Pasture and laminitis: truth or fiction?

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Pat Harris, Annette Longland, Jo-Anne Murray, Andrea Ellis, Catherine Dunnett, Nicola Menzies-Gow, Simon Bailey and Jonathan Elliott discuss research into laminitis, provide an insight into obesity and insulin resistance and suggest ideas regarding management protocols

PASTURE turnout can be a trigger factor for laminitis, even in lean non-obese animals, which do not have raised basal plasma insulin concentrations.

Obesity may increase the risk, as does turning out on a pasture with a high non-structural carbohydrate (NSC) content (simple sugars, fructans and starch).

Horse owners, keepers and veterinarians all appreciate the health and welfare implications of laminitis, and it is difficult to open a “lay” magazine without there being an article on laminitis, or the postulated linked conditions of insulin resistance and obesity.

To provide a consensus and up-to-date review of some key aspects relating to nutrition and laminitis, a group of researchers and nutritionists have collaborated to produce the following article, which we hope will be of value.

Background

Laminitis is not a “modern” condition – it has been recognised for more than 2,000 years.

Aristotle even referred to it in around 350BC as “barley disease”, presumably because, even then,

it was associated with excessive consumption of grain. This cause was confirmed by Garner and others in the mid-1970s[1], when they demonstrated that the administration of a large bolus of starch could trigger laminitis episodes. In the late 1970s/early 1980s[2], clinical reports were published of a link between bedding horses on black walnut shavings and outbreaks of laminitis.

However, there has been increased recognition of the importance of pasture-associated laminitis – perhaps because farms have diversified and ponies/horses are more often kept on "improved" pastures established for maximal outputs of meat and milk from ruminant livestock. In a UK survey conducted in the mid-1990s[3], 61 per cent of animals were kept out at grass prior to an attack, 30 per cent on a combined pasture/stable systems and only nine per cent were stabled. Furthermore, in a US survey[4] approximately 45 per cent of all laminitis cases reported were linked with pasture.

Pasture-associated laminitis

It is well recognised that turning out certain ponies or horses on to "lush" (green and actively photosynthetic) or "stressed" (where environmental conditions are sub-optimal for growth) pastures – especially in the spring and autumn – seems to be a common predisposing and/or triggering factor.

So what is it within pasture that might trigger laminitis? Currently, it is believed that the high levels of water-soluble carbohydrates (WSC: including the simple sugars, as well as the more complex storage carbohydrates/ sugars – fructans) and/or starch may be involved in this process, as outlined in Figure 1.

The fructan controversy

In the late 1990s, a potential link between grass fructans and laminitis was suggested (Longland et al, 1998), and then research showed that a bolus of fructan (after a short adaptation phase) at between 7.5g/kg and 12.5g/kg bodyweight (BW; the equivalent of approximately 3.75kg to 6.25kg to a 500kg horse in one meal) could reliably produce laminitis[5],[6], even in a breed not believed to be prone to the condition.

However, it has been questioned whether grazing animals would ever reach the threshold amount of rapidly fermentable carbohydrate required to initiate the sequence of events during normal grazing conditions, and even whether fructans can reach the hindgut and, therefore, initiate the cascade of events illustrated in Figure 1.

The authors of this review feel it is likely a combination of rapidly fermentable carbohydrates (including fructans) within the pasture (see Figure 2) may be involved and, although no proof exists that fructans are an important component of this triggering factor for laminitis, the following aspects support their involvement, as well as the other nonstructural carbohydrate (NSC) components in

grass.

• Crucially, it is important to realise that temperate grass fructans are complex in nature[7], and very different from the type of fructans that have been used in the experimental studies that have suggested, for example, significant digestion in the stomach and small intestine[8]. In fact, recent work using grass fructan has confirmed that the majority of it is likely to reach the hindgut largely unchanged9.

• Analytical methods that rely on techniques designed for the more simple fructans found in human feeds are likely to considerably underestimate the levels in grass and hay[10].

• Research has shown that the dose of “short-chain fructans: oligofructose” needed to induce laminitis (even in adult horses) is perhaps lower than previously thought – in one study, three out of eight horses provided with 5g of oligofructose/kg BW developed laminitis, as did four out of four provided with 7.5g/kg BW[11].

• Ponies can potentially ingest significantly more dry matter per day (up to approximately five per cent[12]) than the textbooks suggest. Although considerably lower, total daily intakes were recorded in another study, which suggested that ponies can ingest more than 30 per cent and up to 41 per cent of their daily dry matter intake (DMI) within a three-hour turnout period[13] (and personal communication).

