Australian Government
Rural Industries Research and Development Corporation

Risk Factors for Gastric Ulceration in Thoroughbred Racehorses

RIRDC publication number 08/061

Australian Government
Rural Industries Research and Development Corporation

Risk Factors for Gastric Ulceration in Thoroughbred Racehorses

by Guy D. Lester, Ian Robertson, and Cristy Secombe

May 2008
RIRDC Publication No 08/061
RIRDC Project No UMU 33A

ISBN 1 74151 650 1
ISSN 1440-6845

Risk Factors for Gastric Ulceration in Thoroughbred Racehorses
Publication No. 08/061
Project No. UMU 33A

Researcher Contact Details
Associate Professor Guy D. Lester
School of Veterinary and Biomedical Sciences
Murdoch University
Murdoch, Western Australia 6150
AUSTRALIA
Phone: 08.9360 7676
Fax: 08.9360 2603
Email: G.Lester@murdoch.edu.au

In submitting this report, the researcher has agreed to RIRDC publishing this material in its edited form.

RIRDC Contact Details
Rural Industries Research and Development Corporation
Level 2, 15 National Circuit
BARTON ACT 2600
PO Box 4776
KINGSTON ACT 2604

Phone: 02 6271 4100
Fax: 02 6271 4199
Email: rirdc@rirdc.gov.au.
Web: http://www.rirdc.gov.au

Published in May 2008

Foreword

Ulceration of the stomach is a common health problem of performance horses across the world. The reported prevalence in racing thoroughbred or standardbred horses is high, commonly quoted between 55 and 90%. The economic impact of disease is difficult to calculate, as the impact on athletic performance has not been accurately determined. There are well defined costs attributable to diagnosis, medication, and the labour required for treatment. The risk factors for gastric ulceration are poorly defined, principally because most studies have not had access to populations with variable prevalence. In a study published in 2005 examining the effect of treatment on ulcer healing Prof. Lester and his colleagues unexpectedly exposed a large variability in ulcer prevalence between stables of racing thoroughbreds in Western Australia. Prof. Lester recognised that this population of horses was ideal to investigate risk factors of this disease. In December 2005 a RIRDC-funded study on a larger population of thoroughbreds throughout Western Australia commenced.

Data were collected from 402 racehorses under the care of 37 different trainers. Ulcer disease was determined through the use of endoscopy in sedated horses, where the squamous gastric mucosa was graded for evidence of disease. A large number of variables were collected for each trainer and for each animal. These were tested for significance using univariate methods and those factors with a p-value < 0.25 were subsequently entered into a multiple logistic regression model. As anticipated by the authors there was a significant effect of the trainer on ulcer prevalence; trainer was incorporated as a random effect in the final model. There were several trainers that had no horses showing moderate or severe lesions.

There were a large number of factors that significantly impacted on ulcer disease when examined independently, but only 6 variables remained in the final multivariate model. The trainers of affected horses were more likely to report that animals with moderate or severe ulcer disease had difficulty in maintaining body condition, and not surpringly this was closely related to the length of time that the horse had been in the current campaign. Although not included in the final model, a poor appetite was also significantly correlated with both of these factors. It appears likely therefore that ulceration may limit feed intake and, as the training program continues and the caloric demands increase, the horse will begin to demonstrate outward signs of weight loss. This may contribute to reduced athletic performance. There were a number of environmental factors that were also significantly associated with gastric ulceration. Training at the same location that the animal is stabled and turning out with other animals were both associated with a decreased risk of disease. Playing of a radio within the barn increased risk. Horses that demonstrated sterotypic or altered behaviour, specifically crib biting or wind sucking, were more likely to have ulcers than those that did not. These results indicate that both physiological and psychological stress may be imporant determinants of ulcer disease in this population.

Ulcer disease is clearly a multi-factorial problem. The results of this study provide trainers, owners, and veterinarians with important information regarding the prevalence and likely clinical signs of ulcer disease. It also highlights the importance of reducing environmental stress in reducing the overall prevalence within individual stables. Horses are herd animals, and maximising contact between horses would seem appropriate. At the time of the study only a relatively small percentage of horses were under treatment with anti-ulcer medication. Ulcer disease was found in many of these treated horses indicating the need for continued research on efficacy of drug formulations and dose rates.

