Rumensin for Ruminants

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Introduction

Rumensin (trade name for monensin Na, Eli Lilly and Company) is a biologically active compound produced by *Streptomyces cinnamonensis* and initially proven to be an effective anticoccidial in poultry rations. Currently, Rumensin is used by a large percentage of cattle feeders through the country to increase feed efficiency. The efficacy of this compound is well documented, however, much still needs to be known about its effects on animal health, protein utilization and mode of action.

Chemical Properties: Rumensin (monensin sodium) is a polyether with a molecular weight of 692. The structure of this compound is illustrated in Figure 1.

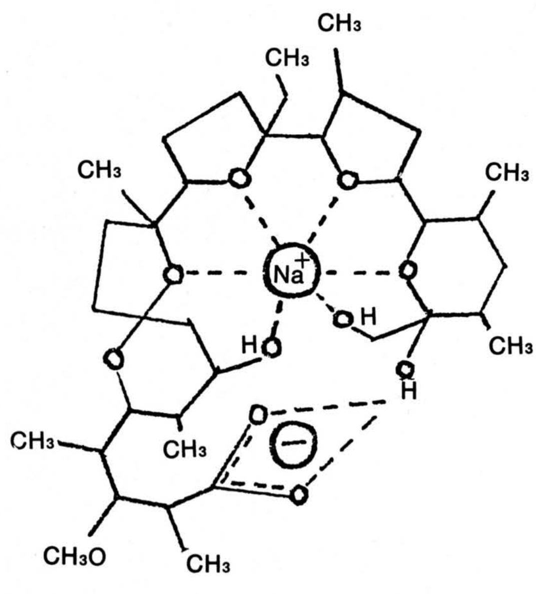

Figure 1. Monensin Sodium ($C_{36}H_{61}O_{11}Na$)

Monensin is only slightly soluble in water and is soluble in most organic solvent.

Influence of Rumensin on Animal Performance

Feedlot Performance: Studies by Embry and Swan (1976); Sherrod, et al. (1975); Sherrod and Burnett (1976); Lofgreen (1976); Perry, et al. (1976); and Raun, et al. (1976) have all indicated that Rumensin depressed feed intake to a certain extent, had no effects on average daily gains, and increases in feed efficiency averaging 10% were reported. The effects of Rumensin dosage on feed intake, average daily gain, and feed efficiency were reported by Raun, et al. 1976, and are illustrated in Figure 2.

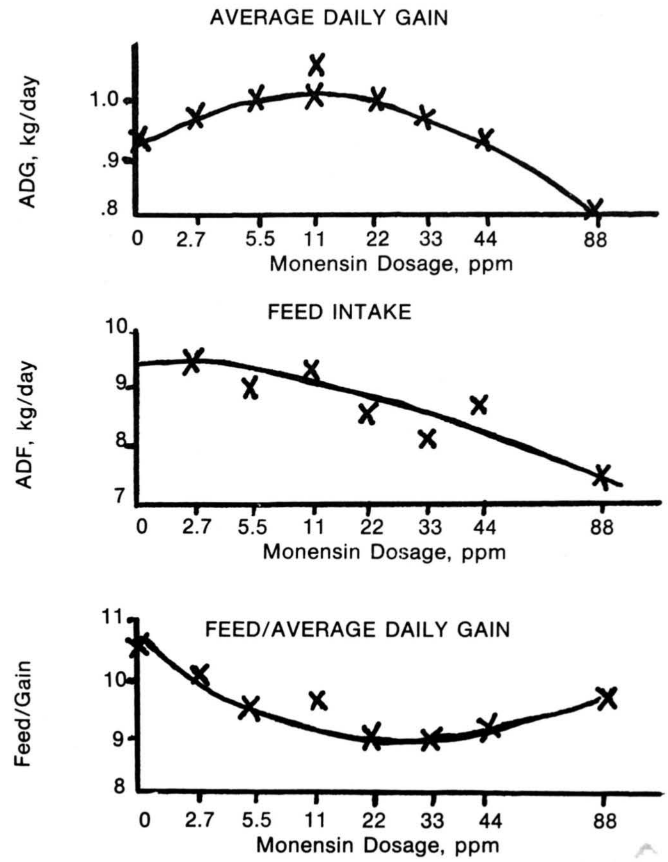

Figure 2. Rumensin on feedlot performance.

Rumensin was found to cause a decrease in feed intake with increased dosage. Average daily gains, however, tended to increase slightly at 11 ppm and were approximately equal to the control steers at 33 ppm. The optimal level for the feed efficiency response was reported to be 33 ppm, which equals 300 mg per head/day assuming 20 lbs. consumption or 30 g per ton of feed. No changes in carcass composition have been reported with Rumensin feeding (Potter, et al. 1976b).

