

# Prevention of Acidosis and Laminitis Associated with Grain Feeding in Horses<sup>1</sup>

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## EXPANDED ABSTRACT

### Indexing Key Words:

• horses • acidosis • laminitis

It is well known that if horses consume large quantities of rapidly fermentable carbohydrate in the form of starch or sugars they may develop laminitis. The pathway linking fermentable carbohydrate intake and damage to the lamellae of the hoof is not well understood. It is, however, almost certain that the primary effect of high levels of sugars and starches in the gut is a rapid fermentation and the development of acidic conditions particularly in the cecum and colon. Severe acidosis in the gut, characterized by the accumulation of lactic acid and reduced concentration of volatile fatty acids, does not occur uniformly in all animals given an equal amount of grain (Aitchison et al. 1987). This variation between animals in the development of acidosis within the gut in response to high levels of grain is similar to the variation between animals in development of laminitis (Garner et al. 1977) in response to carbohydrate overload. This provides further indirect evidence that gut acidosis may be the primary event leading to laminitis.

The accumulation of lactic acid during rapid fermentation can be controlled by antimicrobial feed additives active against the gram positive bacteria such as *Streptococcus bovis* and *Lactobacillus sp.*, which are primarily responsible for production of lactic acid at low pH. The feed additive virginiamycin has been shown in vitro (Nagaraja et al. 1987) and in studies in sheep (Godfrey et al. 1992) to be effective in controlling the accumulation of lactic acid. We have developed a formulation of virginiamycin for horses that retains its activity in the hindgut and, through its particulate nature, is also retained in the cecum. The aim of the experiment reported here was to investigate the control of laminitis using this formulation in horses given free access to a high-grain diet.

**Materials and Methods.** Twelve standardbred horses weighing an average of  $496 \pm 13$  kg (mean  $\pm$  SE) were used in the experiment. These horses came from a background of training and harness racing and were of unknown age. Before the start of the experiment, all animals were given a detailed clinical and lameness examination and were treated against intestinal parasites. Only animals that were completely sound and in good health were used in the experiment.

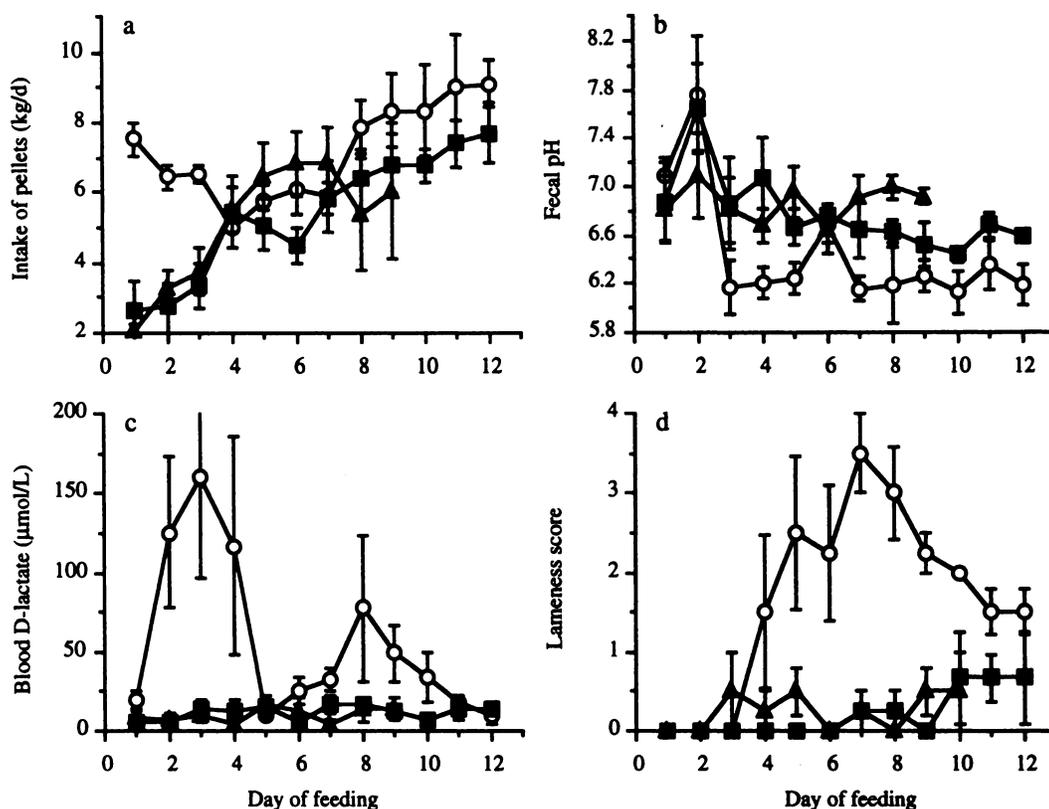
There were three dietary treatments and four horses per treatment. The three groups were balanced for body weight. The three pelleted diets contained 85% ground maize, 13% soybean meal and 2% minerals and vitamins providing 13.9 MJ/kg digestible energy and 120 g/kg crude protein (Glen Forrest Stockfeeders, Perth, Australia). Two of the diets contained a feed additive containing virginiamycin (as Founderguard<sup>TM</sup> Granular Feed Additive for Horses, Hancroft, PO Box 972, Armidale NSW, Australia) to provide either 4 or 8 g feed additive/kg feed. The third control diet contained no virginiamycin. The pelleted portion of the diet was fed twice each day (08:00 and 16:00). On the first day of feeding, 8 kg of pellets was offered to each animal and thereafter horses had free access to the pelleted feed for the remaining 11 d of the trial. Long hay (2 kg/d) was fed each morning. The treatment with the lower level of virginiamycin was only fed for 9 d because of other demand for the stables.

