

A SINGLE DRENCH OF VIRGINIAMYCIN TO CONTROL ACIDOSIS IN SHEEP AND CATTLE

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SUMMARY

Previous studies have shown that a single drench of virginiamycin (2.6 mg/kg liveweight) can prevent mortalities due to lactic acidosis in sheep. It was proposed that the most important feature of a successful drench of virginiamycin is the length of time that lactate production in the rumen is suppressed. The aim of this study was to examine the likely effective dose prior to testing the technique on cattle exposed to grain feeding and therefore prevent mortalities. This was achieved by drenching both sheep and cattle with equivalent doses of virginiamycin and taking periodic rumen samples which were incubated with glucose *in vitro* to determine potential lactate production. Drenching with virginiamycin decreased L-lactate in both sheep and cattle. D-lactate was increased in sheep but was variable in cattle. Rumen L-lactate levels were suppressed for 4-5 days in sheep when they were drenched with virginiamycin at 2.6 mg/kg and twice that dose did not appear to increase the period of L-lactate suppression. L-lactate was suppressed for only 3 days in cattle drenched with virginiamycin at 5.2 mg/kg, indicating that cattle require larger doses of virginiamycin than sheep to achieve similar levels of protection against lactic acidosis.

Keywords: Virginiamycin, sheep, cattle, single drench, rumen lactate

INTRODUCTION

Virginiamycin has been shown to control mortalities in sheep due to grain poisoning when included in the ration (Godfrey *et al.* 1995). Virginiamycin has also been used in cattle rations to allow sudden introduction to grain feeding without grain poisoning problems (Rowe *et al.* 1994; Zorrilla-Rios *et al.* 1994a; Zorrilla-Rios *et al.* 1994b and Tudor *et al.* 1994). Under commercial conditions, dosing animals directly would protect sheep and cattle from grain poisoning when virginiamycin cannot be added to the grain such as when grazing crops or stubbles containing sufficient grain to cause grain poisoning. It would also be useful in situations where mixing equipment was not available to add virginiamycin to grain.

Thorniley *et al.* (1996) have shown that a single drench of virginiamycin at 2.6 mg/kg liveweight prevented mortalities due to grain poisoning when weaner wethers were fed wheat *ad lib* under pen conditions. A drench of virginiamycin at 1.3 mg/kg liveweight significantly reduced mortalities due to grain poisoning. This method of administering virginiamycin could also be useful in cattle production systems such as lotfeeding or feeding grain to grazing cattle.

Murray *et al.* (pers. comm.) have determined the likely dose required to prevent grain poisoning by administering virginiamycin to sheep fed a roughage diet and 'collecting samples of digesta for *in vitro* incubation with glucose to determine the potential lactate production of the digesta.

The important characteristics of an effective single drench are firstly the extent of reduction in lactate production and secondly, but perhaps more importantly, the length of time lactate production remains reduced. Murray *et al.* (pers. comm.) have shown that lactate production in sheep is reduced as the dose of virginiamycin is increased at low dose rates. However at high dose rates, increasing dose did not reduce the level of lactate production but suppressed lactate production for longer periods. They found that virginiamycin at 2.94 mg/kg reduced rumen lactate production for 72 hours in sheep.

It was assumed that cattle would require the same period of reduced lactate production as that achieved in sheep receiving virginiamycin at 2.6 mg/kg liveweight, which was the dose previously shown to prevent grain poisoning in sheep (Thorniley *et al.* 1996). Our hypothesis was that the same dose rate in sheep and cattle would provide the same period of residual activity and therefore the dose required to prevent grain poisoning in cattle would be the same as that in sheep.

MATERIALS AND METHODS

Animals

Experiment 1 Twenty Merino wethers weighing approximately 45 kg (SEM \pm 0.7) were drenched against intestinal parasites with 10 mL of Ivomec (Merck Sharp and Dohme, South Granville, N.S.W.) then randomly allocated to individual pens in a shed with mesh flooring. Sheep were stratified on liveweight 3 days prior to treatments starting, and allocated to treatments from within strata.

Experiment 2 Twenty Angus x Friesian bulls weighing approximately 338 kg (SEM \pm 7.6) were treated against intestinal parasites with Ivomec Pour-on (Merck Sharp and Dohme, South Granville, N.S.W.) then randomly allocated to individual feedlot pens. Cattle were stratified on liveweight the day that the treatments started, and allocated to treatments from within strata.

