

# Risk factors associated with equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark

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## Summary

**Reasons for performing study:** The prevalence (up to 93% in Thoroughbred racehorses) and severity of equine gastric ulceration syndrome (EGUS) has been associated with type of training and differing management practices. However, there have been few studies to confirm these findings in nonracehorses in Europe.

**Objectives:** To investigate the prevalence of EGUS in a population of Danish horses, during winter when the horses had been housed and fed for at least 8 weeks and to analyse the influence of feed, work level and environment on the risk of EGUS of  $\geq$  grade 2 in severity.

**Methods:** A total of 201 horses, not in active race-training, were evaluated, representing 23 different stables from all 5 regions within Denmark. All horses were considered to be healthy and not on medical treatment for EGUS. Endoscopically observed ulcer lesion scores were based on the number present (0–4) and severity (0–5). Univariate and multivariable mixed effects logistic regression models were developed using EGUS score as the dependent variable. An ulceration severity score of  $\geq 2$  was regarded as being clinically significant. Separate models were developed for horses with ulcers in either the glandular or nonglandular regions of the stomach graded  $\geq 2$  (EGUS  $\geq 2$ ) and for those horses that had nonglandular ulcers graded  $\geq 2$  (NG  $\geq 2$ ).

**Results:** In this population, 53% (107/201) of horses were graded as having EGUS  $\geq 2$  with 95 (47%) horses having NG  $\geq 2$ . Three variables were significantly ( $P < 0.05$ ) associated with EGUS  $\geq 2$ : straw being the only forage available; exceeding 2 g/kg bwt of starch intake/day or  $> 1$  g/kg bwt/meal; and water not being available in the turn out paddock. Risk of NG  $\geq 2$  significantly increased when straw was the only forage available, 1 g/kg bwt of starch/meal was exceeded, water was not available in the turnout paddock and the interval between forage feeding was  $> 6$  h.

**Conclusion and potential relevance:** This study has confirmed that components of the diet, readily modifiable, may have an important impact on the risk of EGUS in the nonracehorse. Differences in the multivariable models produced for all ulcers and nonglandular ulcers support differences in the aetiology of ulcers in different locations of the stomach.

## Introduction

In the mature horse the stomach secretes approximately 1.5 l/h of gastric juice which contains 4–60 mmol of hydrochloric acid. The pH of the gastric contents depend on the feeding regimen and the region of the stomach measured. However, typically there is a stratified pH (Andrews and Nadeau 1999) from around neutral in the dorsal portion of the oesophageal region, with a more acidic pH near the *margo plicatus* (pH  $\sim 3$ –6) and a lower pH near the pylorus (pH  $\sim 1.5$ –4.0). The horse, as a nonruminant herbivore, being well suited to a high fibre, low starch diet, would naturally spend up to 18 h/day foraging and rarely fast voluntarily for more than 2–4 h at a time. Saliva is produced in response to chewing and, under a natural grazing environment, helps to buffer gastric acid, which is secreted in a continuous variable pattern into the stomach. In addition, living under natural conditions encourages the horse to move freely, which may assist in the normal movement of stomach contents through the gastrointestinal tract. Modern management practices, which may include meal feeding often within an enclosed environment with limited opportunity for free movement, low fibre/high concentrate diets, early weaning and intensive training programmes, are believed to ahvev the potential to produce a poorly buffered, acidic environment in the stomach (Davidson and Harris 2002; Andrews *et al.* 2006). This is thought to be associated with the high prevalence of gastrointestinal ulcers, particularly in intensively managed horses such as performance and racehorses.

The majority of the work in mature horses has been carried out in racehorses (Hammond *et al.* 1986; Murray *et al.* 1989, 1996; Vatisstas *et al.* 1999a; Jonsson and Egenvall 2006), whereas only a few studies have been performed on leisure horses, breeding mares and young horses. Contributing factors to the reported high prevalence in racehorses, in particular, have been suggested to be high concentrate diets, low hay diets, meal feeding, fasting, training and the administration of certain drugs (Vatisstas *et al.* 1999b; Merritt 2003; Lester 2004; Jonsson and Egenvall 2006). However, there have been few studies to confirm these findings in nonracehorses in Europe.

