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FIBRE-BEET MASH & ULCERS

Ulceration is an industry wide problem. Clinical signs of ulcers include colic, diarrhoea, poor appetite, teeth grinding, salivation and poor physical performance. However, in many cases these signs are missing, and the only way of detection is through gastroscopy. Unfortunately, as the name implies, this can only investigate the stomach and it is becoming increasingly apparent that ulcers occur along the whole length of the gut.

Ulceration is caused by a number of factors but the two main culprits are excessive acid in the stomach and stress, with the former impacting on the latter.

The horse continuously generates acid from the fundus layer of the stomach. Under normal conditions this is partially neutralised by saliva produced by chewing feed, with excess acid absorbed onto the fibrous material entering the stomach.

However, with an exercising animal, fed high levels of concentrate and with limited access to grazing this can lead to two ulcerative conditions.

Squamous Gastric Ulcer Disease (SGUD)

This occurs in up to 90% of performance horses and the reason is fairly obvious. The stomach of the horse has two areas, separated by a band. The upper layer – the squamous mucosa – does not contain any secretory cells and is not fully protected by the mucus lining that covers the fundus mucosa – the secretory area. Feed with a high starch or cereal content does not stimulate much saliva production or absorb much acid. Also, as it is fed in discrete meals, there are periods where there is nothing to bind the acid. So when we exercise our horses, acid sloshes up into the squamous region and burns the stomach lining. It sounds crude but that is what happens.

Equine Gastric Ulcer Syndrome (EGUS)

This is more complex. Feeding high levels of starch is a major cause. Bacteria in the stomach ferment the starch forming lactic acid which increases the acidity of the stomach and this encourages the growth of acid-loving bacteria such as heliobacter. They can penetrate the mucus lining – especially in areas where stressful conditions compromises mucin secretion – and infect the stomach wall. The infection leads to ulceration and this, more than SGUD, can lead to perforation. Additionally, releasing a highly acidic mix into the small intestine can overwhelm the buffering capacity of the gut and allow infection to progress along its length.

Treatment includes drugs to inhibit acid production, antacids and barrier protectants, such as sucralfate, that try to strengthen the mucus linings.

Pectins, especially those with high esterification, demonstrate high mucoadhesion along the whole gut, can stimulate mucin release and, in the case of beet pectin, produce emulsions that improve inclusion into the mucosal layer.

Alfalfa has been shown to have a positive effect in lowering acidity in the stomach, even when fed with concentrates, an effect that can last 6 hours.

Fibre-Beet contains these ingredients. It also contains phospholipids that improve emulsification. As such it may have a significant role to play in offsetting ulcers both in the stomach and the intestine.

And because it is a combination of three super-fibres, feeding Fibre-Beet alongside a high energy concentrate won't mean compromising energy, rather the prebiotic effects reported in research articles will maintain gut health and provide quality energy for the performance horse.

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Ulceration of the horse's digestive tract, discussed the role of fibre in the diet and how it has a role in combatting acid driven perforation of both the stomach and the gut linings. Since then there has been additional information that may explain why Fibre-Beet Mash has been perceived to help in the control of EGUS.

Primarily it was shown that the pectic substances, and emulsifiers present in Fibre-Beet Mash were substances found in proprietary brands and the main ingredients – beet pulp and alfalfa – had the ability to soak up excess stomach acid.

Ulceration

The stomach of the horse has two areas separated by the margo plitacus. The first section is the squamous mucosa and represents the non-glandular portion. It is identical to the lower oesphagus (gullet).

The second section, the glandular mucosa, is sub-divided into three and it is the second section – the fundus – that has all the glandular activity.

The parietal cells of the fundus produce the acid secretions, others such as zymogen cells releasing pepsinogen, and goblet cells releasing mucin. As such it is the glandular portion that has a protective layer of mucilage which protects it against acid burning.

In the normal course of events the majority of stomach acids occur in the squamous mucosa – as this is unprotected and it is usually associated with acid reflux that may be caused by a number of factors, including exercise.

However, an estimated 20% of gastric ulcers occur in the glandular portion and these are the result of microbial invasion of the stomach lining where mucus barriers are compromised. This is the result of a number of factors, including feeding regime, stress and insufficient fibre.

