 1989. Feedstuffs common to the trial location were used. Total mixed rations were fed at all sites throughout the duration of the study. Composition of each TMR was recorded and allowed estimation of
intakes of dry matter, net energy, protein and macrominerals.

Results for each site were pooled into the previously mentioned 9 Trial North American efficacy study. Pooling did not consider evaluation of individual trial results after the statistical term in the model, Trial X Treatment, was shown to be non significant. Following submission to FDA, my colleagues at Elanco Animal Health, Mr. Howard Green, Dr. John Wilkinson, Dr David McClary, and I began to evaluate the data on an individual trial basis.

The concept of quadrant analysis (Figure 1) was applied to the 9 Trial North American efficacy study after discovering that milk fat percent behaved significantly different between sites. Quadrant analysis is a method of examining the importance of two traits on an outcome. The two traits may respond independently but taken together indicate a degree of the desired state.

**Figure 1.** Numbering of Quadrants.

![Figure 1: Numbering of Quadrants](image)

Differences in response of milk fat percent to Rumensin between trial sites suggested that dietary differences might cause a differential response to Rumensin. Performance data from the individual trials in the 9 Trial North American efficacy study was evaluated in an attempt to identify additional differences in response to Rumensin. Because of major differences in feeds as a result of the geographical diversity of sites, a hypothesis was developed that stated chemical measures of the diet and behavior of the diet in the rumen affected response of lactating cows to Rumensin.

**The Data**

Milk yield, fat and protein percents and yields, dry matter intake and body weight
averages for 44 weeks of lactation were calculated for each cow completing at least 100 days during the first lactation of the 9 Trial North American efficacy study (Symanowski et al., 1999). Average trial site means for milk, fat percent and yield, protein percent and yield and dry matter intake were calculated for each level of Rumensin based upon the number of cows and number of weeks completed in lactation by each cow.

Cows were fed ad libitum a total mixed ration (TMR) throughout the study. For the lactation period, the average TMR was calculated based on the proportion of each feed in the TMR and the number of days fed. Feeds were analyzed routinely during the course of the study for dry matter, crude protein and neutral detergent fiber. Fat and ash were from analysis and nutrient composition tables. Total composition expressed on a dry matter basis allowed calculation of non-fiber carbohydrate by difference. The average feed composition of TMR and the nutrient analysis for each feed at each site were entered into the Cornell Net Carbohydrate Net Protein model (CNCPS v 4.1). Similarly, concentration of individual fatty acids was calculated on of each TMR using data from Dr. Peter Moate (personal communication), refereed publications and analysis by Dr. Tom Jenkins, Clemson University (personal communication). The outputs from CNCPS and from fatty acid calculations were used to determine nutrient characteristics of TMR for each site.

RESULTS APPLIED TO QUADRANT ANALYSIS

Inspection of the individual treatment means at each site allowed grouping of trial sites into one of three quadrants in the four quadrant model (Figure 1) based on the yields of fat and protein of cows fed Rumensin compared to control cattle. Quadrant 4 included three sites, California, Florida and Ontario, where mean yields of fat and protein by cows fed Rumensin were higher than Control. Quadrant 2 included four sites, Michigan North Carolina, Indiana, Quebec, where mean yield of fat was lower and mean yield of protein was higher for cows fed Rumensin compared to that of Control. Quadrant 1 included three sites, Alberta, New York 1, and New York 2 where the mean yields of fat and protein from cows fed Rumensin were lower than the Control. Within a quadrant, information from approximately 70 cows per level of Rumensin provided confidence in the performance measure.

Performance of cows by Quadrant is shown in Table 1. Cows fed Rumensin at Quadrant 4 sites averaged 2.5 to 4.5 pounds per day more milk than control. There was little difference in milk fat percent (<0.1%) and milk protein percent (<0.05 %) between control cows and Rumensin fed cows. Dry matter intake was similar between treatments. Milk production efficiency at Quadrant 4 sites provided greater output of economic value on the same level of feed intake.

Cows fed Rumensin at Quadrant 2 sites averaged 1.6 to 3.5 pounds per day more milk than control. There was a major difference in milk fat percent, especially at the highest dose (Δ= 0.4%) between control cows and cows fed Rumensin. Differences in milk protein percent between control and Rumensin treatments in Quadrant 2 were of similar magnitude as that in Quadrant 4. Dry matter intake was similar between treatments. Milk production
efficiency at Quadrant 2 sites provided greater output of economic value on the same level of feed intake.

Cows fed Rumensin at Quadrant 1 sites averaged 0.8 pounds per day less to 0.6 pounds per day more milk than Control. There was an apparent linear decrease in milk fat percent with increasing dose between control cows (3.63%) and Rumensin fed cows (3.40% at 22 g/t Rumensin). Differences in milk protein percent between control and Rumensin treatments in Quadrant 1 was of similar magnitude as that in other Quadrants. Dry matter intake of cows fed Rumensin at Quadrant 1 sites were less than Control cows averaging 2.2 and 2.6 pounds per day less for 15 and 22 g/t levels of Rumensin. Milk production efficiency at Quadrant 1 sites provided the same output on less feed intake.

Output from CNCPS of diets at individual trial sites was used to evaluate ration characteristics that might influence the nature of the response of cows fed Rumensin. The chemical composition of diets in each of the Quadrants is in Table 2.

Table 1. Performance of Cows Fed Rumensin based on Quadrant Analysis.