This study therefore aimed to investigate the prevalence of EGUS in a subpopulation of Danish horses not in active race training and to evaluate the influence of feed, work level and environment on the risk of EGUS  $\geq$  grade 2 severity (MacAllister *et al.* 1997).

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## Materials and methods

### Horse recruitment

For details of the 201 horses, which formed the population studies in 23 different stables from all 5 geographical regions within Denmark, see Luthersson *et al.* (2009). Evaluations occurred in 2 winter periods between November and March 2005/2006 and 2006/2007, when the horses had access to a shelter or stable and had been fed supplementary feed for at least 8 weeks.

### Examination

All horses were fasted from 17–23 h prior to endoscopy and water was withheld for 5–10 h. The horses were sedated with detomidine (Domosedan 10–15 µg/kg bwt i.v.)<sup>1</sup> and butorphanol (Torbugesic; 15–20 µg/kg bwt i.v.)<sup>2</sup> and examined for the presence of gastric ulcers using a 300 cm flexible video endoscope<sup>3</sup>. A thorough as possible examination of all areas of the stomach was performed (Luthersson *et al.* 2009). Ulcer lesion scores were based on the number present (0–4) and severity (0–5) (MacAllister *et al.* 1997) as described in the companion paper. The location of the ulcers was also recorded as being either glandular only, nonglandular only or both. The same investigator (N.L.) performed all scoring without any prior knowledge of any horse level factors.

Bodyweight was estimated using a commercial height specific weigh tape<sup>4</sup> (Ellis and Hollands 2002), and body condition score determined using the 1–9 scale adapted from Henneke *et al.* (1983).

### Questionnaire

In addition to the details relating to the individual horse evaluated in Luthersson *et al.* (2009), information was collected with respect to feeding, management and workload. This included details of the purpose/type of work, workload for riding horses, type of stable, type of bedding, time spent outdoors per day, type of paddock, availability of water in the outdoor paddock, number of concentrate and forage meals fed per day, longest intervals between meals (for both forage and concentrate), meal size, type of forage and appetite. A copy of the questionnaire is available from the corresponding author.

An estimate of the starch intake/day (g/kg bwt) as well as starch intake/meal (g/kg bwt/meal) was made for each horse, excluding any starch provided from forage (hay/haylage/straw or pasture) and using standard analytical data provided for Danish cereals (Strudsholm *et al.* 1997) and the manufacturer provided information for proprietary feeds.

### Statistical analysis

Univariate and multivariable single-level and mixed effects logistic regression models were developed using EGUS score as the dependent variable. The yard at which the horse was located was included as a random effect. An EGUS severity score of  $\geq 2$  was regarded as clinically significant gastric ulceration. All variables with a P value of  $\leq 0.25$  during the univariate screening process were available for inclusion in the final multivariable models. However, all variables were also forced into the final models to assess for confounding. Mixed effects multivariable logistic regression models were developed, using a forward selection procedure. Variables with strong *a priori* biological reasons for

inclusion were also considered in the final model. Variables were retained in models if they significantly reduced the residual deviance of the model (likelihood ratio statistic [LRS]  $P < 0.05$ ).

Two multivariable models were developed. The first used all horses with EGUS  $\geq 2$  as the dependent variable. In the second, only those horses with EGUS  $\geq 2$  in the nonglandular region of the stomach were defined as cases (NG  $\geq 2$ ).

The degree of yard level clustering was assessed by calculation of intraclass correlations and variance inflation factors (Dohoo *et al.* 2003).

The fit of the final single-level multivariable models was assessed using the Hosmer-Lemeshow goodness-of-fit test (Hosmer and Lemeshow 2000). Covariate patterns with the greatest leverage, delta betas, delta  $\chi^2$  and delta deviance values were identified for final multivariable models. Individual observations within these covariate patterns were identified and removed from the models and the change in the value of the coefficients was reassessed (Hosmer and Lemeshow 2000). The predictive ability of the single level models was determined by generating a receiver operating characteristic (ROC) curve.

All models were built using STATA version 10.0<sup>5</sup>.

## Results

### Endoscopic findings

Details of endoscopic findings and the effect of age, breed, gender and behaviour on the prevalence of EGUS are discussed in Luthersson *et al.* (2009). Overall, taking the glandular and nonglandular regions into consideration, in this study population 53% (107/201) of horses were graded as having a severity score  $\geq 2$  (24%: score 2; 16%: score 3; 9%: score 4 and 3%: score 5). Sixteen percent were graded 0 and 30% (61) as score 1.