Davis and Enhart (1975), Utley (1975), Nelson (1975), and Gill (1976) have all reported that the increased performance with Rumensin in the ration is additive to the anabolic response of implants such as DES, Ralgro and Synove. This response would be as expected since the modes of action of these compounds are different.

Studies by Gill (unpublished data) indicate that a strong protein level and Rumensin interaction exists, which suggests that Rumensin-treated rations might require less protein for optional performance. This indication is not unexpected in light of some of the effects Rumensin is reported to have on protein metabolism.

Forage-Fed Cattle: In contrast to cattle fed high concentrated rations, studies by Potter, et al. 1976a; Neumann, 1975; Anthony, 1975; Bolsen, 1975; and Oliver, 1975, reported increased average daily gain for roughage-fed cattle.

The difference observed in gains between pasture and feedlot studies has been explained by Potter (1975). On high roughage rations feed intake is limited by the capacity of rumen-reticulum. The amount of roughage consumed depends on the initial bulk of the feed and the rate and extent to which it is digested. Forage with comparatively high nutritive value will be digested rapidly in the rumen, thus more can be consumed and rate of gain should increase. Once the energy density of a ration increases beyond a certain point, chemostatic factors limit its intake. According to Potter, in roughage rations the increased energy availability due the Rumensin will tend to increase gains with consumption being the same, while in high concentrate rations the increase in energy availability will decrease consumption. The amount of energy retained will remain constant since intake is controlled by available energy; however, less feed would be required.

A major problem with feeding Rumensin to pasture cattle is the delivery system. If the cattle are going to be fed a daily supplement there may be no problem in incorporating Rumensin into this supplement. Feeding a supplement daily is quite often not practical. Studies using Rumensin incorporated into a molasses block (De Muth, et al. 1976, and Neumann, 1976) have shown mixed results.

Influence of Rumensin on Energy Metabolic

Fermentation and Products: The microorganisms of the rumen have the ability to hydrolyze the α1-4 glucosidic linkage of starch and the β1-4 linkage of cellulose and hemicellulose to form glucose. The glucose is then metabolized via anaerobic glycolysis to pyruvate. Pyruvic acid is the common intermediate in rumen volatile fatty acid (VFA) fermentation. Acetic, propionic and butyric acids are the major VFA's formed in the rumen and they serve as the major energy source for ruminants.

One of the most consistent changes occurring when Rumensin is added to a ration is the shift in the molar proportion of the VFA produced. As reported in Table 1 (Raun, et al. 1976) little change was observed in total VFA levels, however, an increase in the molar percentage of propionate produced at the expenses of acetate is apparent.

Table 1

Monensin and Rumen Volatile Fatty Acids Level[1]

VFA's	Monensin Dosage, ppm 0	33
Total VFA (mm/liter)	83.4	80.6
Acetic Acid[2]	52.8	49.5
Propimic Acid[2]	33.3	38.8
Butyric Acid[2]	9.8	8.3

[1]Raun, et al. 1976. [2]Percent of total moles of acid.

This change in acetate-to-propionate ratio has been one of the most consistently observed responses to Rumensin in both *in vitro* and *in vivo* studies. In Figure 3, the efficiency of VFA formation as reported by Raun, 1975, is illustrated below.

