Case Report
Poor performance associated with equine gastric ulceration syndrome in four Thoroughbred racehorses

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Summary
Equine gastric ulceration syndrome (EGUS) is commonly recognised in Thoroughbred racehorses. Although EGUS has previously been associated with reduced athletic performance, no objective studies have been reported.

This case report describes 4 racehorses referred for investigation of poor athletic performance where EGUS was the only abnormal finding during a thorough investigation of all body systems. All horses showed considerable improvement in performance following treatment with omeprazole. Therefore, this is the first report in which evidence is presented suggesting a direct link between EGUS and decreased performance, other causes of poor performance having been excluded.

Introduction
Equine gastric ulceration syndrome (EGUS) is commonly recognised in Thoroughbred racehorses. Studies indicate the prevalence of gastric ulceration in racehorses to be from 52–100% (Hammond et al. 1988; Murray et al. 1996; Vatistas et al. 1999; Begg and O’Sullivan 2003; Bell et al. 2007). The severity of lesions is observed to increase as training intensity increases (Murray et al. 1996; White et al. 2007) and an increased prevalence is reported in horses in active race training and those that have recently raced (Hammond et al. 1988; Murray et al. 1996; Jonsson and Egenvall 2006).

Adult horses with gastric ulceration may show vague clinical signs including poor appetite (Murray et al. 1989; Vatistas et al. 1999), poor body condition or weight loss (Murray et al. 1989; Dionne et al. 2003), poor coat condition (Murray 1991; Vatistas et al. 1999), behavioural changes including an aggressive or nervous attitude (McClure et al. 1999) and, less commonly, mild colic or post prandial discomfort (Murray et al. 1989; Vatistas et al. 1999). However, in many cases no obvious signs are reported and in some horses poor athletic performance or a reluctance to train may be the only complaint. Anecdotally EGUS is associated with reduced athletic performance (Murray 1992; Collier and Stoneham 1997). However, there is little scientific evidence to confirm this. To date, only 2 studies have reported a significant association between presence of gastric ulcers and decreased performance (Vatistas et al. 1999; Jonsson and Egenvall 2006). No objective studies have been reported.

This case report describes 4 horses referred for investigation of poor athletic performance in which EGUS was the only abnormal finding and where improved performance was achieved after treatment with omeprazole.

Case details
During the period between October 2005 and August 2007 100 horses, referred to the Equine Sports Medicine Centre at the University of Bristol for investigation of poor athletic performance, underwent a complete clinical examination that included gastroscopy. Four cases were identified where gastric ulceration was the only clinically significant finding.

Histories
Case 1 was a 7-year-old gelding National Hunt (NH) racehorse. The horse had been raced since a 2-year-old, with over 50 starts and had previously performed well both on the flat and over hurdles. The horse’s form had, however, deteriorated over the previous season and he was reported to be reluctant to work on the gallops. No obvious reason for this could be determined. At the time of the examination the horse was in moderate work and had not raced for 7 months.

Case 2 was a 6-year-old female NH racehorse in full work. This horse had a previous history of recurrent exertional rhabdomyolysis that was being successfully managed by dietary manipulation and oral dantrolene sodium when training. The horse had previously won a NH flat race and been placed over hurdles but had a recent history (over the past 6 months) of slowing towards the end of races. At the time of the examination the horse was in full race training and had last raced 31 days beforehand.

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Case 3 was a 7-year-old gelding NH racehorse in full work. This horse had won a point-to-point with a previous trainer and had been placed once over hurdles. However, during the current season, the horse had failed to perform well and was reported to stop suddenly in races. At the time of the examination, the horse was in full work and had last raced 22 days previously.

Case 4 was a 5-year-old gelding NH racehorse. This horse had raced successfully in France during the previous season, where he won 4 of his 5 starts. Since moving to the UK (5 months previously) he had run disappointingly and was reluctant to exercise on the gallops during training. At the time of the examination, he was in full work and had last raced 41 days previously.

**Clinical examination**

All 4 horses underwent a thorough clinical examination at rest and during high-speed treadmill exercise over a period of 3 days. This included a lameness assessment (horses were observed at walk and trot, in a straight line and on the lunge), endoscopic examination of the upper airways at rest and during strenuous exercise, exercising ECG, post exercise endoscopy of the lower airways, tracheal wash and bronchoalveolar lavage, blood samples for haematology and measurement of pre- and post exercise muscle enzymes and gastroscopy.

Horses were starved for 12 h before gastroscopy and water was withheld for 3–4 h. Horses were sedated with detomidine hydrochloride (Domosedan) (0.01 mg/kg bwt i.v.) and butorphanol (Torbugesic) (0.02 mg/kg bwt i.v.) and gastroscopy was performed using a 3.2 m gastroscope. The squamous and glandular mucosae were examined throughout the stomach including the fundus, greater curvature, lesser curvature and the antrum and pyloric regions. Ulcers were graded on a scale 0–4, according to the recommendations of the EGUS Council (Anon 1999):

- **Grade 0**: The epithelium is intact and there is no appearance of hyperaemia or squamous hyperkeratosis.
- **Grade 1**: The mucosa is intact, but there are areas of reddening or squamous hyperkeratosis.
- **Grade 2**: Small, single or multifocal lesions.
- **Grade 3**: Large, single or multifocal lesions or extensive superficial lesions.
- **Grade 4**: Extensive lesions with areas of apparent deep ulceration.

