Controlling Acidosis in the Equine Hindgut

J.B. Rowe, D.W. Pethick* and K.G. Johnson*

Department of Animal Science, University of New England, Armidale, NSW 2350 Australia
**School of Veterinary Studies, Murdoch University, Murdoch, WA 6150

Summary

When cereal grain is fed to horses there is a risk of laminitis and it is also common to find behavioural changes characterised by chewing wood, eating bedding and grasping (wind sucking). This paper discusses the link between acidity in the gut, as a result of grain feeding, and the risk of laminitis and adverse behaviour. A proportion of the starch consumed as cereal grain passes undigested to the caecum and colon where it is rapidly fermented. The process of fermentation can lead to very low pH which is characterised by the presence of lactic acid. The feed additive virginiamycin (as the granular formulation Founderguard™) is effective in controlling the pattern of acid build up and accumulation of lactic acid even in horses given high levels of grain. The control of acidic conditions in the hindgut reduces the risk of laminitis associated with grain-based diets and prevents some of the behavioural changes associated with feeding grain supplements to horses.

Introduction

The equine gut can make up around 20% of the animal's liveweight with most of this weight in the caecum and colon. These are very large organs and sites of active fermentation. The contents of the hindgut are not buffered by saliva in the same way as rumen contents and are susceptible to the accumulation of organic acids associated with rapid fermentation of sugars and starch. In this paper we describe a method of controlling the development of acidic conditions in the hindgut and the consequences that this has on the disorders normally associated with feeding starch and soluble carbohydrate to horses and ponies.

There are a number serious disorders associated with horses and ponies consuming high levels of cereal grain or soluble carbohydrates present in lush green pasture. The most serious of these is founder or laminitis which can be caused by a number of different factors but is most commonly associated with the consumption of large quantities of high-starch grain or in animals grazing lush green pastures in spring. Other conditions associated with grain feeding and very lush pastures include behavioural changes such as "***fizzyness***", and eating bedding or wood. While the consequences of grain feeding are diverse it is almost certain that the primary effect is the development of acidic conditions in the gut and our research has focussed on this aspect.

The accumulation of acids and low pH in the gut

The rapid ferrnentation of carbohydrate in the caecum and large colon results in high levels of volatile fatty acid (VFA) production and a rapid decrease in pH. Under these conditions, of low pH, lactic acid can accumulate since the more acidic environment favours those organisms producing lactic acid and is unfavourable for the bacteria which utilise lactic acid. Once lactic acid starts to accumulate the pH falls even further and, if there is sufficient substrate to continue the fermentation very acidic conditions can occur (Godfrey et al 1993)

The acidic conditions in the large intestine may initiate secondary effects in a number of ways. The acidity causes irritation and damage to the intestinal mucosa. In addition, the drop in pH is known to cause bacterial lysis and, associated with this, a release of endotoxins which can be absorbed and are active at the tissue level. The acidic conditions in the large intestine may also affect the animal through systemic acidosis. Any one, or all, of these factors could initiate the biological pathway(s) leading to laminitis.

What is the significance of D-lactic acid?

Conditions of very low H in the caecum and colon do not normally occur without accumulation of lactic acid. Therefore, while it appears unlikely that lactic acid itself is an important trigger in the chain of events leading to laminitis, it is a very important indicator of an acidic pattern of fermentation in the gut. There are two isomers of lactic acid which are produced in almost equal proportions by bacteria in the gut. These are L- and D-lactate. On the other hand, only one of these isomers, L-lactate, is produced by body tissue and can be measured at elevated levels in blood during strenuous exercise or in response to pain or stress.

There is no D-lactate present in the blood unless there is a significant accumulation of lactic acid in the gut. For this reason, measurable amounts (μ moles/L) of blood D-lactate always provides an accurate indication of abnormal fermentation/digestion and very acidic conditions in the hind gut. For these reasons the control of lactic acid accumulation during carbohydrate fermentation is an important step in preventing excessive acidity. As part of our studies we have used the in vitro fermentation of caecal, colonic and faecal material with excess glucose as a way of measuring the capacity of the digesta to produce lactic acid in the presence of readily fermentable carbohydrate. The hypothesis behind this has been that if we can develop methods to control lactic acid accumulation then we will have a basis for preventing conditions of very low pH in the gut.