Furthermore, estimates of intakes when out at grass of up to five per cent BW have been made[14].

• In summer (but not winter), laminitis-prone ponies had higher serum insulin concentrations in one study[15] when compared to age and body condition score (BCS) matched control ponies, suggesting that consumption of summer pasture (high in NSC, including fructans) could be important.

• A marked increase in the serum insulin concentrations of laminitis-prone ponies was recorded during the transition from winter to spring, in association with an increase in the forage WSC content (simple sugars and fructans[16]). The most marked increases were seen in ponies that subsequently developed laminitis.

• Finally, and perhaps most crucially, the threshold levels determined under controlled conditions may be very different from the naturally occurring situation in an individual animal predisposed to the condition (see Figure 3). In addition, predisposing events and/or threshold-lowering factors may be additive in nature.

Survey work[17] has shown that of 245 samples of pasture grasses (perennial ryegrass, Timothy and fescue) harvested throughout a growing season, about 20 per cent contained more than 20 per cent WSC on a dry matter (DM) basis, circa five per cent contained more than 25 per cent WSC

and three per cent contained more than 30 per cent WSC.

Furthermore, the average fructan content of two of the ryegrasses was 27 to 28 per cent when harvested in May. If a 250kg pony was turned on to grass with a 27.5 per cent fructan content, it would only need to ingest 4.5kg DM grass (1.8 per cent BW DM) to ingest the amount of fructan used by Kalck[11] et al to induce laminitis in some of their non-laminitisprone animals.

Obviously, in such grass other NSCs would also be present. It is, therefore, theoretically possible (especially in an animal predisposed to the condition) that horses and ponies could ingest sufficient NSC (including fructan) from a high-yielding fructan or NSC-rich pasture in a relatively short space of time to initiate a laminitic episode, along a similar pathway to the bolus fructan/ starch experimental models.

However, it is also possible that such pastures influence the risk of laminitis in other ways. Grazing a high-NSC-providing pasture, for example, may promote insulin resistance development. At some times of the year, grazing high-NSC pastures may result in peaks of insulin similar to a high-starch diet[18].

This, in turn, may lower the threshold for laminitis to be triggered. It is also possible that the various predisposing events and/or threshold-lowering factors may be additive in nature (see Figure 3).

Obesity and insulin resistance

Coffman and Colles suggested, in 1983[19], that laminitic ponies were significantly less sensitive to insulin than others.

Other researchers subsequently proposed insulin resistance as a contributing factor in the apparent association between obesity and laminitis[20]. A BEVA-EBM study on pastureassociated laminitis concluded that “overweight animals that develop laminitis tend to have more severe signs than those of optimal weight. When laminitis does occur, overweight animals are more likely to die of the disease than their thinner counterparts[23]”. Certain breeds (such as quarter horses and Morgans) and types (for example, ponies) are perhaps more prone to obesity, and benefit from even closer attention to their diet and exercise.

In a group of inbred ponies with a high incidence of laminitis, generalised obesity (BCS equal or greater than seven/nine), regional accumulation of neck crest adipose tissue (“cresty neck” equal or greater than four/five), hyperinsulinaemia (more than 32uU/ml) and hyperleptinaemia (more than 7.3ng/ml) all had reproducible diagnostic accuracy for predicting the development of laminitis when ponies were subsequently exposed to spring pasture[21,22]. For an individual that surpassed three out of four of these criteria, the sensitivity was 86 per cent and the specificity was 87 per cent. However, these levels of sensitivity may not apply more generally.

A consensus statement has suggested most equids with equine metabolic syndrome (EMS)[24] exhibit the following characteristics:

• generalised obesity or increased adiposity in specific locations, such as the nuchal ligament region (cresty neck);

• insulin resistance characterised by hyperinsulinaemia or abnormal glycaemic and insulinaemic responses to oral or intravenous glucose and/or insulin challenges; and

• a predisposition towards laminitis – clinical or subclinical laminitis that has developed in the absence of recognised causes, such as colic, colitis or retained placenta. Australian researchers[25] have shown that laminitis can be induced in healthy ponies through maintaining a high-plasma insulin concentration (with normal glucose). Collectively, this body of work has led to today's endocrine theory of laminitis. It is, however, important to realise that[21],[22],[26],[27],[28] (and personal communication):

• not all ponies that develop laminitis out on pasture, or that are fed high-NSC forage or other feeds, are obese;

• an animal does not need to be obese to be insulin resistant;