**Fig 1**: Case 1 had multiple shallow ulceration of the squamous mucosa extending up to the fundus (a) and deeper ulceration at the lesser curvature (b).

**Fig 2**: Case 2 had linear ulcers extending from the margo plicatus (a) and multiple lesions at the pylorus.
**Clinical findings**

In all 4 cases no abnormal findings were found during a resting examination. Video-endoscopy during high-speed treadmill exercise confirmed that there were no upper respiratory tract obstruction and post exercise tracheal wash and broncho-alveolar lavage revealed no evidence of lower airway disease. No cardiac abnormalities were detected at rest or during exercise. Muscle enzymes were normal in all 4 horses and no lameness was detected. The only finding of clinical significance in all 4 cases was that of gastric ulceration.

**Case 1** had grade 4 EGUS with extensive superficial ulceration radiating from *margo plicatus* up to and including the fundus (Fig 1a) with some areas of deeper ulceration at the lesser curvature (Fig 1b). Only a few small ulcers were observed in the glandular mucosa, at the pylorus.

**Case 2** had grade 3 EGUS, with multiple superficial linear ulcers in the squamous mucosa extending from the *margo plicatus* towards the fundus (Fig 2a). Ulceration of the pylorus was also observed (Fig 2b).

**Case 3** had grade 4 EGUS, with multiple deep, punched out ulcers within the squamous mucosa (Fig 3a) with several linear ulcers within the glandular mucosa (Fig 3b).

**Case 4** had grade 4 EGUS with multiple ulcers of the squamous mucosa adjacent to the *margo plicatus* of the greater (a) and lesser (b) curvatures.

**Treatment**

All horses were treated with omeprazole (GastroGard)\(^5\) at a dose of 4 mg/kg bwt per os q. 24 h for 4 weeks. Case 3, which had marked glandular ulceration, was also treated with sucralfate at a dose of 20 mg/kg bwt per os q. 8 h for 2 weeks. Following the initial treatment, all 4 horses were kept on a maintenance dose of omeprazole (1 mg/kg bwt per os) whilst in training.

In addition to medical treatment, management changes were recommended in 3 of the 4 horses. This included daily access to pasture and the provision of *ad libitum* forage.
Case 2 was already kept at pasture and no further recommendations were made regarding management. During the treatment period, Case 3 was turned out to pasture for one month before resuming race training. The other 3 horses remained in full work. Once training was resumed, Cases 1 and 3 were turned out to graze daily. However, management changes were not implemented in Case 4.

**Outcome**

All horses showed marked improvement in their racing form after treatment (Table 1).

**Case 1:** Gastroscopy was repeated after one month. No ulceration was observed in the squamous mucosa. Small areas of hyperaemia (grade 1) were observed at the pylorus. However, no areas of erosion or ulceration were observed. The horse resumed race training and was reported to be more willing to work. The horse raced 3 months later, was placed in the second race and won the third race.

**Case 2:** Returned to racing after one month. The horse was placed in its first race and won the next 2 races.

**Case 3:** Underwent repeat gastroscopy after one month. This revealed no ulcers within the squamous mucosa. Occasional areas of hyperaemia were observed within the glandular mucosa. However, no erosions or ulceration were observed. The horse raced 2 months later and was placed in the first race and won the second race.

**Case 4:** Showed a dramatic improvement in its willingness to work on the gallops and returned to racing successfully after 6 weeks, winning the first race and being placed second in the next race.

**Discussion**

This is the first report in which evidence is presented suggesting a direct link between EGUS and decreased performance, other causes of poor performance having been excluded.

Poor performance in racehorses is frequently multifactorial (Morris and Seeherman 1991; Martin et al. 2000; Lane et al. 2006). It is often, therefore, difficult to assess the effect of any one component on athletic performance. A thorough clinical examination both at rest and during strenuous exercise is recommended in order to make a complete assessment in horses referred for investigation of poor performance, hence optimising the chance of a successful return to racing.

Anecdotally EGUS has been associated with poor performance (Murray 1992; Collier and Stoneham 1997). However, there has been little scientific study to confirm this. To date, only 2 studies have attempted to examine any association between the presence of ulcers and performance: Vatistas et al. (1999) showed a significant association between the presence of gastric ulceration and decreased performance (described as below the trainers’ expectations) in Thoroughbred racehorses. Poor performance was reported to be associated with the presence of gastric ulcers independent of their severity or the number of ulcers. Jonsson and Egenwall (2006) also found a significant association between the presence of ulcers and actual compared with expected performance in Standardbred racehorses. Horses with ulcers were significantly more likely to perform ‘worse than expected’ than ‘as expected’ (odds ratio = 0.3) or ‘better than expected’ (odds ratio = 0.2). In that study the effects of different grades of lesions on performance was not examined. Neither study examined objective measures of performance.