Control of lactic acid accumulation and development of Founderguard

The main lactate-producing bacteria are Streptococcus bovis and *Lactobacillus spp* . *These* are gram +ve organisms which may be controlled through the use of a range of antibiotic compounds. There are a number of these compounds which we have examined in some detail. The data in Table 1 summarises the effects of different doses of two antibiotic feed additives on lactic acid accumulation, volatile fatty acid concentrations and pH when hind gut digesta was fermented with corn starch. These data show the control of lactic acid production, particularly with virginiamycin, and the effect that this has on pH.

Being able to control lactic acid during in vitro incubation is an important starting point but the important test is the control of lactic acid in vivo in the hind gut. Two things are important in this regard: getting the compound to the hindgut in an active form; and secondly, to ensure that it enters the caecum and mixes with digesta in that organ. The data in Table 2 shows that avoparcin given as the fine powder pre-mix does not enter the caecum in sufficient concentration to control lactic acid accumulation in horses given high levels of ground wheat. While the avoparcin was present in sufficient concentrations in the colon to reduce lactic acid accumulation, this was not the case in caecal digesta. All of the animals used in this experiment which had high levels of lactate in the digestive tract developed signs of lameness within 24 hours of dosing with the ground wheat. The one horse which did not show signs of lameness had significantly lower concentrations of lactate than the others. The three animals showing signs of lameness all had high levels of D-lactate in the blood.

The results of this experiment emphasised the importance of delivery into the caecum and we have devoted considerable effort to develop an appropriate formulation to ensure that this occurs. Founderguard is a granular product which is palatable and can be mixed with feed or fed on its own. The dose rate is 5 g per 100 kg body weight and this small amount is easily fed out of the hand.

Efficacy of virginiamycin (as Founderguard) in acute carbohydrate overload

Eight standard bred horses were used for this experiment. Four animals received Founderguard and 4 animals received the same treatment but without Founderguard. The animals which received Founderguard were pre-dosed for two days prior to

Table 1 Effect of the antibiotics avoparcin and virginiamycin on pH, L-lactate and VFA during the incubation of mixed caecal/large colon digesta with buffer and cornstarch (15 mg/ml).

Antibiotic	pH		Lactate		Total VFA		Acet:Prop	
(μg/ml)	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Avoparcin								
0	5.34	0.042	46.8	2.93	97.5	6.56	2.9	0.18
2	6.14	0.054	8.9	1.04	102.6	1.92	2.9	0.05
4	6.17	0.017	12.0	2.41	100.9	2.12	2.8	0.06
8	6.33	0.006	11.0	0.42	82.1	4.92	3.3	0.06
16	6.40	0.017	10.3	1.40	87.1	2.49	3.5	0.15
32	6.43	0.012	15.1	0.57	89.8	2.33	3.6	0.16
Virginiamycin								
0	5.34	0.042	46.8	2.93	97.5	6.56	2.9	0.18
2	6.57	0.009	0.2	0.05	93.2	5.15	2.9	0.10
4	6.63	0.003	0.2	0.07	80.8	8.99	3.1	0.07
8	6.66	0.009	0.2	0.10	79.7	8.75	3.2	0.03
16	6.64	0.010	0.0	0.01	71.3	10.59	3.8	0.23
32	6.66	0.009	0.1	0.04	87.1	0.74	3.7	0.08

administration of the wheat slurry. The doses of wheat administered on the third day provided 12 g ground wheat/kg liveweight. This was given in two equal amounts with an interval between doses. The animals were examined at 8 hour intervals over a period of 48 hours. Each examination included temperature, heart rate and respiration in addition to assessment of lameness by an independent operator and samples for analysis of blood gases, D-lactate and faecal pH. After 48 hours all animals were euthanased.

Table 2 Concentrations of D- and L- lactic acid isomers and avoparcin in the caecum and large colon of three horses 24 hours after administration of ground wheat (15 g/kg liveweight). Also shown is whether or not the animals developed signs of lameness.