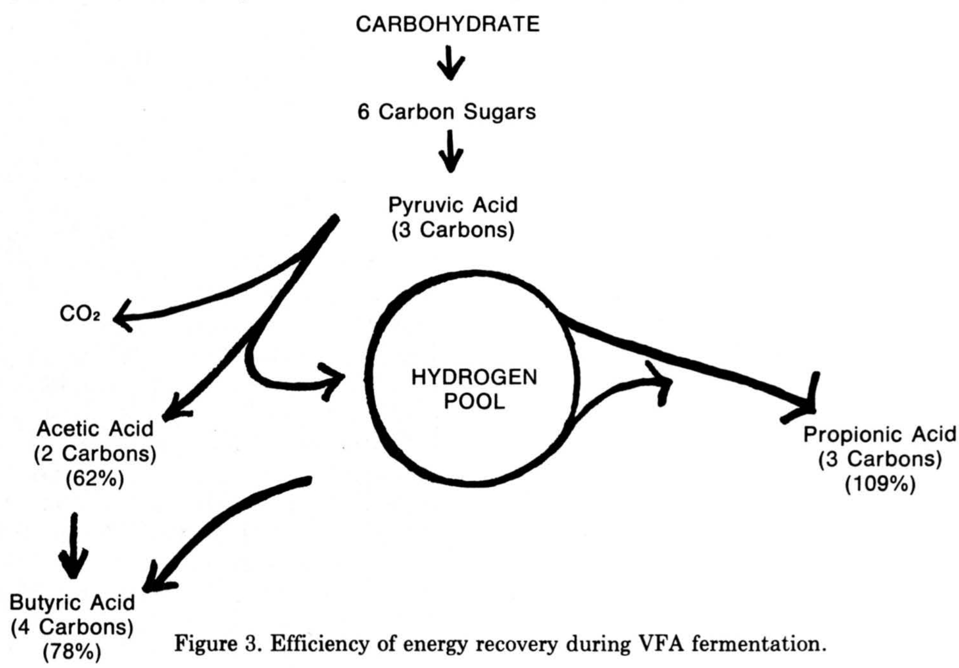

Figure 3. Efficiency of energy recovery during VFA fermentation.

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The efficiency of energy recovery in which glucose is converted to acetate is reported to be 62%, while 109% of the energy was retained by conversion to propionate. The efficiency of greater than 100% is due to the addition of energy from hydrogen. This energy would otherwise be lost as methane. Data reported by Thornton, et al. 1976, seems to indicate methane production is decreased with Rumensin feeding.

If the molar percentages of acetate, propinate and butyrate (Table 1) are multiplied by the percentage of energy we expect to recover, we recover approximately 77% of our initial energy from the control ration and 80% from the Rumensin ration–only a 3% difference. Thus, we cannot account for the 10-17% increase observed by Raun.

Another explanation for the increase in feed efficiency is the possible difference in heat increment from the utilization of acetate and propinate by the ruminant tissue (Raun, 1975). With a maintenance diet, Holter, 1970, calculated that a 40% energy loss occurred as heat increment with the metabolism of acetic acid and only 18% loss during the metabolism of propionic acid. Values by Armstrong and Blaxter, as reported by Raun (1975) indicated that for fattening cattle 67% of the energy was lost with acetic acid metabolism and 44% with propionic acid metabolism. If these values are correct one could explain the increased efficiency observed for Rumensin-fed cattle. It was suggested by Bull (1970) that a sudden influx of a high level of acetic acid could result in the observed high heat increment. In experiments by Orskov and Allen as summarized by Smith (1971) the efficiency of utilization of acetate and propionate were similar when fed to growing and fattening lambs.

Although lower heat increment associated with high propionate levels has been suggested as a possible mode of action, it must be questioned until more knowledge of the subject is obtained. It was stated by Smith, 1971, that further insight into the utilization of VFA's is needed before one can assess the contribution of acetic and propionic acids to the energy requirement of ruminants.

In addition to the changes in the acetate propinate ratios in the rumen, Rumensin is reported (Raun, 1975; Beede, et al., 1975) to have an effect on the pathway in which propionate is produced. Propinate can be produced either by the succinate or acyrlate pathway with the succinate pathway being of primary importance. When Rumensin is added to the ration, propionate production via the acyrlate pathway tends to predominate. The impact of this shift on animal performance has yet to be determined.

Digestibility: No consistent effect has been observed in dry matter digestibility with Rumensin. Potter and Richardson, 1975, reported a slight increase in dry matter digestibility with Rumensin, and Prigge and Owens, 1975, observed an increase in *in vitro* dry matter digestibility. Studies by Dinius, et al. 1976, however, showed no change in dry matter or cellulose digestibility.

Rumen Microflora: The effects of Rumensin on the rumen microflora has been studied by Dinius, et al. 1975, and no change in total bacteria cellulolytic bacteria or protozoa numbers were observed with Rumensin added to the ration at 33 ppm. Protozoa number have been observed by Potter and Richardson, 1975, to remain comparable to the control on either pasture or feedlot ration. It was reported by Richardson, 1974, that a shift to gram negative bacteria in the rumen of Rumensin-treated cattle occurred.

Blood Parameters: Potter and Richardson (1975) found that Rumensin tended to increase blood glucose on an average of three to five mg/100 ml and small increases in plasma insulin levels were also observed. The increase in propionic acid production was suggested by Potter and Richardson to be responsible for the increased plasma glucose and insulin levels.