This project was funded from industry revenue which is matched by funds provided by the Australian Government.

This report, an addition to RIRDC's diverse range of over 1800 research publications, forms part of our Horse R&D Program, which aims to assist in developing the Australian horse industry and enhancing its export potential.

Most of our publications are available for viewing, downloading or purchasing online through our website:

- downloads at www.rirdc.gov.au/fullreports/index.html
- purchases at www.rirdc.gov.au/eshop

Peter O'Brien
Managing Director
Rural Industries Research and Development Corporation

Acknowledgments

We would like to acknowledge the assistence of many thoroughbred trainers and owners throughout Western Australia who particpated in the study. The investigators would also like to acknowledge the expertise and help of Dr. Rachael Smith, Dr. Belinda Black, Ms. Carissa Lalich, and Ms Tania Sundra who assisted in the collection of data. We would also like to acknowledge the assistence of the following veterinarians in faciliating contact with trainers in their region: Dr. Charlie Stewart, Dr. Julia Vines, Dr. Warwick Vale, Dr. Graham Harradine, and Dr. Malcolm Webb.

Abbreviations

EGUS: Equine Gastric Ulcer Syndrome

Contents

Executive Summary

What the report is about

Gastric ulceration represents an important health concern to the performance horse industries. Economic impacts of this disease are difficult to quantify but include the costs associated with diagnosis, cost of medication, and labour involved in administering treatment. Perhaps of even greater economic significance are the costs attributable to reduced athletic performance. Superimposed on any economic impacts are the emerging industry concerns regarding the welfare of performance horses. The reported high incidence of disease across the world is of concern given the common lay opinion that peptic ulcer disease in humans is commonly correlated with emotional stress. The focus of this report is to identify specific factors that may be involved in the development and maintenance of gastric ulcers in racing thoroughbreds.

Who is the report targeted at?

The primary target group of the report is the thoroughbred racing community. It is however likely that risk factors will be common to other industries where animals are performing high levels of exercise. The information will also guide attending veterinarians with respect to identifying horses at risk of ulceration.

Project Aims

As stated above the principal aim of the project was to elucidate specific risk factors that may predispose racing thoroughbreds to gastric ulceration. The ideal outcome would be to identify factors that could be easily manipulated through management to reduce the risk and/or severity of disease. This should be of benefit to both trainers and owners. Another aim was to investigate associations between disease and clinical signs, as well as disease and athletic performance.

Study Methods

Data were collected from 402 thoroughbred horses over a 10 month period in 2006. There were 37 trainers enrolled in the study and they were based from the following regions in south Western Australia: Albany 420 km south of Perth; Mandurah/Larkhill 60 km south of Perth; Swan Valley 60 km northeast of Perth; Bunbury 170 km south of Perth; and Ascot/Belmont located within the metropolis of Perth. Endoscopy was performed in fasted free-standing horses and the squamous mucosa of the stomach was evaluated for ulcer disease. The gastric squamous mucosa was scored using a simplified system that ranged from 0 to 3. Grade 0, normal - intact mucosal epithelium with or without reddening or hyperkeratosis; Grade 1, mild - single or multiple small ulcers; Grade 2, moderate - single or multiple large ulcers; and Grade 3, severe - extensive ulceration with coalescing of ulcerated areas. Horses graded as 0 or 1 were classified as not having ulcers and those graded as Ulcer score 2 or 3 were classified as having ulcers. Trainers were then asked a series of questions pertaining to each horse. All variables were then assessed independently for association with gastric ulceration and those with a significance of $P \leq 0.25$ in the univariable analyses were considered eligible for inclusion in the logistic multiple regression.