	Control		Avoparcin	
Avoparcin (mg/kg wheat)	0	0	120	120
Caecum				
L-lactic acid (mmol/L)	0.04	32	33	37
D-lactic acid (mmol/L)	0.0	35	33	34
avoparcin (µg/g digesta)	0	0	17	15
Large colon				
L-lactic acid (mmol/L)	1.4	27	2	10
D-lactic acid (mmol/L)	1.0	34	2	11
avoparcin (µg/g digesta)	0	0	30	25
Lameness	none	Obel 2	Obel 2	Obel 2

The results are summarised below in Figures 1. These show that treatment with Founderguard prevented accumulation of lactate in the gut (as shown in blood D-lactate levels) and this had an important effect on blood gases and pH.

Of the animals treated with Founderguard there was no animal which showed any signs of lameness. In contrast three out of the four control animals which received no Founderguard developed Obel grade 2 lameness (Obel 1948). There was therefore a significant ($p<0.05$) reduction in lameness associated with the use of Founderguard. The assessment of lameness by physical examination was confirmed by histological examination of hoof sections. There was a close correlation ($p<0.002, R^2=0.94$) between lameness assessed according to the Obel grades and the incidence and severity estimated by histological examination by Dr C. Pollitt.

Use of Founderguard in a grain-based diet

Having established that Founderguard is effective in preventing the development of laminitis following acute carbohydrate overload, the following experiment was conducted to study its use under conditions which might more closely reflect a practical situation. However to test the product as thoroughly as possible the diet selected for this experiment had higher quantities of grain than would be used under normal circumstances. Two dose rates of Founderguard were investigated. These concentrations represented the recommended dose rate for Founderguard and twice the recommended level (Rowe et al 1994).

There were three dietary treatments and four horses per treatment. The three pelleted diets contained 85% ground maize, 13% soybean meal and 2% minerals and vitamins. Two of the diets contained 4 and 8 g Founderguard/kg feed. The third "control" diet contained no Founderguard. These pelleted diets were fed in a single feed each morning. Long hay (2 kg/d) was fed each morning. All animals were given a detailed clinical examination each morning and samples of blood were taken for analysis of D-lactic acid. Samples of faeces were collected at the same time for measurement of pH. As part of the daily physical examination, lameness was assessed by a veterinarian who was not aware of the animal's identification or the dietary treatment each animal was receiving. Lameness was described on a scale of 1 (hardly noticeable) to 4 (clearly identified when walking). Most of the lameness was classified as 2 or 3 indicating either mild or sever lameness while trotting.

The intake of the pelleted portion of the diet is shown in Figure 2. The horses fed pellets containing Founderguard had a lower intake of pellets than those fed the unmedicated control diet, particularly during the first 3 days. By the fourth day of the experiment the average intake of the maize-based pellets was approximately 8 kg/d and increased still further during the remainder of the experiment.

Changes in blood D-lactate concentrations with time are summarised in Figure 3. In the control animals there were two peaks of D-lactate, one after three days on feed and the other after 8 days. The drop in lactate concentration during days 5, 6 and 7 correspond to reduced pellet intake compared days 1 to 3

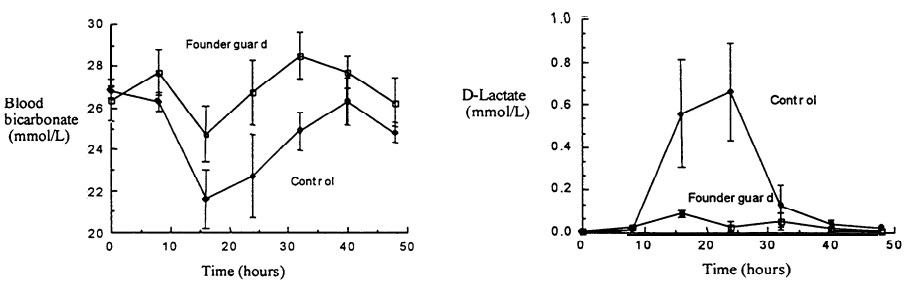


Fig 1 The changes with time in D-lactate and blood bicarbonate measured following dosing with ground wheat. The horses in one group were treated with Founderguard and those in the other received no medication (Control).

and from 8 to **12.** In animals fed Founderguard there was not a significant increase in the concentration of D-lactate during the period of feeding.