Influence of Rumensin on Nitrogen Metabolism

The effects of Rumensin on the nitrogen metabolism of ruminants is even less understood than its effect on energy metabolism. Potter and Richardson (1975) reported the Rumensin tended to decrease rumen ammonia levels and increase blood urea nitrogen. Several explanations exist for the decrease in rumen ammonia, which are: increase absorption into the blood, increased microbial utilization, or Rumensin acting as a deaminase inhibitor. It is difficult to accept the fact that more ammonia is being transported across the rumen wall in spite of higher blood urea levels since little or no change in rumen pH occurs with the feeding of Rumensin. The findings of Owens (unpublished data) using abomasally fistulated steers indicate that more ammonia is incorporated into microbial protein. Chalupa (personal communications) feels that Rumensin might act as a deaminase inhibitor, thus less intact protein would be degraded to ammonia.

In addition to the increase in microbial protein synthesis and possible deaminase inhibition, the increased production of propinate in the rumen has been reported to have a protein-spearing effect. Two major precursors of glucose in the rumen are propionic acid and amino acids. Thus, if a greater percentage of the propinate is used to synthesize glucose, amino acids could be speared from use as an energy source and thus will be available for tissue synthesis.

Decrease in plasma essential amino acids with levels of nonessential remaining unchanged were observed by Potter and Richardson which further suggest that Rumensin might enhance protein deposition. Nitrogen digestibility has been reported to (Potter and Richardson, 1975) increase with Rumensin and 11 to 22% increase in crude protein retention was observed.

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Studies by Gill (unpublished data) indicated that feed efficiency was improved the greatest when Rumensin was fed with 9.5% crude protein rations and the cattle performed superior to cattle on 12% protein ration both with and without Rumensin. Dratt, et al. (1976), indicated that withdrawing protein during the finishing phase of feeding had less of a depressing effect on animal performance when Rumensin was fed.

Effects of Rumensin on Animal Health

Liver abscesses: A slight increase in the number of condemned livers due to abscesses has been observed by Lilly workers. In a summary of 25 trials, feedlot trials, in which Rumensin was fed at the recommended level (30 g/ton), an increase of 3% in abscesses was observed. Although this increase is not considered large, its potential economic input could be great in light of the widespread use of Rumensin.

Coccidiosis: Rumensin under the name of Coban has been used effectively as a coccidiostrate in poultry ration for many years. In ruminants, levels of less than 5 g/ton have been effective in suppression of the number of oocysts in both cattle and sheep. Eimerin bovis has been controlled with Rumensin in calves (Fitzgerald, 1973) and in sheep *E. ouina, E. ahsata, E. ninakohlyakinovae, E. parva,* and *E. pallida* have been effectively controlled by feeding Rumensin at or below the level required for maximum response in feed efficiency.

Polioencephalomalacia: It was indicated by Potter and Richardson (1975) that a relationship between polioencephalomalacia (PEM) might exist in cattle with feeding of a combination of Rumensin and Tylan (trade name, Eli Lilly and Co.). Studies were then conducted in cattle to measure parameters associated with PEM and no differences in ruminal thiamine, plasma pyruvate and plasma lactate levels were reported. Their results suggest that PEM might not be a real problem when feeding Rumensin.

Health of Stressed Calves: Studies were conducted by the University of California stress calf study group (Prigge, et al., 1975) and involved starting newly-received 300-450 wgt. feeder calves. When cattle were first placed on Rumensin, feed intake was reduced considerably. This could indicate that problems might exist on the health of newly-received feedlot cattle which often have depressed appetites when they enter a feedlot. Studies conducted by the University of California stress calf group (Prigge, et al., 1975) have indicated that feed consumption was decreased during the first week after arrival by 30% with Rumensin feeding. However, the medical cost average of $3.30 for the Rumensin cattle (30 g/ton) as opposed to $5.15 for the controls, and feed efficiency was superior over the duration of the study to both the controls and cattle started at 0 and 10 g/ton of Rumensin for the first two weeks and then shifted to 30 g/ton. A second study indicated that 40% of the cattle had temperatures over 103°F for the controls during the first seven days and 30% for the Rumensin cattle.

Although the data is not completely analyzed the results do suggest at least that no adverse effect on animal health existed for Rumensin-treated steers.

Toxicity: Various clinical parameters were reported by Van Duyn, 1975, on cattle fed Rumensin at levels up to 100 g/ton for 160 days. No changes indicative of toxicity were observed for hematology, serum chemistry, urinalysis, or organ weight parameters. No gross or microscopic tissue changes relating to treatment were observed. The 100 g/ton level did, however, reduce gain.