### Risk factors

*Horses with gastric ulceration graded  $\geq 2$  severity, regardless of location in the stomach (EGUS  $\geq 2$ ):* Two single level and 2 mixed effects multivariable logistic regression models were developed, using the 2 different measures of starch intake (per day or per meal). The same risk factors were identified in both single level models but the point estimates of odds ratios, 95% CI and P values were altered (Tables 1 and 2). Three variables were associated with

**TABLE 1: Single and mixed effects (fitting yard as a random effect) multivariable logistic regression models developed for EGUS  $\geq 2$  (including starch intake per day)**

Variable	Single level model			Mixed effects model		
	Odds ratio	95% CI	P	Odds ratio	95% CI	P
<b>Available forage</b>						
Hay or haylage (ref)	1			1		
Straw only	4.4	1.5–12.6	0.006	4.5	1.4–14.9	0.01
<b>Water available in the paddock?</b>						
Yes (ref)	1			1		
No	2.7	1.4–5.5	0.005	2.6	1.03–6.6	0.04
<b>Starch intake</b>						
<2 g/kg bwt/day (ref)	1			1		
$\geq 2$ g/kg bwt/day	2.0	1.04–3.6	0.037	2.0	0.97–4.1	0.06

ref = Reference category; 95% CI = 95% confidence interval; mixed effects model, variance estimate for random effect Rho = 0.11; P = 0.047

EGUS  $\geq 2$ : Compared to hay or haylage, straw, as the only available forage increased the likelihood of EGUS  $\geq 2$  by 4.4 times (95% CI = 1.5–12.6; P = 0.006) or 5.7 times (95% CI = 2.0–16.7; P = 0.001), depending on which measure of starch intake was used in the model (Tables 1 and 2, respectively). Exceeding 2 g/kg bwt of starch intake per day was associated with an approximately 2-fold increase in the likelihood of EGUS  $\geq 2$  (95% CI = 1.04–3.6; P = 0.037) (Table 1). Alternatively, when included on a per meal basis, a starch intake between 1 g/kg bwt per meal and 2 g/kg bwt per meal, was associated with a 2.6 times increase in the likelihood of EGUS  $\geq 2$  (95% CI = 1.3–5.2; P = 0.006) and an intake greater than 2 g/kg bwt per meal increased the likelihood of EGUS  $\geq 2$  by 3.2 times (95% CI = 1.3–7.7; P = 0.009; Table 2). When water was not available in the paddock the likelihood of EGUS  $\geq 2$  increased by 2.7 times (95% CI = 1.4–5.5; P = 0.005) or 2.5 (95% CI = 1.2–5.1; P = 0.014), depending upon which measure of starch intake was used in the model (Tables 1 and 2, respectively).

*Horses with nonglandular gastric ulceration graded  $\geq 2$  severity (NG  $\geq 2$ ):* Starch intake per day was not associated with NG  $\geq 2$ . Therefore, only one single level and one mixed effects multivariable model, including starch intake per meal, was developed (Table 3). As with the previous models, straw as the only available forage increased the likelihood of nonglandular NG  $\geq 2$  by 4.5 times (95% CI = 1.6–12.5; P = 0.004). When water was not available in the paddock the likelihood of NG  $\geq 2$  increased by 2.4 times (95% CI = 1.2–5.1; P = 0.02). Feeding more than 1g/kg bwt of starch per meal was associated with a 2.4 times increase in the likelihood of NG  $\geq 2$  (95% CI = 1.3–4.6; P = 0.007). Feeding forage in any form other than *ad libitum* increased the likelihood of NG  $\geq 2$  by 3.9 times (95% CI = 1.5–10.4; P = 0.007).