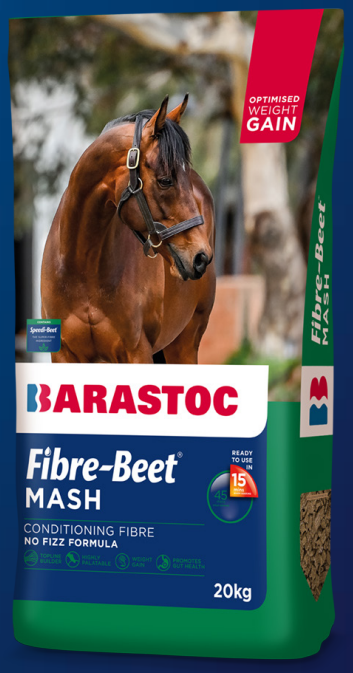
Occurrence

There are distinct differences between foals and yearlings/adults when it comes to ulcers. In foals most ulcers are sub-clinical and squamous, although they tend to show more glandular ulcers compared to adults. They also can suffer from perforations and peritonitis and pyloric stricture resulting in delaying gastric emptying. Adults tend not get these latter.

In both cases the use of non-steroid anti-inflammatory drugs (NSAID) can suppress mucin production and so weaken the protective stomach linings.

Clinical Signs of Ulcers include:

- COLIC
- DIARRHOEA
- POOR APPETITE
- POOR HAIRCOAT - FOALS
- POT-BELLIED APPEARANCE - FOALS
- BRUXISM (TEETH GRINDING)
- PTYALISM. (OVER PRODUCTION OF SALIVA)
- POOR PERFORMANCE
- FOALS MAY ALSO HAVE ENTEROGASTRIC REFLUX WHEN INTUBATED.



horses come first

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Diagnosis

Gastric entubation – gastroscopy – is the major diagnostic tool and leads to quantification of the symptoms;

Grading Score

i. **Grade 0:** intact epithelium with no mucosal changes: Normal

ii. **Grade 1:** mucosal reddening or squamous hyperkeratosis

iii. **Grade 2:** small single or multifocal ulcers

iv. **Grade 3:** large single or multifocal ulcers or extensive superficial mucosal lesions

v. **Grade 4:** extensive lesions with apparent deep ulceration

Treatment

Based on prognosis there are a number of treatments:

Pharmacologic

1. Proton pump blockers, eg, omeprazole (Gastroguard and Ulcerguard)

1. Inhibit the H+-K+ ATPase pump on the parietal cell. Reducing H+ extrusion into the lumen of the stomach. Very effective at lowering gastric pH for 24 hours with a single dose.
2. For clinical disease: 4 mg/kg orally, once daily for 4 weeks.
3. For prophylaxis (eg, before an intense race/show season): 1-2 mg/kg orally, once daily during the season.

2. Histamine -2 receptor antagonists, eg. ranitidine and cimetidine

1. Blocks histamine from binding to the parietal cell H2 receptor, thereby inhibiting H+ secretion
2. Less effective than PPIs
3. Required more frequent administration, eg, two to three times/day
4. See Table 1 for dosages

3. Barrier protectants, eg, sucralfate (Carafate)

1. A polysaccharide that binds to ulcerated mucosa, creating a mucopolysaccharide bandage
2. Poorly defined efficacy in horses. Requires frequent administration and does not address the pathophysiologic underlying problem, ie, excessive acid secretion
3. Symptomatic relief only

4. Acid neutralization products. Aluminium/ magnesium salts, eg, aluminium hydroxide.

1. Will buffer acid immediately, yet have only a temporary effect

5. Prostaglandin E analogue, eg, misoprostol (Cytotec)

1. Believed to enhance mucosal blood flow and assist in healing NSAID-induced ulcers
2. Can cause abdominal discomfort, diarrhea, and bloat in some horses

Other therapy options

1. Diet modifications

a. Feed more frequently. Less meal style feedings
b. Increase the roughage portion of the diet
c. Reduce the concentrate portion of the diet
d. Corn oil. Rich in linoleic acid, which is a precursor fatty acid for the synthesis of PGE series. Gradually introduce 20 mL/kg body weight once a day

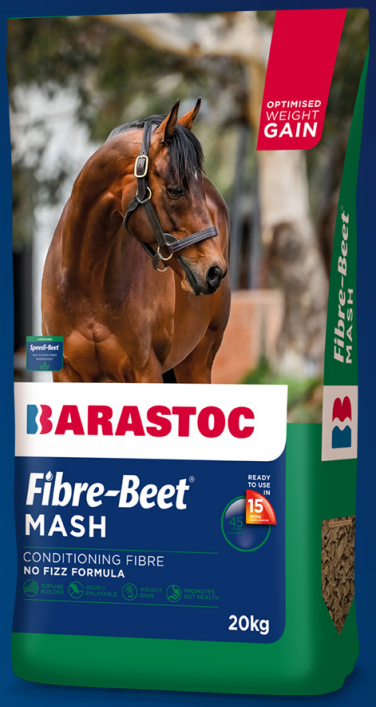
2. Stress reduction methods

a. House together
b. Modify training practice.

horses come first

Potential role of Fibre-Beet Mash

All the above treatments work around either reducing acid secretion or by enhancing or bolstering mucin production.