Van Duyn (1975) reported that when oral doses of Rumensin were fed to cattle, the lowest dose to cause mortality was 55 mg/kg of body weight which is 80 times the recommended dose of 30 g/ton. The LO_0 appeared to be about 32 mg/kg, and the LD_{50} was not calculated.

Oral toxicity trials with horses indicated that they were more sensitive to Rumensin than cattle and deaths occurred when they were offered Rumensin-treated feed at 110 g/ton. However, no mortality occurred at the 30 g/ton level.

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Discussion

Question: How many milligrams are you giving the calves per day?

Answer: We give them 300 milligrams, 30 grams per ton in the feed.

Question: What about pasture programs?

Answer: On pasture rations they don't have a recommendation–it is not cleared for pasture feeding programs. But, they recommend 20 grams per ton or 200 milligrams per head–that is based on 20-pound consumption.

Question: Can you explain the adaptation to acetic acid that you mentioned?

Answer: I can't really explain it–it was an observation. Bole, 1970, observed that he had completely different heat increments during the first 10 days of this study when he was feeding acetic acid to dairy cattle as opposed to, say, at 20 days. They were utilized fairly equally at that point. I don't know why this is.

Question: Then there would be no advantage to increasing the percentage of propionic acid after 21 days?

Answer: It would be an advantage to increase the percentage of propionic acid. First of all, it seems to indicate feed efficiency. You get a 3% increase in energy-available by increasing propionic acid. It is also a fairly good indicator of feeds that perform more efficiently. In high concentrate rations we get more propionic acid. It is used as a screening process. Rumensin was found on that screening process. I could not really tell you why; it probably has something to do with pathways. I tend to think that when rumen bacteria are turning over at a faster rate, we tend to get more propionic acid production. Bacteria turning over at a faster rate are much more efficient in the energy utilization than those that are turning over at a slower rate. But that's only theory. We should look at this and it should not be ignored and I know that a lot of the pharmaceutical companies use this volatile fatty acid ratio as a screening process for these chemicals, rumensin type chemicals, that show some rumen activity.

Question: In your pathology data on toxicity, you stated no lesions, but I wondered has anybody looked at the rumen wall because you showed a lot of biochemical changes and suddenly there was a slight increase in liver abscesses. It looks like some of the rumen wall functions are perhaps being modified metabolically.

Answer: It is a possibility. In some of the studies we ran at Oklahoma we found twice the level of potassium in the rumen with rumensin-fed cattle as opposed to control cattle. Rumensin has the ability to chelate monovalent cations and this could affect transport across membranes, so it is a possibility.

Question: Did you have any information on the type of coccidial species that was in the trial and do you have any information on whether in cattle it is coccidiostatic or coccidiocidal?

Answer: No, I don't. These were observations that were passed along to me. It is not a controlled study, it is an observation only.

Question: Do you see a future for the cow/calf operator in, first of all, his cow herd and, secondly, pre-feeding calves, and, thirdly, growing heifers?

Answer: Yes, it is effective if you can get it to them. It will work on heifers equally as well as it will work on steers; it will work on young animals and old animals; the problem is the delivery system. I guess creep-feeding heifers with an energy source is uneconomical with the price of grains we have today. Some work has been done with molasses blocks, and I imagine some work has been done with mineral blocks, but they have been mixed. So, the problem with the cow/calf man is the delivery system. If you are going to feed them a supplement of cottonseed cakes or something like that daily, rumensin will work. But, if you are not, then you have a delivery system problem of getting it to them.

Question: Have you tried other levels?

Answer: Yes, we tried 20 grams per ton and 30 grams per ton; 30 worked best with us.

Question: If you are feeding a one-pound supplement, and the horse gets into that feed, it is pure poison. Three or four pounds of that calculated out gets into the toxic stage.

Answer: Yes, I have heard stories about horses getting into the rumensin bag as it comes from the factory and dying.

Question: What about poultry?

Answer: I assume it is not toxic in poultry rations. I can't remember any specific data but I think it is safer in pigs than it is in horses. Why horses are extremely sensitive to it, I don't know. But I don't think pigs are.

Question: Is any work being done now on reducing the percentage of rumensin and still maintaining the feed efficiency in it?

Answer: Yes, there is a study in Oklahoma that is currently under way. They started 600-weight cattle on 9-1/2, up to about 12-1/2% protein, and the 9-1/2% rumensin ration was the best ration compared to the other rumensin rations, and compared to the control rations. I think the maximum response in the controls was something like 11-1/2% protein.

Dr. Don Williams: I believe that is going to end up to vary a little bit with the type grain used and the size of the cattle. I would urge that judgment be delayed until other trials are completed.

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