Livestock Science 186 (2016) 2–5

Contents lists available at ScienceDirect

Livestock Science

journal homepage: www.elsevier.com/locate/livsci

A preliminary study of grazing intakes of ponies with and without a history of laminitis

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ARTICLE INFO

Article history:
Received 23 March 2015
Received in revised form
21 August 2015
Accepted 27 August 2015

Keywords:
Equine
Laminitis
Grazing intake

ABSTRACT

One possible factor involved in the aetiology of laminitis is grazing intake. Whilst some studies have looked at grazing intake in healthy animals, there has been little comparison made between animals with and without a history of laminitis. The aim of this study was to compare grazing intake between health animals and those with a known history of laminitis. Sixteen mature grass-kept (maintained at grass 24 h a day) native breed ponies from World Horse Welfare in Norfolk were used in the study, which was conducted in the month of July for a period of 12 days. All animals were grazed under identical conditions. Grazing areas were of that suitable for the management of animals predisposed to laminitis (for ethical reasons) and therefore herbage mass was low (Yield: 124 kg dry matter/ha; sward height of 1–2 cm). Faecal samples were collected from 8 clinically normal horses (NOR) and from 8 that were predisposed to laminitis (LAM) in July 2005. Grazing intake was measured using the alkane technique. Dry matter intakes (DMI) per kilogram bodyweight were low in both groups of animals: 1.32 ± 0.31 percent versus 1.62 ± 0.74 percent for NOR and LAM, respectively. There was no difference in DMI between the two groups of ponies (4.43 versus 4.25 kg/day for NOR and LAM, respectively). Mean DMI per kilogram bodyweight per day were 1.32 and 1.62 for NOR and LAM, respectively (20 percent difference). There was a greater variability of DMI within the LAM group with intakes ranging from 0.81 to 2.36 percent bodyweight. The low DMI values were attributed to the overgrazed nature of the pasture used in this study, which was unavoidable due to the welfare issues associated with grazing overweight, laminitis-prone horses on good grazing pasture. Further work is required with a larger study population grazing pastures with greater herbage mass.

1. Implications

Pasture-induced laminitis is thought to be due to excessive ingestion of grass. What is unknown is why only certain animals develop laminitis even when grazed on identical pastures, which may be due to some animals eating more than others. This study investigated grazing intakes between ponies with (LAM) and without (NOR) a history of laminitis. Results showed no difference in intakes between the LAM and NOR groups. However, the LAM ponies ate 20 percent more grass overall compared to the NOR group.This implies that more research is needed with larger groups of animals to determine if differences in intakes exist between LAM and NOR groups and if this may be a predisposing factor in the onset of laminitis.

2. Introduction

Laminitis has widespread implications for equine welfare; it has the highest death rate of any orthopaedic condition and is the second largest killer of horses in the UK next to colic. It is also extremely common; in a survey involving 113,000 horses in the UK a prevalence of 7.1% was noted (Hinkley and Henderson, 1996). Clinical laminitis represents the end result of a systemic condition that can have many predisposing factors as outlined by Trieber et al. (2006). The pathogenesis of acute laminitis and in particular the relationship between hindgut disturbances and the pathological mechanisms in the digit are crucial to the understanding of the pathogenesis of this disease. Although there are also many other potential factors that may contribute to this condition, pasture-induced laminitis appears to be the most common aetiology in the UK (Hinkley and Henderson, 1996). Moreover, the incidence of laminitis in the US is reported to be 2 percent rising to around 5 percent in the spring and summer (Longland and Byrd, 2006). As its name suggests, this is thought to be associated with excessive ingestion of pasture and/or abrupt change in pasture NSC, and

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http://dx.doi.org/10.1016/j.livsci.2015.08.012
1871-1413/

studies in the UK suggest an increased prevalence of laminitis during periods of rapid grass growth. Grass storage carbohydrates (water soluble carbohydrates) have been implicated in the onset of pasture-induced laminitis (Longland et al., 1999). However, one intriguing area is why only certain individuals appear to be predisposed to laminitis, even when grazing identical pastures? Several schools of thought exist, including differences in individual susceptibility at the level of the large intestine in the ability of this organ to buffer changes in pH due to lactic acid production, microbial populations, genotypic factors and grazing intake to name but a few (Bailey et al., 2004). Intakes of pasture are reported to range from 1.5 percent to 3.3 percent of bodyweight (BW) per day (Holland et al., 2000; McMeniman, 2000), indicating a large variation in grazing intakes. However, despite the prevalence of pasture-associated laminitis and the links between intakes and onset of laminitis in some individuals, there have been no studies investigating whether differences in grazing intakes between individuals may affect susceptibility to laminitis. Consequently, the aim of this study was to measure grazing intakes in ponies with and without a history of laminitis, with the hypothesis that animals with a history of laminitis may have greater intakes than those without.