*Yard level clustering:* There was some evidence of yard level clustering. Intraclass correlations using all EGUS  $\geq 2$  and NG  $\geq 2$  as the outcomes were 0.18 and 0.23, respectively. Corresponding variance inflation factors (VIF) were 0.54 and 0.69. Inclusion of yard as a random effect had little impact on the point estimates of the odds ratios apart from an increase from 3.9 to 5.3 for the odds ratio associated with the interval between forage feeds (Table 3). There was a moderate effect on the 95% confidence intervals in all 3 models, resulting in marginal significance for starch intake per day (Table 1) and water availability (Tables 2 and 3). The degree

**TABLE 2: Single and mixed effects (fitting yard as a random effect) multivariable logistic regression models developed for EGUS  $\geq 2$  (including starch intake per meal)**

Variable	Single level model			Mixed effects model		
	Odds ratio	95% CI	P	Odds ratio	95% CI	P
<b>Available forage</b>						
Hay or haylage (ref)	1			1		
Straw only	5.7	2.0–16.7	0.001	5.7	1.7–18.7	0.005
<b>Water available in the paddock?</b>						
Yes (ref)	1			1		
No	2.5	1.2–5.1	0.014	2.4	0.96–5.9	0.06
<b>Starch intake</b>						
<1 g/kg bwt/meal (ref)	1			1		
1–2 g/kg bwt/meal	2.6	1.3–5.2	0.006	2.6	1.3–5.5	0.01
$\geq 2$ g/kg bwt/meal	3.2	1.3–7.7	0.009	3.5	1.3–9.8	0.02

ref = Reference category; 95% CI = 95% confidence interval; mixed effects model, variance estimate for random effect Rho = 0.08; P = 0.09

of yard level variance was statistically significant in the first model (rho = 0.11; P = 0.047; Table 1) but not in models 2 and 3 (Tables 2 and 3).

*Model diagnostics, goodness of fit and predictive ability:* The final multivariable models were not significantly affected by influential covariate patterns. The Hosmer-Lemeshow goodness-of-fit statistics for the 3 single-level models were 2.3 (3 degrees of freedom [df], P = 0.51), 1.44 (5 df, P = 0.92) and 4.9 (4 df, P = 0.29), indicating that there was no evidence that any of the models did not fit the data well. The models were therefore considered to be reasonably calibrated. The predictive ability of the models as measured by the area under the ROC curve varied from 69–75%, i.e. moderate to good (Hosmer and Lemeshow 2000).

**Discussion**

In the current study, 53% of horses were defined as having clinically significant EGUS. The prevalence and severity of EGUS has been described previously in a number of different groups of horses. This figure compares with reports of up to 93% in racehorses and 37% in pleasure horses (Murray *et al.* 1996), up to 40% in Western performance Quarter Horses (Bertone 2000) and 58% in show horses (McClure *et al.* 1999). It is important to note that the exact definitions and inclusion characteristics varied between these different studies and therefore the reported prevalences are not entirely due to differences in breed, management and training practices.

For this current survey, an EGUS severity score of  $\geq 2$  was taken as being the significant criteria (see the companion paper) on which to base the analysis of risk factors. At this score and above the ulcers are considered to involve more than the superficial tissues and therefore more likely to have clinical significance. Fasting has been reported to create superficial lesions (Murray and Eichorn 1996) and therefore we considered that ulcer scores of 1 should not be considered as significant ulcers. EGUS severity score, rather than the number of lesions, was used as the response variable as McAllister *et al.* (1997) reported greater consistency between endoscopists recording severity compared with number of lesions and this was supported by Andrews *et al.* (2002).

**TABLE 3: Single and mixed effects (fitting yard as a random effect) multivariable logistic regression models developed for nonglandular EGUS (NG)  $\geq 2$**

Variable	Single level model			Mixed effects model		
	Odds ratio	95% CI	P	Odds ratio	95% CI	P
<b>Available forage</b>						
Hay or haylage (ref)	1			1		
Straw only	4.5	1.6–12.5	0.004	4.2	1.3–13.8	0.02
<b>Water available in the paddock?</b>						
Yes (ref)	1			1		
No	2.4	1.2–5.1	0.02	2.3	0.9–6.3	0.1
<b>Starch intake</b>						
<1 g/kg bwt/meal (ref)	1			1		
$\geq 1$ g/kg bwt/meal	2.4	1.3–4.6	0.007	2.6	1.2–5.4	0.01
<b>Interval between forage feeds</b>						
<6 h (effectively <i>ad lib.</i> )	1			1		
>6 h	3.9	1.5–10.4	0.007	5.3	1.4–20.0	0.01

ref = Reference category; 95% CI = 95% confidence interval; mixed effects model, variance estimate for random effect Rho = 0.13; P = 0.056