Key Findings

The overall prevalence of ulcer disease was 53%. This was reduced to 33% of the entire population when the definition of ulceration was restricted to horses having either moderate (grade 2) or severe (grade 3) disease. As anticipated, there were highly significant differences between trainers with respect to ulcer prevalence. The time that an animal had been in training was significantly associated with the prevalence of ulcers, with the odds of a horse developing moderate or severe ulceration increasing by 1.7 times for every week that the horse was in training. The location of exercise was also important with animals exercised at a track on the property where they reside being 3.3 times less likely to have ulcer disease than those that were not. Animals that the trainer reported as having difficulty in maintaining adequate body condition were more likely to have moderate or severe

ulceration than those where no problems were reported. Interestingly, this was closely correlated with the time in work and quality of appetite. A poor appetite in response to gastric ulceration may be the critical response that leads to problems in maintaining body condition and reduced athletic performance. As would be expected these problems are accentuated with the length of time in work when caloric demands would be at their greatest. There is some evidence that psychological stress may also be a key factor in the development of squamous ulcer disease. Animals demonstrating stereotypic behaviour were more likely to have disease; the most significant vice was crib-biting or wind sucking. It could be argued that this vice could lead to ulceration or conversely could arise as a relief mechanism for on-going gastric pain. Alternatively crib-biting, along with other stereotypic behavioural traits, may simply be a marker for environmental stress, which in turn may independently increase the risk of ulcer disease and increase the likelihood of developing an abnormal behavioural trait. Horses that had access to some turnout were less likely to have ulcer disease; perhaps more importantly horses that not only had access to turnout but were turned out with other horses were even less likely to have disease. This indicates that horse-to-horse contact may be an important preventative strategy against ulcer disease. Playing of the radio within the barn was associated with increased risk of ulceration, indicating that this may be a form of stress for some animals. Of interest was the fact that we were unable document any direct link between aggression, diet, gender or age with ulcer disease.

Implications and Recommendations

The prevalence of ulcer disease is greater in metropolitan stables than in rural or semi-rural stables. Location by itself is not the critical factor; rather it appears that features common to these environments are increasing or decreasing the risk of ulcer disease. It may be that the focus of reducing disease is in evaluating the environment and making appropriate adjustments to reduce any stress on the animal. It appears that direct horse contact is an important protective strategy.

It is also clear that the generation of ulcer disease is multi-factorial and therefore adjusting any one factor may have negligible impact on overall disease prevalence. It is also important to consider what impact ulceration has on the individual horse. We would contend that horses, as individuals, differ widely in the expression of clinical signs that result from ulcer disease. Some horses with severe ulceration appear to demonstrate little to no outward signs of disease and continue to perform successfully, whereas in other animals ulcer disease appears to be highly debilitating and has a dramatic effect on appetite, body condition, and therefore athletic performance. Many of these animals can be effectively managed with anti-acid drugs, but may also respond to radical changes in their training environment.

Introduction

Anatomy

The horse is a hindgut fermenter, with an extensive and complex caecum and large colon suitable for fermentation of structural carbohydrates and soluble carbohydrates that escape digestion in the small intestine. Horses have a simple stomach, similar in shape to that of domestic carnivores and omnivores. There are significant differences however between species with respect to the internal lining of the stomach. The non-glandular, squamous mucosal lining of the distal oesophagus spreads through the lower oesophageal region and covers the proximal or top half of the stomach. This region of the stomach has minimal defensive strategies to protect itself from acid-induced damage. The *margo plicatus* defines the lower border of the squamous mucosa where it abuts glandular mucosa. The glandular mucosa covers the remainder of the stomach through the antrum and down to the pylorus. On a histological basis the glandular mucosa is further divided into 3 areas: the cardiac, fundic, and pyloric regions. The cardiac gland region is a narrow strip of mucosa that lies immediately beneath the *margo plicatus*. The fundic glandular mucosa covers most of the lower half of the stomach and is responsible for the production of hydrochloric acid. The natural bend to the stomach permits further anatomical classification into greater and lesser curvatures.