The mechanism by which gastric ulceration may affect performance has not been established. It has been proposed that reduced performance may arise as a direct consequence of gastric pain or as a result of indirect factors such as decreased food intake (Vatistas et al. 1999).

Murray (1991) suggested that the effect of ulcers on performance could only be appreciated once ulcers had been treated. All 4 horses described here raced successfully following treatment with omeprazole. Previously, Johnson et al. (2001) reported improved performance after horses with gastric ulceration were treated with omeprazole, by virtue of the fact that 46% of horses finished races in the top 3 positions after treatment, compared with 40% before treatment. Omeprazole has been shown to have no performance-enhancing properties per se in healthy animals (McKeever et al. 2006) and hence any improvement in performance is most likely due to resolution of lesions.

Omeprazole is the treatment of choice for EGUS (Johnson et al. 2001; Orsini et al. 2003; Lester et al. 2005). This drug is a K⁺/H⁺ ATPase ‘proton pump’ inhibitor that results in decreased gastric acid production by blocking the final step in parietal cell acid secretion (Dauro et al. 1999, Merritt et al. 2003). Studies have shown resolution of gastric ulceration after 28 days of treatment with omeprazole (Andrews et al. 1999; Doucet et al. 2003). This was confirmed in 2 of the cases described here in which gastroscopy was repeated following treatment.

One horse in this study was treated with sucralfate in addition to omeprazole. Sucralfate is a complex salt of sucrose and aluminium hydroxide that has been used successfully to treat gastric and duodenal ulcers in humans (Jensen and Funch Jensen 1992). It acts by adhering to the ulcerated mucosa, thereby protecting it from further damage. It is also reported to inhibit pepsin, enhance the protective mucus bicarbonate layer and stimulate local protective prostaglandins and it has therefore been speculated that sucralfate may also aid healing.

### Table 1: Mean Raceform ratings and total earnings (GB £ Sterling) for 2 races before and 2 races after treatment of gastric ulceration in 4 Thoroughbred racehorses

<table>
<thead>
<tr>
<th>Case</th>
<th>Mean Raceform rating (before treatment)</th>
<th>Mean Raceform rating (after treatment)</th>
<th>Earnings (before treatment)</th>
<th>Earnings (after treatment)</th>
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<td>146</td>
<td>£9282</td>
<td>£29,094</td>
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of glandular ulcers in horses (MacAllister 1999; Orsini 2000). Recent reports have questioned the efficacy of sucralfate in treating squamous ulceration (Borne and MacAllister 1993; Orsini et al. 2003). However, to date the effect of sucralfate on glandular ulcers in horses has not been studied.

Management changes including daily access to pasture, feeding of ad libitum forage and the provision of smaller, more frequent concentrate meals may provide prolonged reduction of gastric acidity (Murray and Schusser 1993) and are commonly suggested in an attempt to reduce the recurrence and severity of gastric ulceration. In addition, the type of forage may be important; it has been shown feeding alfalfa hay results in an increase in gastric acid pH and a reduction in the number and severity of ulcers compared with feeding grass hay (Nadeau et al. 2000). However, in light of studies that suggest the prevalence of ulceration is high both in stabled horses receiving ad libitum forage (Murray and Eichorn 1996) and racehorses with access to pasture (Jonsson and Egnell 2006; Bell et al. 2007), it is unlikely that management changes alone will result in resolution of ulcers or prevent the recurrence of EGUS in racehorses that remain in race training.

Once treatment of gastric ulcers has been discontinued, recurrence of ulcers is common in horses remaining in training (Andrews et al. 1999). Low dose (1 mg/kg bwt per os q 24 h) omeprazole has been shown to be effective in preventing recurrence of ulcers (Doucet al. 2003; McClure et al. 2005; White et al. 2007) and was instituted in all of the cases reported here. It should be noted, however, that under British Horseracing Authority regulations, omeprazole is classed as a prohibited substance. Detection times for omeprazole are short (one day). However, metabolites of omeprazole can be detected for up to 6 days in horse urine (Chung et al. 2004) and a withdrawal time of 8 days prior to racing is recommended. It has recently been shown that ulcers may develop within 8 days in horses engaged in both light and heavy training regimes (White et al. 2007) and hence it is possible that ulceration may recur in the 8 days prior to racing when omeprazole is withdrawn. However, Lester et al. (2005) found that withdrawal of omeprazole after 28 days of treatment resulted in only subtle worsening of ulcer scores during the subsequent 28 day period, despite horses remaining in race training.

In conclusion, this case series suggests that EGUS may be an important cause of poor athletic performance in some racehorses and that treatment with omeprazole may result in a successful return to racing.

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Manufacturers’ addresses

1Sato, Upsala, Sweden.
2Ziver Animal Health Ltd, Sandwich, Kent, UK.
3Fort Dodge Ltd., Southampton, Hampshire, UK.
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