Gastric ulcers probably occur when there is an imbalance between those factors that are protective to the gastric mucosa (such as mucus, bicarbonate, prostaglandins, mucosal blood flow and epithelial restitution) and those that are ulcerogenic (including the presence of hydrochloric acid, volatile fatty acids [VFAs], pepsin or bile acids) (Ethell *et al.* 2000; Lester 2004). The current study identified a number of management practices that might affect this balance. The amount of grain (starch) fed per day and per meal in particular may, for example, be very important factors in the development of EGUS. Whether measured in g/kg bwt/day or per meal, starch intake over a certain amount increased the likelihood of EGUS  $\geq 2$  or NG  $\geq 2$ . It is appreciated that the starch intake per day and per meal was estimated, from manufacturers' information and analytical data provided for Danish cereals, rather than being determined directly. However, starch content can be specifically and simply determined and starch is often analysed routinely by manufacturers (P.A. Harris, unpublished data).

The authors recognise that, with both weight and starch content being estimated, there may be some inaccuracy in the starch fed/kg variable. However, there is no reason to believe that any inaccuracy would result a systematic bias resulting in a consistent over- or under-estimation of the amount of starch fed/kg bwt.

Vatistas *et al.* (1999b) were able to show the development of EGUS in all their horses within 14 days of being taken from pasture, stabled and entering a simulated training regimen. They were fed Bermuda hay b.i.d. together with 6 kg of a concentrate feed (likely to be high in starch). Similarly, Frank *et al.* (2006) noted that stabling and feeding grain once a day, one hour before the morning hay feed, at 1% bwt together with grass hay to make up a diet providing 1.5x maintenance requirements was an ulcerogenic diet. They suggested that such practices were similar to those found in many show barns and racing stables. These studies and others suggest that grain feeding is likely to be a risk factor for gastric ulceration, although specific guidelines as to the amount have not been previously identified.

High starch diets tend to result in higher VFA production in the upper layers of the gastric mat (Nadeau *et al.* 2000) and possibly higher lactic acid production in the lower layers due to failure of the gastric acid to penetrate the less moist bolus and stop rapid fermentation from occurring (Harris *et al.* 2006). These factors have been implicated in the development of gastric ulceration. When exposed to stomach mucosa *in vitro*, VFAs have been shown to reduce mucosal integrity and to affect the bioelectric properties of the mucosal tissue (Andrews *et al.* 2006, 2008). High starch diets also tend to reflect a high cereal intake and more fluid gastric contents which might promote acid splashing (Argenzio 1999; Lorenzo-Figuera and Merritt 2002). Cereals also tend to be low in calcium and, possibly, other buffering agents that may also contribute to increased risk.

Although the exact link between grain feeding and gastric ulceration is not known, this study has suggested that reducing the total amount of starch given each day as well as the amount provided in each meal may reduce the likelihood of EGUS. Additional energy could be provided through supplemental vegetable oil, which might have other advantages. Cargile *et al.* (2004) concluded that corn oil supplementation might be an effective and inexpensive way to increase the protective properties of the equine glandular gastric mucosa. However, Frank *et al.* (2006) were not able to prevent ulcer formation by oil supplementation in horses in which ulcers had been induced through the use of a high grain ration. It is possible that the level

of supplementary oil provided was not sufficient to counteract the effect of the high grain diets in these horses. Further work should include field trials of oil supplementation in horses at risk of EGUS.

An increased likelihood of EGUS  $\geq 2$  and NG  $\geq 2$  was demonstrated when straw was the only forage provided. These horses had access to straw from their bedding (without any other forage) or were being specifically provided with straw as forage and received none or only very small amounts of hay or haylage (<0.25 kg dry mass/100 kg bwt) in their daily ration. The authors are not aware of this being reported as a risk factor in previous studies. However, this may reflect differences in feeding practices as racehorses and other performance horses are not typically fed high amounts of such low energy forage. Straw is also low in protein and calcium and, therefore, may not provide additional buffering support. It can also be highly lignified and there is the potential that, especially if not chewed thoroughly, some irritation of the gastric mucosa could result from high levels of intake. It is also possible that the high lignin and silica nature of straw alters the fibrous mat in some way to increase the risk of the squamous epithelium being exposed to acidogenic factors. In one study, feeding alfalfa hay and grain resulted in higher gastric pH and less peptic injury to the gastric squamous mucosa than bromegrass hay (Nadeau *et al.* 2000). In a small study in yearlings, exercised on a mechanical walker, alfalfa hay inclusion in the diet reduced the ulcer severity compared with coastal bermuda grass hay (Lybbert *et al.* 2007). The horses were all fed concentrates (T. Lybbert, personal communication). It is not currently known whether any potential beneficial effect of alfalfa may be due to protein intake, protein quality intake, calcium intake or cation-anion difference effects (Nadeau *et al.* 2000; Lybbert *et al.* 2007).