Definitions

Spontaneous and drug-induced gastric ulceration occurs in humans and in many domesticated species, including horses. Equine gastric ulcer syndrome (EGUS) is not a single disease entity, but rather a term that encompasses a group of distinct disorders that can affect horses of all ages. [1] These include not only primary gastric disorders, but also related diseases of the distal oesophagus and the proximal duodenum. Many diseases share aspects of pathology, but likely differ widely in terms of primary pathophysiology. In recent years there has been a focus on bacterial-induced gastritis and ulceration in humans and a number of other species, including dogs, cats, pigs, monkeys, and captive cheetahs. The causative bacteria, *Helicobacter* species, have not been definitively associated with gastritis or ulceration in horses although a species of *Helicobacter* may populate the normal stomach.

- Neonatal gastric ulceration. As the name indicates this syndrome is usually limited to diseased or otherwise highly stressed newborn foals. Ulceration and occasional perforation frequently, but not exclusively, occurs in the cardiac gland region of the stomach beneath the *margo plicatus*. This syndrome is often clinically silent due to the co-existence of severe primary diseases such as systemic sepsis or peripartum asphyxia syndromes. The first signs may not be apparent until fatal perforation has occurred. Attenuated mucosal protection through reduced blood flow is a likely key component of the pathophysiology of neonatal gastric ulceration.
- Gastro-duodenal ulcer disease (GDUD). This form of EGUS occurs primarily in suckling foals and in its most severe form involves the proximal duodenum, pylorus, stomach, and distal oesophagus. [2] It is highly likely that the initial lesion in affected foals is a diffuse duodenitis. This initially results in a functional delay to gastric emptying with secondary gastric and esophageal irritation, probably due to prolonged exposure of susceptible mucosa to acidic luminal contents. During the healing process strictures may form in the duodenum and/or pylorus which result in a mechanical obstruction to emptying, again leading to gastric distention and secondary gastric and oesophageal erosion. Of all of the forms of EGUS this syndrome is the one most likely to have a precipitating infectious component. This is further supported by the observation that cases frequently occur in clusters and are often preceded by episodes of diarrhoea. Acid suppression is a key component in the management of GDUD when delayed gastric emptying (either functional or mechanical) is present, but does not have a role in prophylaxis.
- Glandular and pyloric ulceration. Experimental induction of glandular ulceration is easily achieved using repeated high doses of non-steroidal anti-inflammatory drugs, such as phenylbutazone. [3] Lesions in the glandular mucosa also occur spontaneously in both athletic non-athletic horses and often co-exist with squamous mucosal lesions. They are usually seen in response to some form of stress, such as training or concurrent diseases. Glandular lesions and

lesions around the non-glandular pylorus are often associated with the most overt clinical signs of EGUS, such as post-prandial colic and inappetence.

- Squamous mucosal ulceration. Most discussion of ulcer disease in horses refers to erosion or ulceration of the non-glandular squamous mucosa, the most common form of EGUS in adult performance horses. The regions adjacent to the *margo plicatus* are most frequently diseased and are usually more prominent on the lesser curvature between the cardia and the *margo plicatus*. [4,5]

Gastric squamous mucosal ulceration

Gastric squamous mucosal ulceration has a substantial economic impact on the horse industry due to the costs associated with its diagnosis and treatment. Confirmation of disease is made by endoscopic examination of the horse stomach, a relatively expensive diagnostic test. Management of confirmed or suspected disease typically requires the administration of medication given between one and three times daily. The cost of the medication can be substantial, as much as $60 per day for horses in race training receiving one of the registered commercial omeprazole product. This figure does not include the added labour costs associated with administering the medications. Poor athletic performance is considered to be a key manifestation of gastric squamous ulcer disease. [6-8] Consequently, the true economic impact of the disease is difficult to quantify due to problems estimating the magnitude of lost earnings attributable to reduced athletic performance.