Figure 4 summarises the incidence of lameness during the experiment. From day 4 onwards all of the control horses were diagnosed as being moderately to severely lame at the trot. This compared with 1 or two of the animals given Founderguard 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 Founderguard having little or no evidence of lameness. It is possible that some animals were diagnosed as being lame for reasons other than laminitis of alimentary origin but the difference between treatments indicates a lameness in the control group to result from the level of carbohydrate in the diet and the elevated D-lactate and low faecal pH resulting form starch fermentation in the absence of Founderguard.

It is likely that Founderguard may act in two ways to control the build up of lactic acid and the development of laminitis. Firstly through controlling the intake of pellets during the first three days of feeding and secondly through its specific affect on lactate accumulation. Even when intake of pellets with Founderguard increased to a level similar to that of the control group (days 7 and 8) there was no evidence of a rise in lactic acid. The effect of Founderguard on feed intake in the first week of feeding has been well documented in cattle and sheep Murray et al, 1992) and is therefore not unexpected under these conditions. The temporary reduction in feed intake is seen as providing additional protection to the animal during the critical period of introduction to high levels of readily fermentable carbohydrate.

Possible therapeutic application of Founderguard

Even after acute carbohydrate overload it takes over 12 hours for lactic acid to accumulate and around 24 hours for signs of lameness to be detected. For this reason it was thought possible that animals which eat too much grain through gaining access to the feed store, or other such accident, may be able to be treated with Founderguard as soon as the accident is detected. We carried out an experiment to investigate this possible use of Founderguard (J. Rowe, D. Pethick and K. Colbourne). In this experiment animals were given Founderguard either for three

Figure 2 Intake of a the pelleted portion of the diet containing no medication, or Founderguard at concentrations of 4 or 8 g/kg.

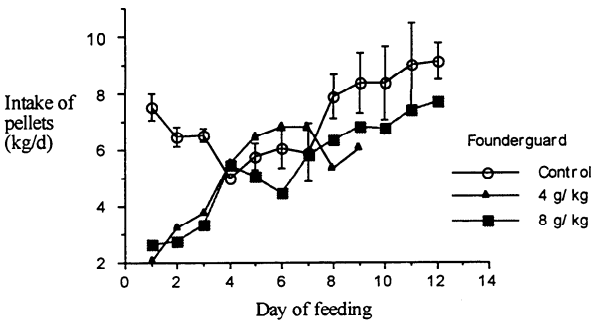


Figure 3 The changes in blood D-lactate concentrations with time measurd in horses fed a diet containing 85% ground maize with or without Founderguard at concentrations of 4 or 8 g/kg.

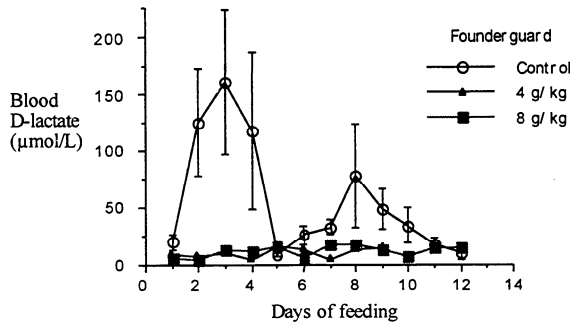
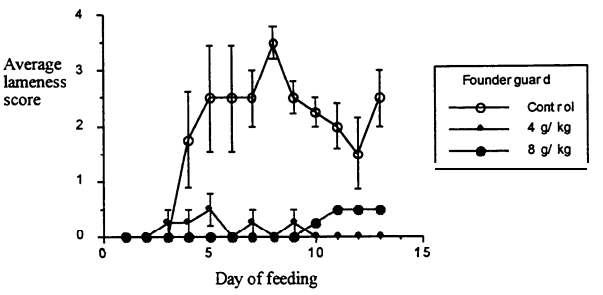


Figure 4 Average lameness score of horses fed the control unmedicated diet and the two diets containing Founderguard at 4 and 8 g/kg. Error bars show the standard error of the mean.



days before a carbohydrate overload challenge or approximately 6 to 8 hours after they had first received a dose of ground corn. While the animals treated with Founderguard before carbohydrate overload were all protected from high levels of lactic acid and laminitis those treated afterwards went on to develop problems associated with acidosis and laminitis in the same way as untreated controls. The results of these studies are summarised in Table 4.