In the present study, time between forage meals of >6 h, compared with frequent forage feeding with intervals of <6 h, increased the likelihood of NG $\geq 2$ . Deprivation of feed for repeated periods has been shown to cause gastric ulcers in the squamous non glandular region but not the glandular region or antrum/pylorus (Murray and Grady 2002). Feed deprivation is associated with highly acidic conditions within the stomach (see Murray and Grady 2002) and the more liquid, less saliva buffered contents may be more easily displaced or 'splashed'. In equine populations where gastric ulceration is common, provision of *ad libitum* forage may significantly reduce the prevalence of EGUS. Where this is not possible or desirable, ensuring frequent access to forage, as well as providing more frequent fibre based, smaller concentrate meals (with reduced starch content) may be advantageous. Finally, using vegetable oil as a low starch, low bulk, high energy supplemental source, where appropriate, may enable more forage to be fed daily whilst maintaining the energy intake.

The current study demonstrated that horses without access to water in the paddock were more likely to have both EGUS  $\geq 2$  and NG  $\geq 2$ . In the study population, 26% of the horses did not have water available in the paddock demonstrating that a simple intervention to make water available could have had a significant impact on the total number of horses with EGUS in this group. Transportation has been associated with an increased risk of EGUS (McClure *et al.* 2005) and may be associated with disturbances in feed as well as water intake. Water intake may result in dilution of gastric fluid (Andrews *et al.* 2006) and therefore pH, although the passage of water through the stomach may depend on the nature of the gastric contents. Interestingly, the number of hours spent without water in the paddock was not a significant factor. This may

be because this study did not have sufficient statistical power to detect subtle differences between groups that were without water for differing periods. However, it may also be the case that the likelihood of EGUS is increased when a horse is deprived of water for a relatively short period (approximately 4 h) and that the risk does not increase as the period without water increases past this initial short period.

Several previous studies have demonstrated potential associations between EGUS and level of exercise (Murray *et al.* 1996; Orsini 2000; Lester 2004; Chameroy *et al.* 2006; Jonsson and Egenvall 2006). However, definitions of levels of exercise and subsequent results from these reports have been inconsistent. This highlights the need in future studies to define workload clearly so that comparisons can be made. It is also important to note that, in normal training as workload changes, so does the management both with respect to the type of feed provided and the nature of any housing. Future studies should, therefore, employ multivariable techniques to account for the confounding effect of many potential risk factors on each other and the outcome of interest.

In the current study, there was no apparent effect of workload on EGUS. This may be due to the fact that even those animals in owner designated 'hard work' were not really working very intensively and perhaps, most importantly, the intake of starch was not linked with designated workload. In Denmark, it is relative cheap to feed horses cereals (a source of starch). Therefore, feeding higher amounts of cereals (oats and barley) and relatively small amounts of hay or haylage is the traditional (less expensive) way of keeping horses/ponies at riding clubs. According to many owners, forage is expensive, difficult to handle and time consuming. In contrast, horses performing at higher levels tend to be fed more meals and less starch, due to the increased use of prepared commercial feeds, typically containing less starch compared to straight grains.

In conclusion, this study highlights the importance of management practices with respect to EGUS. In particular, it suggests that reducing total amount of starch given each day as well as the amount provided in each meal may significantly reduce EGUS prevalence. Ensuring that water is available in any turnout paddock and that straw is not the only provided forage is also advisable. Finally, avoiding leaving horses without forage provision for more than 6 h should also be avoided.

## Manufacturers' addresses

<sup>1</sup>Orion Corporation, Espoo, Finland.

<sup>2</sup>Fort Dodge Animal Health, Southampton, UK.

<sup>3</sup>Video Med, Munich, Germany.