Clinical Signs

The clinical signs attributable to squamous ulceration in adult horses are real, but often vague. [1,6] The most common and consistent finding associated with squamous ulceration relates to problems in feed consumption, with affected individuals taking longer than expected to consume a concentrate-based meal. Trainers often report difficulty in maintaining adequate body condition. Additional signs may include difficult behaviour (nervousness, aggression), postprandial abdominal pain, and rough hair coat. There is little doubt that the clinical manifestations of ulcer disease are inconsistent between animals; some animals with endoscopic evidence of severe ulceration may not demonstrate any classical signs of disease. Improved performance and behaviour in response to anti-acid therapy may unmask previous sub-clinical disease in these horses. Conversely, it is not uncommon to see animals with mild ulceration or erosion that demonstrate classical signs of disease and also improvement in response to anti-acid therapy.

Prevalence

The introduction of 2.5 to 3.0 meter videoendoscopes has provided the means to estimate the prevalence of gastric ulceration across a variety of horse types and activities. The prevalence of lesions in the squamous mucosal region exceeds that of the glandular area. Most studies have utilized racing or training thoroughbreds where the reported point prevalence of squamous mucosal disease varies between 55 and 100%. The prevalence and severity appears to be associated with activity type and intensity, feeding practices, and housing.[8-14] standardbreds and thoroughbreds in race training appear to be at greatest risk for ulceration, with reported frequencies commonly between 80 and 95%. Prevalence data from Australia reported that 86% of animals examined had squamous mucosal ulceration.[9] The lowest incidence in active horses is in elite western performance animals where 40% have ulcer gastric disease.[15] A study of Canadian standardbreds not in active training reported an ulcer rate of around 16%; this increased to 28% in pre-race training and 63% in racing animals.[16] The same investigators in a later study reported an ulcer frequency of 38% at the commencement of training and an increase to 72% after two months of training and then 88% in horses after 4 months.[5]

Risk Factors

Despite the reported high prevalence of ulceration and the widespread administration of anti-ulcer medications, there are very few statistical data on risk factors for ulceration. This is due to the fact that prevalence appears to vary little within individual study populations. The observation that disease is most prevalent in racing thoroughbreds has prompted estimation of risk factors and formulation of possible preventative strategies. Postulated factors include a high concentrate diet, stall confinement,

intense exercise and racing, and "stress".[1,4,12,14] A relationship between gastric ulceration and crib-biitng has been postulated.[17] Consequently, the traditional approach to management usually involves medication with changes to feeding frequency, diet composition, and/or exercise. Concurrent medication, including the administration of non-steroidal anti-inflammatory drugs, does not appear to be an important risk factor for squamous mucosal ulceration. The exception is furosemide, which has been linked to ulcer severity.[8]

Age and gender

Age appears to be a minor factor in determining ulcer frequency and severity within a population.[7,12,14] Ulceration tends to be less severe in young horses, and is probably related to the length of time in race training. Gender is not considered to be an important risk factor, although a recent study reported an interaction between gender and age. The relative risk for ulceration increased with increasing age in geldings, while reducing with age in females and entire males.[14] Vatistas and others (1999) reported that the prevalence of squamous mucosal ulceration was greater in geldings (94%) than in colts (78%) or fillies (82%).[7]

Effect of training

The prevalence data supports a strong link between exercise and gastric ulceration. In a post-mortem survey of thoroughbreds in Hong Kong Hammond and others (1986) reported that horses in training had a greater prevalence of ulceration than those that had been free from racing for a month or longer (80% versus 52%).[4] The difference between these two groups was even greater when ulcer severity was examined. Forty five per cent of horses that were in training had moderate or severe disease compared with only 5% of horses retired or spelling. As part of a larger study investigating the prevalence and severity of gastric ulceration in relation to clinical signs, Murray and others reported a greater prevalence and severity of disease in horses in training compared to those not in training.[6]

Vatistas and others examined the effect of exercise on gastric ulceration in 30 healthy thoroughbreds under controlled conditions.[18] The investigators assessed the animals for gastric ulcers before, during, and at the completion of 8 weeks of training on a high-speed treadmill. All animals developed moderate to severe gastric squamous mucosal ulceration that persisted for the duration of the experiment. When training ceases spontaneous improvement occurs in some horses.[14] A clear effect of training on disease prevalence has also been reported in standardbreds in Canada and Sweden.[5,10]