Low gut pH and behavioural changes

In a recent experiment (J. Tyrell, K. Johnson, D. Pethick and J. Rowe, unpublished) eighteen mature Standardbred horses were selected on the basis that they showed no signs of lameness and that they had no obvious unusual behavioural characteristics. They were assigned at random to one of three treatment groups (6 per treatment) summarised in Table 5.

The "grain" portion of the diet consisted of a pellet containing: Wheat (72%); Soybean (15%); Lupin (10%) and Minerals/vitamins (3%). The hay was fed in the long form (not chaffed). All animals ate all of the feed offered in two equal feeds in the morning and afternoon.

For the week before the experiment started all of the horses were observed daily when grazing as a single group to determine if there were any abnormal (background) behavioural patterns. The horses were then brought into a stable complex with a high overhead walk way from which all of the animals could be observed without being disturbed. Behaviour was observed and quantified during a 2 hour session each morning before feeding and a 1 hour session each evening after feeding. Every aspect of behaviour was accurately defined before the experiment started and each incident of every type of behaviour was recorded during the periods of observation.

In addition to the observations while the animals were in their stalls they were exercised each day and examined for any signs of lameness. Samples of faecal material were taken for analysis of pH. Blood samples were also taken for measuring pH, blood gas concentrations and lactic acid. The animals were weighed each day. At the end of the experiment all animals were humanely slaughtered in order to take samples of the digestive tract. During this process the digestive tract was weighed.

There were marked changes in the behaviour of horses fed increasing levels of grain without Founderguard. During the same time the behaviour of the horses maintained on hay only remained normal indicating that the development of abnormal behaviour was a result of change in diet rather than boredom at being housed in a stable. The behaviour of horses fed grain with Founderguard was marginally, but not significantly different from those fed hay. In

Table 4 The effect of virginiamycin (as Founderguard) given either as a prophylactic for three days prior to carbohydrate overload or therapeutically, 6 hours after carbohydrate overload. Histological changes were graded from 0 (no damage) to 3 (severe damage to both primary and secondary lamellae) and values are averages per animal for the treatment groups. (J.B. Rowe, D.W. Pethick, K. Colboume, and M. Lees, unpublished)

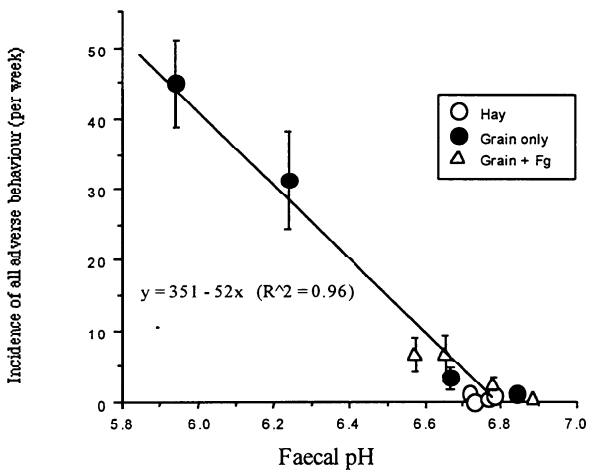
Treatment	Obel grade lameness	Lamellar histological changes	Blood D-lactate µmol/L	Faecal pH
Control (n = 8)	1.38	1.05	81	5.8
"Therapeutic" (n = 5)	0.81	1.00	29	5.5
"Prophylactic" (n = 8)	0.00	0.16	8	6.4

Table 5 Feed intake (kg/d) of horses fed hay alone, or hay with increasing levels of grain-based pellet with or without the addition of virginiamycin (as Founderguard)

Treatment group	Week 1	Week 2		Week 3		Week 4	
	Hay	Hay	Grain	Hay	Grain	Hay	Grain
Hay only	8	8		8		8	
Hay and grain	8	6	2	4	4	2	6
Hay and grain with Founderguard	8	6	2	4	4	2	6

animals fed grain without Founderguard there was a significant decreased in faecal pH with increasing levels of grain in the diet. The faecal pH of horses fed grain with Founderguard was similar to those fed hay. The incidence of adverse behaviour was closely related to faecal pH (r^2=0.96) (see Figure 5). These results are consistent with those of Willard et al (1976) who reported that behaviour such as wood chewing and eating bedding was closely related to the concentration of lactic acid in caecal contents and negatively related to caecal pH.