Dietary treatments

Experiment 1 Sheep were fed a ration of 750 g of wheaten chaff for the first 4 days they were in the shed then 800 g of wheaten chaff for the next 14 days prior to the treatments starting and for the 10 days of the trial. The chaff was fortified with urea (10 kg/tonne) and a mineral-vitamin premix (2.5 kg/tonne) containing the following compounds (g/tonne of complete ration): ferrous sulphate, 30; zinc oxide, 30; manganous oxide, 9; copper sulphate, 4; calcium iodate, 0.5; sodium molybdate, 0.4; selenium, 0.1; and vitamins (MIU/tonne): D, 0.21; E, 0.0225. Animals had free access to fresh water at all times.

Experiment 2 Cattle were fed a basal ration of pasture hay ad lib prior to the treatments starting then at a level equivalent to 2.5% of liveweight after treatment with virginiamycin. Cattle received the same mineral-vitamin mix as described for the sheep. Animals had free access to fresh water at all times.

Virginiamycin administration

Virginiamycin was administered as a single oral drench of virginiamycin suspended in distilled water via a syringe. Dose rates of virginiamycin were 0, 1.3, 2.6 and 5.2 mg/kg liveweight. Virginiamycin was provided as a wettable powder formulation containing 40% virginiamycin. This formulation formed a suspension and was shaken thoroughly before it was administered to each animal.

Sampling and analyses

Rumen samples were taken by stomach tube prior to drenching (0 hours), 6 hours later and then 1, 2, 3, 4, 5, 7 and 10 days after drenching. Rumen fluid was strained through a stocking and pH was measured at the time of sampling. Duplicate 2 mL subsamples were diluted with 3 mL of a solution containing 20 mg/mL glucose in distilled water, thoroughly mixed, then incubated for 24 hours at 40°C. Following incubation, samples were acidified and frozen at -18°C prior to analysis for lactic acid.

L-lactic acid and D-lactic acid concentrations were measured using a Cobas Mira Auto Analyser (Roche Diagnostics Inc., Frenchs Forest, N.S.W.) and enzyme kits (D-Lactic acid/L-Lactic acid kit, Cat. No. 1112821, Boehringer-Mannheim, Mannheim, Germany).

RESULTS

Experiment 1 (Sheep)

The amount of L-lactate produced during incubation with glucose was significantly reduced ($P < 0.001$) to the same level by all doses of virginiamycin within 6 hours of drenching (Figure 1). The L-lactate concentration in rumen fluid from sheep drenched with 1.3 mg/kg virginiamycin was not significantly different ($P > 0.05$) to the control animals 3 days after drenching. Lactate production was reduced for 5 days in sheep drenched with 2.6 and 5.2 mg/kg virginiamycin.

The amount of D-lactate produced during incubation with glucose was significantly increased ($P < 0.05$) by all doses of virginiamycin within 6 hours of drenching (Figure 1). D-lactate appeared to remain increased for approximately the same period as L-lactate was reduced although higher coefficients of variation for D-lactate resulted in a shorter period when D-lactate was significantly different from the control.

There were no significant differences ($P > 0.05$) in chaff intake, liveweight change or rumen pH as a result of drenching with virginiamycin.

Experiment 2 (Cattle)

Lactate concentrations in rumen fluid from cattle were more variable than for sheep. L-lactate was significantly reduced ($P < 0.05$) to the same level by all doses of virginiamycin within 6 hours of drenching (Figure 2). The L-lactate concentration in rumen fluid from cattle receiving 1.3 or 2.6 mg/kg virginiamycin

was not significantly different ($P > 0.05$) from the rumen fluid from control animals 2 days after drenching and the corresponding period in cattle receiving 5.2 mg/kg virginiamycin was 3 days.

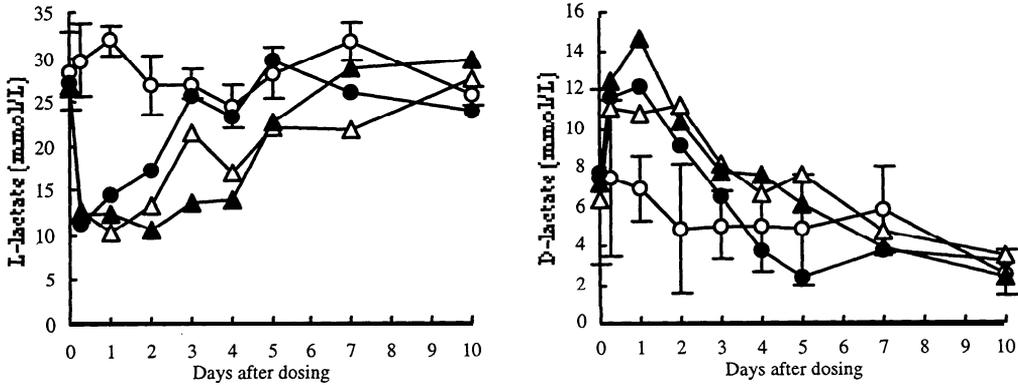


Figure 1. Mean lactate concentration (mmol/L) in rumen digesta of sheep after drenching with virginiamycin (□ - control, 0 mg/kg, ± SE; ● - 1.3 mg/kg; △ - 2.6 mg/kg; ▲ - 5.2 mg/kg) and incubated with glucose