All horses were stabled throughout the experiment and exercised twice daily in conjunction with their

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**FIGURE 1** Intake of the pelleted portion of the diet (a), changes in fecal pH with time (b), changes in blood D-lactate concentrations (c) and average lameness score (d) of horses fed the control unmedicated diet (O) and pelleted diets containing virginiamycin at either 4 (▲) or 8 (■) g granular feed additive/kg pelleted feed. Error bars show the standard error of the mean.

lameness examination. We gave each horse a detailed clinical examination each morning, including measurements of digital pulse, temperature, heart and respiration rates. At this time samples of blood were taken for analysis of D-lactic acid and samples of feces were collected for measurement of pH. Body weight was measured daily. Lameness was assessed by a veterinarian who was not aware of the animal's identification or its diet. Lameness was described on a scale of 1 (just detectable at trot), 2 (clearly lame at trot), 3 (detectable when walking and clear at trot) or 4 (clearly lame when walking).

D-lactate was determined enzymatically in neutralized perchloric acid filtrate of blood using a modification of the L-lactate assay described by Noll (1985). Modifications included the use of D-lactate dehydrogenase (Boehringer Mannheim Australia, Castle Hill, Australia) and an increased incubation time.

**Statistical methods.** Differences between treatments on individual days and for different periods of the trial (e.g., entire experimental period, days 1–4, days 6–9 and days 6–11) were analyzed using analysis of variance. The program SuperAnova (Abacus Concepts, Berkeley, CA) was used for all analyses.

**Results.** The intake of the pelleted portion of the diet is shown in **Figure 1a**. The horses fed pellets

containing virginiamycin had a lower intake of pellets than those fed the unmedicated control diet, particularly during the first 3 d. By the fourth day of the experiment, the average intake of the pellets was ~8 kg/d and increased still further during the remainder of the experiment. Changes in blood D-lactate concentrations with time are shown in **Figure 1c**. In the control animals there were two peaks of D-lactate, one after 3 d on feed and the other after 8 d. The intake of pellets by this group decreased during the initial peak in D-lactate. In animals fed virginiamycin there was not a significant increase in the concentration of D-lactate during the period of feeding. As shown in **Figure 1b**, the fecal pH in samples taken from animals fed pellets containing virginiamycin was significantly higher ( $P < 0.05$ ) than the control group over the duration of the experiment. Whereas feces in the control animals were soft and unformed, the feces of the horses given virginiamycin were normal. **Figure 1d** summarizes the incidence of lameness during the experiment. From day 4 onward all of the control horses were diagnosed as being moderately to severely lame at the trot. This was compared with one or two of the animals given virginiamycin showing signs of mild lameness at varying times during the trial. There was a significant effect of treatment ( $P < 0.001$ )

on the incidence of lameness with animals given virginiamycin showing little or no evidence of lameness.