Figure 5 Relationship between the sum of all incidents of adverse behaviour (wood chewing, eating bedding and grasping) per horse during the total 14 hours of observation during the week, and the average faecal pH during the corresponding week. (J. Tyrrell, K. Johnson, D. Pethick and J. Rowe, unpublished).



Although the animals fed grain consumed higher levels of digestible energy there was an average weight loss of around 7 kg compared to the horses fed only hay. This difference in liveweight was not statistically significant but the corresponding reduction in the weight of the gut of around 20 kg, in horses fed increasing levels of grain relative to hay, was highly significant.

The results in Table 5 demonstrate the potential advantages of feeding grain based supplements to horses in racing or performing strenuous exercise. Grain feeding facilitates a high level of energy intake while at the same time reducing the weight of the gut contents which results in less bulk for the horse to carry as it races, jumps or carries its rider round the polo field. Feeding more grain in the diet improves the "energy to weight ratio" and it is an attractive option in the nutritional management of performance horses. However, feeding high levels of cereal grain involves risks such as laminitis and behavioural changes and this highlights the potential usefulness of a product such as Founderguard in the nutritional management of the equine.

Conclusions

Many of the effects of feeding grain to horses are complicated by the fact that there is an increase in the amount of digestible energy intake, a reduction in the amount of "bulk" consumed and the different pattern of fermentation and digestion within the gut. The use of virginiamycin (as Founderguard) allows a study of the effects of acid build up in the gut without altering the amount of energy available or affecting the physical nature of the diet. The results of these studies indicate that the adverse effects such as laminitis and behaviour changes, associated with feeding cereal grain to horses, can be overcome by controlling the build up of acidity in the hindgut. This finding questions the generally held view that adverse behaviour of horses housed in stables and fed diets high in grain is a result of insufficient fibre the diet and boredom. It also provides a better understanding of laminitis and offers a method for safer grain feeding in the nutritional management of horses which require additional digestible energy. Unfortunately it does not appear to have application in treating animals once they have eaten excessive levels of grain. There is yet only circumstantial information suggesting that it may be useful in the management of horses and ponies on lush pasture but there appears to be significant potential for this use.

References

Garner, H.E., Hutchison, D.P., Coffman, J.R., Hahn, A. W., and Salem, C. (1977) Lactic acidosis: a factor associated with equine laminitis. J. Anim. Sci. 45: *1037*-1041.

Table 5 Summary of changes in liveweight of horses during the final 3 weeks of the experimental period and the weight of the gastrointestinal tract at the end of the experiment. (J. Tyrell, K. Johnson, D. Pethick and J. Rowe, unpublished)

	Hay	Grain only	Grain + Fg	Signif (P)
Average weight change (kg/21 d)	0.7	-7.0	-7.5	0.2
Weight of gut (kg)	86	63	68	0.0002
Weight of gut as % of live weight	18	13	15	0.0001

Nagaraja, T.G.A, Avery, T.B., Bartley, E.E., Galitzer, S.J. and Daeyton, A.D. (1981). Prevention of lactic acidosis in cattle by lasalocid or monensin. J. Anim. scj.53: 206-2 15.

Obel, N. (1948) Studies on the histopathology of acute laminitis. Almqvist and Wiksells Bocktryckeri ab Uppsala.

Willard, J.G., Willard, J.C., Wolfram, S.A. and Baker, J.P. (1976) Effect of diet on fecal pH and feeding behaviour of horses. Journal of Animal Science 45: 87-93.

Rowe, J.B. Lees, M.J. and Pethick, D.W. (1994) Prevention of acidosis and laminitis associated with grain feeding in horses. Journal of Nutrition 124: 2742-4S

Godfrey, S.I., Boyce, M.D. and Rowe, J.B.(1993). Changes within the digestive tract of sheep following engorgement with barley. Aust. J. Agric. Res. 44: 1093-98.

Murray, P.J., Rowe, J.B. and Speijers, E.J. (1992). Lactic acidosis and its affect on wool tensile strength. Aust. J. Agric. Res. 43:1747-1758.