There are a number of aspects when dealing with performance horses such as racehorses, showjumpers and eventers; these animals tend to receive high carbohydrate hard feeds as a source of readily available energy.

In this context there is a direct effect at the gastric level.

Firstly, the presence of a high starch meal will stimulate those bacteria present in the stomach and these will produce fermentation products that will increase the acidity of the stomach contents beyond that normally reached with stomach acid alone.

Secondly these diets are not good at absorbing excess acid and this exacerbates acidity in the stomach. As discussed in the previous article these points have two results. Unbound acid can reflux into the squamous portion and the VFA from fermentation can penetrate the mucosa where it can interfere with mucin production etc.

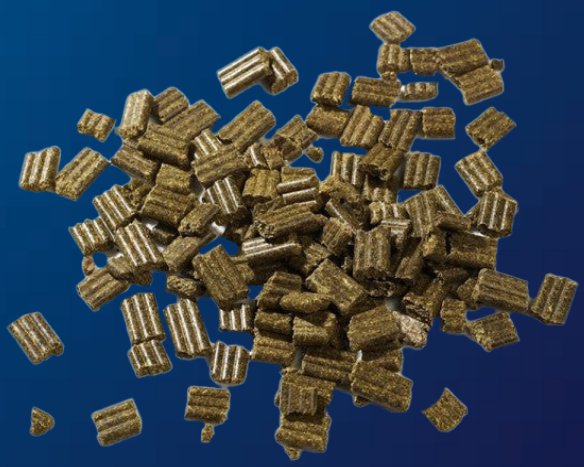
The obvious answer, purely from a health aspect, is to have free access to constant feeding of fibrous material. By looking at individual feedstuffs the ability of some feeds to absorb, and therefore remove from free circulation in the stomach, acid is far greater than others. Acid binding capacity is a measurement of this, at various acidity levels; the higher the value, the greater the binding.

Research has shown that horses grazing have stomach acidity levels of >pH 4 whereas those fed concentrate meals have levels as low as pH 2.

However, it is not always practical for performance horses to be fed continuously and there does need to be some concentrate feeding to supply sufficient energy for activities and concentrates need to be fed in discrete meals.

Work conducted by Andrews (2007) compared feeding bromegrass hay with alfalfa hay and concentrates on stomach acidity, VFA production and incidence of ulcers. The research found that the pH of stomach contents with alfalfa/concentrates was higher, and therefore less acidic, than bromegrass despite higher levels of VFA, and lesion numbers and severity less in squamous mucosa. What was of interest was the effect of this buffering of acidity by alfalfa lasted for 6 hours so it is conceivable that meal feeding of alfalfa can help maintain normal stomach acid levels.

Fibre-Beet Mash contains beet pulp as Speedi-Beet, alfalfa and oat fibre. As such it will have a high acid binding capacity – probable in the order of 400 meq/kg (around 4-5x that of cereals) and so would be a useful additive to concentrate meals. However, the product can also address the other aspect of ulcer control. As discussed above most treatments act either through acid control or by coating or supporting the mucosal layer.



Pectin-lecithin complexes have been shown to help in the reduction of ulcers (Marquez. , 2007). Research by Thirawong et al (2007) looked at the mucoadhesive properties of pectins of various degrees of esterification. They showed that mucoadhesion depended on the pH of gastric mucosa, peaking at a pH of around 4.8, and degree of esterification. Pectins were also good mucoadhesives in the small and large intestine. Hino et al (2013) showed low methoxyl pectins can stimulate mucin production from the small intestine.

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Beet pulp is a highly esterified pectin that also contains acetylated galacturonic acid. This means that beet pectins also act as their own emulsifiers and so we would expect a good level of mucoadhesion.

We also know that pectins are removed from cell wall matrix in the acid environment of the stomach and are used in parenteral nutrition to support mucosa (Bengmark & Jeppson 1995).

It is logical to assume that in a situation where alfalfa helps reduce the acidity of the stomach pectins can form strong mucoadhesives and subsequently improve mucin production in the small intestine. In addition, the oat fibre provides phospholipids that help the already easily emulsified beet pectin, making a good mucosa fortifier. It is also probable that, due to the nature of gastric motion, gastric mucoadhesion will also happen in the squamous mucosa. If gastric ulceration is caused in this region when acid refluxes into the squamous area, reflux with pectin should also occur.

Fibre-Beet can act as a moderator in the control of ulcers. It can be fed alongside concentrates to help maintain a normal acidity level in the stomach, an effect that can last up to 6 hours from a single discrete meal, and also provide materials to maintain and bolster the mucin layers in the stomach and along the length of the gastrointestinal tract, a characteristic that can counter stress and other behavioural factors.

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