The control horses lost  $35.5 \pm 5.1$  kg during the first 6 d of feeding the maize-based pellets compared with losses of  $10.5 \pm 1.3$  and  $11.0 \pm 3.3$  kg for the groups given lower and higher levels of virginiamycin, respectively.

**Discussion.** It is likely that virginiamycin acted in two ways to control the build up of lactic acid and the development of laminitis in this experiment. First, by reducing the intake of pellets during the first 3 d of feeding, it reduced the amount of starch available for fermentation. Second, through specific inhibition of microbes producing lactic acid under acidic conditions, it prevented the development of very low levels of pH in the hindgut. Virginiamycin has been shown to reduce feed intake in the first 1–3 wks of administration in cattle (Zorrilla-Rios et al. 1991) and sheep (Murray et al. 1992). The reduced feed intake in horses given virginiamycin in this experiment is therefore not surprising, but the mechanism by which virginiamycin causes a transitory decrease in feed intake is not known.

Despite a lower feed intake over the first 6 d of feeding, the horses fed virginiamycin lost significantly less weight than the control group. Some weight loss was expected in horses in all treatment groups in response to the increase in overall digestibility resulting from pellets replacing hay in the diet and the effect this has on gut fill. In the case of the control group, it was likely that the weight loss (~35 kg) in the first week would have been due to mild diarrhea and the abnormal effect this can have on gastric emptying and water loss. Diarrhea can be caused through accumulation of lactic acid in the hindgut and weight loss through diarrhea is therefore consistent with the elevated blood D-lactate levels observed in animals in

this treatment group. The inhibition of lactic accumulation in hindgut digesta is likely to have a significant effect on fecal pH and could explain the differences in fecal pH between the fecal pH in the control group and those horses treated with virginiamycin.

We conclude from this study that virginiamycin, in the appropriate formulation as Founderguard™ Granular Feed Additive for Horses, may be useful in the nutritional management of horses fed high levels of cereal grain. Even at the lower dose rate, virginiamycin was effective in maintaining a higher, and more normal, level of fecal pH and low blood D-lactate concentration. These effects on fermentation and digestion were effective in reducing the risk of laminitis.

## LITERATURE CITED

- Aitchison, E. M., McDonald, C. L., Casson, P. & Rowe, J. B. (1987) Feed antibiotics and buffers to control rumen lactic acid. 2. Pre-dosing. *Proc. Nutr. Soc.* 46: 41A (abstr.).
- Garner, H. E., Hutcheson, D. P., Coffman, J. R., Hahn, A. W. & Salem, C. (1977) Lactic acidosis: A factor associated with equine laminitis. *J. Anim. Sci.* 45: 1037–1041.
- Godfrey, S. I., Boyce, M. D. & Rowe, J. B. (1992) Changes within the digestive tract of sheep following engorgement with barley. *Aust. J. Agric. Res.* 44: 1093–1101.
- Murray, P. J., Rowe, J. B., Aitchison, E. M. & Winslow, S. G. (1992) Liveweight gain and wool growth in sheep fed rations containing virginiamycin. *Aust. J. Exp. Agric.* 32: 1037–1043.
- Nagaraja, T. G., Taylor, M. B., Harmon, D. L. & Boyer, J. E. (1987) In vitro lactic acid inhibition and alterations in volatile fatty acid production by antimicrobial feed additives. *J. Anim. Sci.* 65: 1064–1076.
- Noll, F. (1985) L-(+)-lactate determination. In: *Methods of Enzymatic Analysis*. Vol. VIII, 3rd ed. (Bergmeyer, H. V., ed.) pp. 582–588. VCH Verlagsgesellschaft, Weinheim, Germany.
- Zorrilla-Rios, J., May, P. J. & Rowe, J. B. (1991). Rapid introduction of cattle to grain diets using virginiamycin. In: *Recent Advances in Animal Nutrition in Australia* (Farrell, D. J., ed.), p. 10A. University of New England, Armidale, Australia.