<sup>4</sup>Dodson and Horrell Ltd, Kettering, Northamptonshire, UK.

<sup>5</sup>STATA, College Station, Texas, USA.

## References

- Andrews, F.M. and Nadeau, J.A. (1999) Clinical syndromes of gastric ulceration in foals and mature horses. *Equine vet. J., Suppl.* **29**, 30-33.
- Andrews, F.M., Buchanan, B.R., Smith, S., Elliott, S.B. and Saxton, A.M. (2006) *In vitro* effects of hydrochloric acid and various concentrations of acetic, propionic, butyric or valeric acids on bioelectric properties of equine gastric mucosa. *Am. J. vet. Res.* **67**, 1873-1882.
- Andrews, F.M., Buchanan, B.R., Elliott, S.B., Al Jassim, R.A.M., McGowan, C.M. and Saxton, A.M. (2008) *In vitro* effects of hydrochloric and lactic acids on bioelectric properties of equine gastric squamous mucosa. *Equine vet. J.* **40**, 301-305.
- Andrews, F.M., Reinemeyer, C.R., McCracken, M.D., Blackford, J.T., Nadeau, J.A., Saabye, L., Sotell, M. and Saxton, A. (2002) Comparison of endoscopic, necropsy and histology scoring of gastric ulcers. *Equine vet. J.* **34**, 475-478.
- Argenzio, R.A. (1999) Comparative pathophysiology of non-glandular ulcer disease: a review of experimental studies. *Equine vet. J., Suppl.* **29**, 19-23.
- Bertone, J.J. (2000) Prevalence of gastric ulcers in elite, heavy use Western performance horses. *J. vet. intern. Med.* **14**, 366.
- Cargile, J.L., Burrow, J.A., Kim, I., Cohen, N.D. and Merritt, A.M. (2004) Effect of dietary corn oil supplementation on equine gastric fluid acid, sodium and prostaglandin E2 content before and during pentagastrin infusion. *J. vet. intern. Med.* **18**, 545-549.
- Chameroy, K.A., Nadeau, J.A., Bushmich, S.L., Dinger, J.E., Hoagland, T.A. and Saxton, A.M. (2006) Prevalence of non-glandular gastric ulcers in horses involved in a University Riding program. *J. equine vet. Sci.* **26**, 207-211.
- Davidson, N. and Harris, P. (2002) Nutrition and welfare. In: *The Welfare of Horses*, Ed: N. Martin, Kluwer Academic, Dordrecht. pp 45-76.
- Dohoo, I., Martin, W. and Stryhn, H. (2003) *Veterinary Epidemiologic Research*, AVC, Charlottetown. pp 362-367.
- Ellis, J.M. and Hollands, T. (2002) Use of height-specific weigh tapes to estimate the bodyweight of horses. *Vet. Rec.* 632-634.
- Ethell, M.T., Hodgson, D.R. and Hills, B.A. (2000) Evidence for surfactant contributing to the gastric mucosal barrier of the horse. *Equine vet. J.* **32**, 470-474.
- Frank, N., Andrews, F.M., Elliott, S.B. and Lew, J. (2006) Effects of dietary oils on the development of gastric ulcers in mares. *Am. J. vet. Res.* **66**, 2006-2011.
- Hammond, C.J., Mason, D.K. and Watkins, K.L. (1986) Gastric ulceration in mature Thoroughbred horses. *Equine vet. J.* **18**, 284-287.
- Harris, P.A., Coenen, M., Frape, D.L., Jeffcott, L.B. and Meyer, H. (2006) Equine nutrition and metabolic disease. In: *Equine Manual*, Eds: A. Higgins and J. Snyder, Elsevier Saunders, London. pp 151-222.
- Henneke, D.R., Potter, G.D., Kreider, J.L. and Yeates, B.F. (1983) Relationship between condition score, physical measurements and body fat percentage in mares. *Equine vet. J.* **15**, 371-372.
- Hosmer, D.W. and Lemeshow, S. (2000) *Applied Logistic Regression*, 2nd edn., John Wiley and Sons, New York.
- Jonsson, H. and Egenvall, A. (2006) Prevalence of gastric ulceration in Swedish Standardbreds in race-training. *Equine vet. J.* **38**, 209-213.
- Lester, G.D. (2004) Gastrointestinal diseases of performance horses. In: *Equine Sports Medicine and Surgery*, Eds: K.W. Hinchcliff, A.J. Kaneps and R.J. Geor, Saunders Elsevier, Philadelphia. pp 1037-1043.
- Lorenzo-Figueras, M. and Merritt, A.M. (2002) Effects of exercise on gastric volume and pH in the proximal portion of the stomach of horses. *Am. J. vet. Res.* **63**, 1481-1487.
- Luthersson, N., Hou Nielsen, K., Harris, P. and Parkin, T.D.H. (2009) The prevalence and anatomical distribution of equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark. *Equine vet. J.* **41**, 619-624.
- Lybbert, T., Gibbs, P., Cohen, N. and Sigler, D. (2007) Gastric ulcer syndrome in exercising horses fed different types of hay. In: *Proceedings of the 20th Equine Science Symposium*, Maryland. pp 128-129.
- MacAllister, C.G., Andrews, F.M., Deegan, E., Ruoff, W. and Olovson, S.G. (1997) A scoring system for gastric ulcers in the horse. *Equine vet. J.* **29**, 430-433.
- McClure, S.R., Glickman, L.T. and Glickman, N.W. (1999) Prevalence of gastric ulcers in show horses. *J. Am. vet. med. Ass.* **215**, 1130-1133.
- McClure, S.R., Carithers, D.S., Gross, S.J. and Murray, M.J. (2005) Gastric ulcer development in horses in a simulated show or training environment. *J. Am. vet. med. Ass.* **227**, 775-777.
- Merritt, A.M. (2003) The equine stomach: A personal perspective. *Proc. Am. Ass. equine Practnrs.* **49**, 75-102.
- Murray, M.J., Grodinsky, C., Anderson, C.W., Radue, P.F. and Schmidt, G.R. (1989) Gastric ulcers in horses: A comparison of endoscopic findings in horses with and without clinical signs. *Equine vet. J., Suppl.* **7**, 68-72.
- Murray, M.J., Schusser, G.F., Pipers, F.S. and Gross, S.J. (1996) Factors associated with gastric lesions in Thoroughbred horses. *Equine vet. J.* **28**, 368-374.
- Murray, M.J. (1999) Pathophysiology of peptic disorders in foals and horses: a review. *Equine vet. J., Suppl.* **29**, 14-18.
- Murray, M.J. and Grady, T.C. (2002) The effect of a pectin-lecithin complex on prevention of gastric mucosal lesions induced by feed deprivation in ponies. *Equine vet. J.* **34**, 195-198.