• not all obese animals are insulin resistant;

• some "acceptably conditioned" animals are "insulin resistant", but may or may not have raised basal insulin levels;

• even in known insulin-resistant, laminitis-prone animals, insulin levels of more than 1,000IU/L do not necessarily trigger an episode of laminitis;

• not all obese ponies develop laminitis when turned out to grass;

• insulin resistance is likely to be a graded continuum, rather than ponies either being insulin resistant or not, and the true relationship between the level of insulin resistance in a pony and its susceptibility to laminitis is not known; and

• other endogenous and as yet unknown risk factors for laminitis, apart from insulin resistance, are highly likely to exist. This means that not all ponies that develop laminitis will have a background of insulin resistance.

Management

How can we manage those prone to laminitis – especially pasture-associated laminitis? Based on

existing knowledge and scientific reports, we would recommend the following protocols.

• Base the horse’s diet on forage and/or fibre.

• Especially in recurrent or problem cases, it is recommended to analyse the forage and try to feed forage or forage replacers with less than 10 per cent NSC – many UK hays will be higher than this. Soaking hay in clean water (more than 8°C, and ideally around 16°C) for at least three hours may help to reduce the WSC content. The amount lost is highly variable, and does not necessarily reduce to less than 10 per cent NSC.

• Research has suggested that feeding straw as the main forage may significantly increase the risk of gastric ulcers[28] and high-lignin/silicated forages may increase the risk of impaction colics in some animals.

• Feed a broad-spectrum vitamin and mineral supplement if no or low levels of hard feed are provided, especially if forage quality is low. Ensure an adequate and balanced intake of magnesium. However, no evidence suggests that high levels will be protective and reduce the risk of laminitis.

• For many animals, additional energy sources will not be required. However, for those requiring additional energy:

– if no contraindications are apparent, consider using oil as an energy source, rather than cereal starch if required – especially for those animals not being exercised (remember to add gradually, balance the overall diet and add additional vitamin E at 100IU to 150IU/100ml of oil and, without further advice, not more than 100ml oil/100kg BW per day);

– if any cereals other than oats are fed, make sure they are processed by cooking (such as steam flaking or micronising) to make the starch more easily digestible, reducing the risk of starch overload in the hindgut; and

– avoid feeding large grainbased meals: restrict meal sizes to less than 0.5kg/100kg BW of a cereal-based feed. For all horses, ensure overall starch intake is less than 1.0g starch/kg BW per meal.

• Prevent horses or ponies having access to grain bins.

• Make all dietary changes slowly and avoid abruptly starv ing animals, especially pregnant animals, in an attempt to reduce their BW or prevent them from eating for prolonged periods due to the risk of hyperlipaemia.

• Maintain regular exercise wherever possible – carefully assess how much exercise can be

undertaken. It is not known how much exercise is required to improve insulin sensitivity[29].

• Avoid allowing horses or ponies to become obese. Aim to maintain a moderate BCS between four and five (moderate) out of nine. For animals that are overweight, plan an active weight management programme, linked with increasing exercise whenever possible.

Set realistic targets and monitor the horse's weight and condition on a regular basis.

Turn-out advice

• Consider zero grazing (while providing the horse with suitable forage, as well as roaming and socialising alternatives) if it is essential that the horse ingests minimal levels of sugar, starch and fructans, or you need a strict weight management programme. Consider dirt paddocks or large group housing practices.

• Turn horses out to pasture when fructan/WSC levels are likely to be at the lowest, such as very late at night to early morning. Remove them from the pasture by mid-morning.

• Do not graze on pastures that have not been properly managed by regular grazing or cutting. Try to maintain a young leafy sward, as mature stemmy grasses contain higher levels of stored fructans.

• Avoid or restrict turning out in spring (before flower development) and autumn, as well as during flowering and early seeding.

• Do not allow horses to graze on pastures exposed to low temperatures (such as frosts) with warm, bright sunny weather or those "stressed" through drought.

• Consider maintaining turnout by use of grazing muzzles (ensure the animal can obtain sufficient water intake, be aware of possible behavioural issues, and that they are fitted correctly and ensure animals are provided with significant periods of time without the muzzles); strip grazing behind other horses or sheep; mowing and removing clippings; putting a deep layer of wood chips over a small paddock or using dry lots/indoor schools.

• Rotate the use of paddocks regularly, preferably with other species (such as sheep or cattle) to keep the grass levels at an appropriate height to avoid the paddocks becoming "stressed" through either under or over-grazing.

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