

Changes in hindgut pH of ponies following feeding with fructan carbohydrate in the form of inulin

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Introduction

Fructans, the main storage form of carbohydrates found in many grasses, have been implicated in causing changes in the equine hindgut leading to the development of acute equine laminitis (Longland et al. 1999). The production of lactic acid, increasing the permeability of the large intestinal mucosa to toxins and larger molecules (Weiss et al. 2000), is thought to be important in this process. Understanding the quantities of fructan carbohydrate necessary to cause significant changes in hindgut pH will provide information which will be helpful in assessing the likely effects of grazing different forages on hindgut bacterial fermentation. The objective of this study was firstly to determine the concentration-dependent effect of inulin, a form of fructan, on caecal pH using an in vitro model. Secondly, an in vivo study was conducted where controlled amounts of inulin were fed to normal ponies and ponies predisposed to laminitis, to determine whether any differences would be seen in the hindgut pH changes.

Materials and Methods

Firstly, equine caecal contents, obtained from ponies euthanased for purposes unrelated to the study, were incubated in an anaerobic environment in vitro at 37°C (Mk 3 anaerobic work station, Don Whitley Scientific Ltd., Basingstoke, UK) in several 100 ml aliquots. No supplemental buffer was included. Inulin was added to the aliquots in quantities ranging from 0-10 g/litre, and the pH was measured at 6 hour intervals for 24 hours.

For the in vivo studies, 11 non-obese adult native breed ponies were used, 5 normals and 6 ponies predisposed to laminitis, but not affected by the condition at the time of the study. Following an initial period of 2 weeks on a basal hay diet, inulin was then fed at 1g or 3g /kg bodyweight (BW), fed either once daily (1g/kg) or split into two equal feeds (3g/kg). Hay was provided ad lib, and a high protein dried grass (15% protein) was fed as one third of the forage ration, to provide an amino acid source which would mimic that present in lush grass. Faecal pH was measured twice daily throughout the study period.

Results

Inulin produced a dose-dependent decrease in the pH of caecal contents in vitro, from a baseline of 7.0 ± 0.2 to a minimum of 4.9 ± 0.3 , when inulin was added at 10 g/litre (Figure 1). In the in vivo study, none of the animals showed signs of laminitis or any other clinical problem. When inulin was fed at 1 g/kg BW, faecal pH decreased from 7.0 ± 0.1 to 6.4 ± 0.1 after 3 days (Figure 2), and at 3 g/kg BW, the pH decreased from 6.8 ± 0.1 to a minimum of 6.3 ± 0.1 (Figure 3). There were no significant differences between the normal ponies and those predisposed to laminitis.

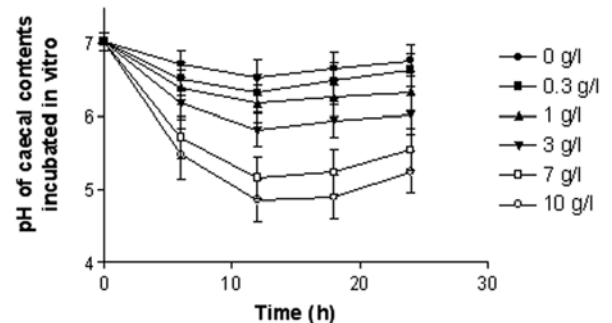


Fig 1 Effect of addition of inulin on pH change in equine caecal contents.

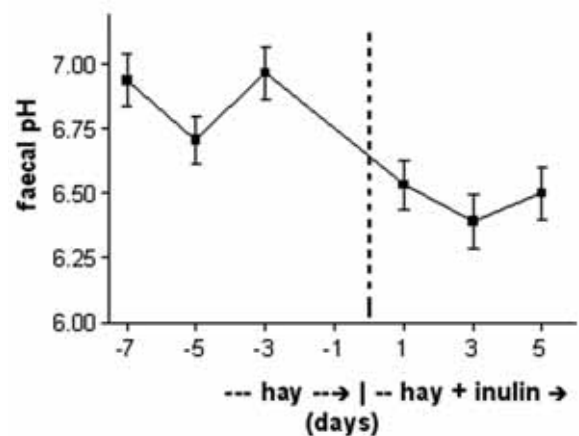


Fig 2 Effect of feeding inulin to ponies, at 1g/kg bodyweight, on faecal pH.

Discussion

These data demonstrate the changes in hindgut pH in response to the addition of varying amounts of fructan carbohydrates in vitro and in vivo. Feeding inulin at 3 g/kg produced a slightly lower minimum faecal pH value, compared with feeding 1g/kg, although this was not significantly different. This amount of inulin is well below the amount reported to cause laminitis by bolus administration (greater than 7.5 g/kg; French and Pollitt 2004). However, this model may mimic some of the changes in carbohydrate fermentation by hindgut bacteria following grazing of ponies on lush grass.

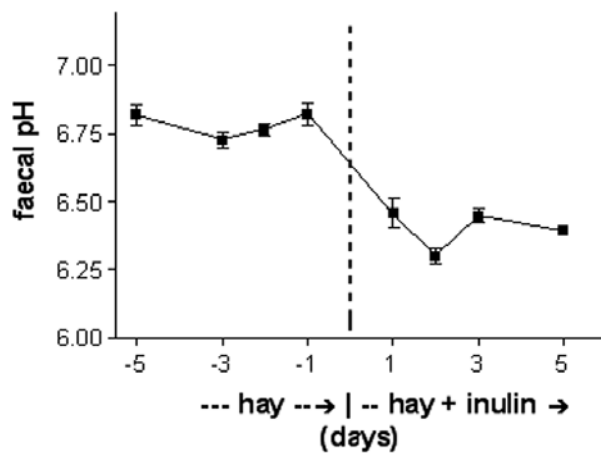


Fig 3 Effect of feeding inulin to ponies, at 3 g/kg bodyweight, on faecal pH.

Conclusions

There was no evidence of differences in faecal pH in ponies predisposed to developing laminitis, compared to normal ponies. Therefore, production of lactic acid in the hindgut per se, may not be the only factor which renders particular animals prone to laminitis. Other factors may be involved, for

example the production, absorption, and activity of other bacterial toxins, such as amine compounds (Bailey et al. 2002).

References

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