- Murray, M.J. and Eichorn, E.S. (1996) Effects of intermittent feed deprivation, intermittent feed deprivation with ranitidine administration and stall confinement with *ad libitum* access to hay on gastric ulceration in horses. *Am. J. vet. Res.* **57**, 1599-1603.
- Nadeau, J.A., Andrews, F.M., Mathew, A.G., Argenzio, R.A., Blackford, J.T., Sohtell, M. and Saxton, A.M. (2000). Evaluation of diet as a cause of gastric ulcers in horses. *Am J. vet. Res.* **61**, 784-790.
- Orsini, J. (2000) Gastric ulceration in the mature horse: a review. *Equine vet. Educ.* **12**, 24-27.
- Strudsholm, F., Nielsen, E.S., Flye, J.C., Kjeldsen, A.M., Weisbjerg, M.R., Soegaard, K., Kristensen, V.F., Hvelplund, T. and Hermansen, J.E. (1997). Sammensætning og foderværdi af fodermidler til kvæg. *Landbrugets rådgivningscenter Rapport nr. 69*. Landsudvalget for Kvæg. ISSN 0907-5275.
- Vatistas, N.J., Snyder, J.R., Carlson, G., Johnson, B., Arthur, R.M., Thurmond, M., Zhou, H. and Lloyd, K.L.K. (1999a) Cross sectional study of gastric ulcers of the squamous mucosa in Thoroughbred racehorses. *Equine vet. J., Suppl.* **29**, 34-39.
- Vatistas, N.J., Sifferman, R.L., Holste, J., Cox, J.L., Pinalto, G. and Schultz, K.T. (1999b) Induction and maintenance of gastric ulceration in horses in simulated race training. *Equine vet. J., Suppl.* **29**, 40-44.

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