3. Materials and methods

3.1. Animals and management

Sixteen mature grass-kept (maintained at grass 24 h a day) ponies from World Horse Welfare in Norfolk, United Kingdom were used in the study, which was conducted in the month of July 2005 for a period of 12 days. Eight mares and eight geldings were used in the study, split into two groups of ponies, 8 clinically normal (NOR) and 8 that had a history of laminitis (LAM). There was an equal distribution of mares and geldings in each group. Ponies with a history of laminitis were included in the study if they were diagnosed with acute laminitis 3 or more times during the preceding three years. Bodyweights at the start of the study averaged 308 ± 92 kg, with body condition scores averaging 3 ± 1 on the 0–5 scale. Animals were weighed at 0900 h on days 1, 4, 8 and 12 and condition scored on days 1 and 12 (Carroll and Huntington, 1998). All animals were grazed under identical conditions in the same paddock. Grazing areas were of that suitable for the management of animals predisposed to laminitis (for ethical reasons), with low herbage mass and small paddock sizes (0.3 acres per pony). Grazing intake was measured according to the techniques described by Dove and Mayes (1991).

3.2. Marker preparation and administration

Ponies were hand fed a bite-sized Weetabix® (WB: Weetabix Ltd., Kettering, UK) labelled with C_{32} alkane (Fisher Scientific, Loughborough, UK: 10162190) 3 times per day for a period of 12 days. The alkane-labelled WB was prepared in a fume cupboard. 38 g of C32 was dissolved in 380 ml of heptane using a hotplate stirrer on low heat. The resultant solution contained a concentration of 100 mg of C_{32} per ml of heptane and 10 ml of this was added to each WB. The WB then remained in the fume cupboard overnight at ambient temperature to allow for the absorption of the C_{32}/heptane solution before being placed into a force-draught oven at 60 °C for 16 h. Prior to removal the temperature was increased to 90 oC for one hour to ensure the C_{32} was fully absorbed. A sub-sample of 5 alkane-labelled WB was retained for laboratory analysis to determine C_{32} dose rate.

3.3. Sward sampling

Quadrat samples (900 cm_2) were taken to determine the herbage mass of the field on days 5, 7, 9 and 12 of the study. Six herbage samples were taken at random in a large "W" shape across the whole field with grass cut as close to the soil as possible without any visible contamination of the sample. Sward height was determined using a plate meter (F100 Plate Meter, AgriSupplyServices, UK).

3.4. Herbage sampling

Herbage sampling began on day 5 of the study and continued to day 12. Samples were taken twice daily at 10 am and 3 pm to 4 pm depending on the grazing activity of the horses. A quadrat sample (900 cm_2) was taken of the grass each horse was eating by placing the quadrat as close as possible to where each horse was grazing. Samples were weighed and then dried at 60 °C until constant weigh and ground (to pass through a 1 mm dry mesh screen) prior to alkane analyses.

3.5. Faecal sampling

Faecal sampling for alkane analyses occurred during the last 5 days of the study (days 7–12). One complete faecal deposit was collected per horse each day, weighed and a 250 g sub-sample taken, dried at 60 °C to constant weight and ground (to pass through a 1 mm dry mesh screen) prior to alkane analysis. An additional faecal sample was collected for each horse on day 12 of the study, frozen immediately and transported to the laboratory for determination of microbial populations.

3.6. Alkane analysis

Herbage and faecal samples were analysed for the natural odd-chain alkane C31 and faecal samples were also analysed for the dosed C_{32} alkane at the Macaulay Institute, Aberdeen, United Kingdom using the method described by Ali et al. (2004). The 5 sub-samples of the WB were also analysed for C_{32} alkane by crushing them and placing them into separate 100 ml glass bottles, which were capped and weighed. Heptane (30 ml) was added to each bottled and these were then re-weighed. The bottles containing the samples were then heated at 55 °C for 1 h in an ultrasonic bath to dissolve the alkane. A sample (0.2 ml) of the warmed solution was then removed from each glass bottle and placed into pre-weighed screw-capped vials. Vials were then capped and re-weighed and 1.3 ml of alkane internal standard (C22 = 0.80131 mg/g and C34 = 0.80166 mg/g) added to each vial and the vial re-weighed. Samples (0.1 ml) were then taken from each vial and placed in separate gas chromatography (GC) vials to which 0.3 ml of dodecane was added. The concentration of C32 was then determined by GC using the conditions described by Ali et al. (2004).

Herbage intake was calculated using the herbage and faecal concentrations of consecutive even- and odd-chain alkanes using the following equation:

Herbage intake (kg DM/day):

$$D_j \times (F_i/F_j)$$

$$H_i - \left((F_i/F_j) \times H_j \right)$$

where

D_j = dose rate of even chain alkane (C_{32})
F_j = faecal concentration of even chain alkane (C_{32})
H_j = herbage concentration of even chain alkane (C_{32})