D-lactate production in rumen digesta from cattle drenched with virginiamycin was not significantly different from that of the controls (Figure 2) although there was a trend ($P = 0.064$) for rumen fluid from cattle drenched with 5.2 mg/kg virginiamycin to have lower D-lactate production than the controls.

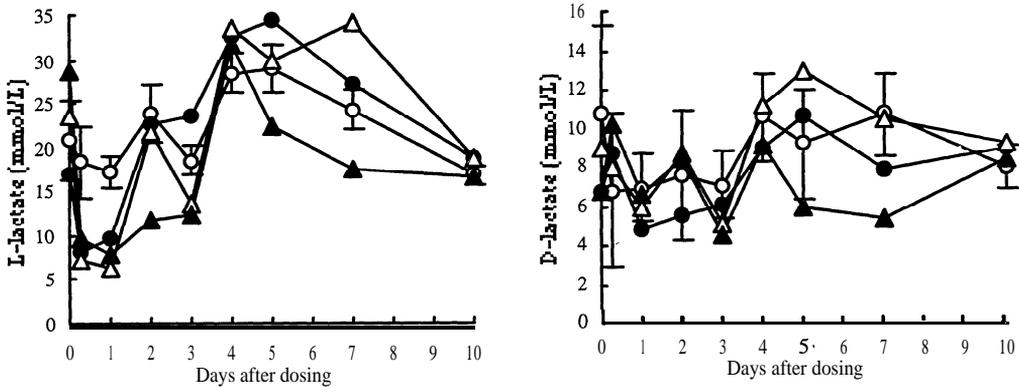


Figure 2. Mean lactate concentration (mmol/L) in rumen digesta of cattle after drenching with virginiamycin (□ - control, 0 mg/kg, ± SE; ● - 1.3 mg/kg; △ - 2.6 mg/kg; ▲ - 5.2 mg/kg) and incubated with glucose

In general cattle ate all the feed offered and there were only occasional feed refusals. Rumen pH was very variable in cattle and tended to be high with means of up to 7.5. Saliva contamination was noted in some rumen samples from cattle although this was not correlated with pH or lactate production.

DISCUSSION

It appears that cattle require a higher dose of virginiamycin than sheep for prolonged reduction of L-lactate production in rumen digesta. A single drench of virginiamycin at the rate of 2.6 mg/kg was

previously shown to successfully prevent grain poisoning in sheep (Thorniley *et al.* 1996). This dose of virginiamycin depressed L-lactate production in sheep rumen fluid for 4-5 days. If this is, in fact, the period of lactate suppression required to prevent grain poisoning in cattle, then the results of our experiment indicate that the effective dose for cattle would be greater than 5.2 mg/kg.

The shorter suppression of L-lactate production in cattle compared to sheep could be due to a greater dilution rate in the rumen. This could be the case if the cattle had a larger rumen volume relative to body size or shorter retention time in the rumen compared to the sheep. This explanation is supported by Parra (1978), who suggested that cattle generally have a larger rumen volume than sheep and Van Soest (1982), who indicated that retention time of liquid in the rumen of cattle is less than in sheep. Rumen volume and retention time were not assessed in these experiments. Differences in residual activity could also be due to differences in the type or number of lactate producing bacteria present at the time of drenching. There could also be differences in the amount of the liquid drench which passes directly to the omasum and abomasum.

It is unclear why the D-lactate levels in sheep were increased by drenching with virginiamycin. Considering that drenching sheep with virginiamycin has prevented acidosis, this increase in D-lactate levels does not appear to be a factor in terms of whether or not acidosis occurs. This may be due to the increase in D-lactate being smaller than the reduction in L-lactate.

The variability in pH and lactate production in cattle is likely to be due to contamination with saliva. The pH of rumen digesta was a significant covariate for analysis of D-lactate production on days 1-3 after dosing.

Previous experiments (Thorniley *et al.* 1996) have found a reduction in feed intake in response to a single drench of virginiamycin; however this was not observed in either of these experiments. This may be due to the restricted amount of feed offered hiding differences in voluntary feed intake.

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