<table>
<thead>
<tr>
<th>Item</th>
<th>Milk, lb/d</th>
<th>Fat, %</th>
<th>Fat, lb/d</th>
<th>Protein, %</th>
<th>Protein, lb/d</th>
<th>DMI, lb/d</th>
<th>MPE$	extsuperscript{1}$</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>63.8</td>
<td>3.73</td>
<td>2.38</td>
<td>3.14</td>
<td>2.01</td>
<td>44.6</td>
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<td>3.09</td>
<td>2.08</td>
<td>44.7</td>
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<td>66.3</td>
<td>3.65</td>
<td>2.42</td>
<td>3.12</td>
<td>2.07</td>
<td>44.0</td>
<td>1.82</td>
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<table>
<thead>
<tr>
<th>Item</th>
<th>Milk, lb/d</th>
<th>Fat, %</th>
<th>Fat, lb/d</th>
<th>Protein, %</th>
<th>Protein, lb/d</th>
<th>DMI, lb/d</th>
<th>MPE$	extsuperscript{1}$</th>
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<tr>
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<td>3.09</td>
<td>2.23</td>
<td>44.5</td>
<td>1.88</td>
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</table>

$	extsuperscript{1}$ Milk Production Efficiency
Diet Carbohydrates

Sites in Quadrant 4 had higher (P < 0.05) NDF and lower (P=0.07) NFC as percentages of dry matter than diets at Quadrant 1 and Quadrant 2 sites. Diets at Quadrant 4 sites were also lower (P= 0.07) in Fraction B1 compared to diets at Quadrant 1 and Quadrant 2 sites. Fraction B1 in CNCPS represents starch and pectin, major constituents of NFC. Other carbohydrate fractions were not different between Quadrants. Total carbohydrate digested was higher (P < 0.05) for Quadrant 2 diets and intermediate for Quadrant 4 diets. The components of carbohydrate digestion in CNCPS, diet (P < 0.07) and bacteria (P < 0.005), followed the same pattern. Bacteria yield in CNCPS is predicted from bacteria CHO digested and was greatest for diets in Quadrant 2 and Quadrant 4.

Diet Protein

Diets averaged 17.9, 17.9 and 18.6 percent crude protein for Quadrants 4, 2 and 1, respectively. Total dietary metabolizable protein tended (P < 0.10) higher for Quadrant 4 diets than Quadrant 1 diets (Table 6). The diet for Quadrant 2 was intermediate. Components of metabolizable protein, bacteria and undegraded feed protein were numerically higher for Quadrant 4 > Quadrant 2 > Quadrant 1. Metabolizable protein from bacterial was greater (P< 0.01) in diets from Quadrant 4 and Quadrant 2 sites than in Quadrant 1 sites. Other measures of protein utilization from CNCPS were not different between Quadrants.

Table 2. Chemical composition of diets by Quadrant.

<table>
<thead>
<tr>
<th>Item</th>
<th>Quadrant 1</th>
<th>Quadrant 2</th>
<th>Quadrant 4</th>
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<tbody>
<tr>
<td>Crude protein, % DM</td>
<td>18.6</td>
<td>17.9</td>
<td>17.9</td>
</tr>
<tr>
<td>Metabolizable protein</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bacterial, % of CP</td>
<td>32.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>34.1&lt;sup&gt;b&lt;/sup&gt;</td>
<td>34.2&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Undegraded feed, % of CP</td>
<td>28.9</td>
<td>30.4</td>
<td>31.7</td>
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<tr>
<td>Total, % of CP</td>
<td>61.5</td>
<td>64.5</td>
<td>65.9</td>
</tr>
<tr>
<td>NDF, % DM</td>
<td>28.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>29.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>32.9&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>NFC, % DM</td>
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<td>41.8&lt;sup&gt;y&lt;/sup&gt;</td>
<td>38.7&lt;sup&gt;y&lt;/sup&gt;</td>
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<tr>
<td>CHO Fraction B&lt;sub&gt;1&lt;/sub&gt;</td>
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<td>31.2&lt;sup&gt;x&lt;/sup&gt;</td>
<td>27.3&lt;sup&gt;x&lt;/sup&gt;</td>
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<tr>
<td>Fat, % DM</td>
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<td>5.6</td>
</tr>
<tr>
<td>Linoleic acid, % DM</td>
<td>1.54</td>
<td>1.71</td>
<td>1.82</td>
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</tbody>
</table>

<sup>A,B</sup> Means with unlike superscripts are different (P<0.01).
<sup>a,b</sup> Means with unlike superscripts are different (P<0.05).
<sup>x,y</sup> Means with unlike superscripts are different (P<0.10).
Diet Fat

None of the fatty acids, percent fat in the diet or indices of fat utilization were different based on Quadrant grouping of sites. Diets from Quadrant 1 (sites with the largest decrease in fat percent and yield) had the highest level of dietary fat but lowest concentration of linoleic acid (1.54 % of DM) compared with the fat and linoleic acid (1.82 % of DM) in TMR from Quadrant 4 (sites with the least change in fat percent and yield). This finding suggests that linoleic acid may be present in sufficient amounts in many diets of dairy cows to cause a decrease in milk fat percent. Factors that produced an altered ruminal fermentation, e.g. inadequate dietary fiber, highly fermentable starch, as suggested by Griinari et al., (1998) become the diet characteristics that must be managed in order to prevent lower milk fat per-cent with Rumensin or any other contributor to low milk fat.

SUMMARY

Rumensin affects Gram-positive bacteria allowing for the proliferation and dominance in ruminal metabolism of Gram-negative bacteria. Changes in bacterial populations favor organisms that produce propionic acid and a reduction in methane production. Rumensin also reduces deamination so that more feed protein passes intact to the small intestines. Rumensin brings value to the cattle producer through performance enhancement and reduced risk of digestive and metabolic disorders. In dairy cattle, the greatest value of Rumensin is obtained from diets that promote fiber utilization and microbial growth. Under these conditions, changes in milk fat are minimal with Rumensin.

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