Physiological mechanisms linking high-intensity exercise and racing with squamous mucosal ulceration are not clear. Exercise results in increased intra-abdominal pressure, reduced gastric volume, and exposure of gastric squamous mucosa to the acidic contents normally located within the fundus.[19,20] Whether exposure during the relatively brief period of high-intensity exercise is sufficient to induce or propagate ulceration is not known, but *in vitro* contact of squamous mucosa with hydrochloric acid leads to damage within minutes.[21] Exposure of previously ulcerated mucosa to acidic contents during maximal exercise could elicit acute gastric pain. This could provide a physiological basis to the association between ulcer disease and poor athletic performance. Relocation of healthy horses from a pasture to a stall will often result in gastric squamous ulceration within 7 days. This occurs in the absence of exercise or exposure to concentrate feeds.

Concurrent disease

Gastric ulceration is commonly reported in horses with other diseases of the gastrointestinal tract. These include impaction colic, colonic tympany, intussusception, and primary inflammatory bowel diseases such as duodenitis proximal jejunitis.[22] In a study of horses presenting to a referral veterinary hospital with the complaint of abdominal pain 49 of 100 animals had endoscopic evidence of gastric ulceration.[23] It was also noted that the prevalence of ulcer disease was higher in animals that responded to medical therapy in contrast to those that required surgical treatment.

Diet

Diets fed to exercising horses are generally higher in energy and lower in roughage than diets fed to horses performing low-level exercise or at rest. The prevalence of ulceration is clearly higher in exercising animals, leading to a belief that diet could be an important precipitating factor in the initiation and propagation of gastric ulceration.[4] The impact of diet has been difficult to investigate, given the consistency of feeding practices in many of the reported clinical trials. We do know that feed deprivation leads to increased gastric acidity, a reduction in intraluminal pH, and the development of squamous mucosal ulcers.[24] The importance of acid in the initiation of ulcer disease in this model was demonstrated by the improvement noted when animals were medicated with the acid suppressing drug ranitidine.[25] In a study of ponies fed either a hay-only diet or a mixed hay and grain diet for a minimum of 14 days the prevalence of non-glandular ulceration was greater in the latter group suggesting a negative impact of soluble carbohydrate.[26]

The group at Tennessee, led by Dr. Frank Andrews, has published several studies that have examined the association between diet and gastric ulceration. Several of their studies have utilised animals with gastric cannulae, a mechanism allowing routine collection of gastric fluid. In an unexpected finding the group reported a lower intragastric pH and increased incidence of gastric ulceration in horses fed a diet of grass hay (bromegrass) compared to feeding of a grain and legume hay diet; the latter diet being more commonly fed to exercising horses.[27] The authors suggested that grain-legume hay diet might be protective through a buffering effect of dietary protein and/or calcium on intra-luminal pH. Alternatively there was a developing appreciation that limited fermentation of feed was occurring in the stomach, indicating a potential role of resultant volatile fatty acids in either initiating or protecting against ulceration. The group investigated these effects using an *in vitro* chamber system where initial studies indicated that the volatile fatty acids acetic, butyric and propionic acid could potentiate the harmful effects of hydrochloric acid on the squamous mucosa. [28] A marked effect on the bioelectric properties of gastric squamous mucosa was reported after the addition of the valeric acid.[29] Importantly these detrimental effects were present at a pH of less than or equal to 7.0. The concentrations of volatile fatty acids used in the initial studies were thought to exceed those found spontaneously. Consequently, the studies were repeated using lower physiologic concentrations of VFAs.[30] Acetic acid, at a concentration of 20 mM, in the presence of an intra-gastric pH less than or equal to 4.0, was harmful to the non-glandular mucosa. This mimicked the concentrations observed by he same group in horses fed 5.5 kg of lucerne hay and 2.6 kg of grain[27]. Based on these findings, the authors recommended maximum grain intake of 0.5 kg/100 kg BW no more frequently than every 6-8 hours. This is in accordance with the recommendation of feeding multiple small concentrate feeds, rather than large feeds once or twice daily which is common in clinical practice.

Nevertheless the role of diet composition in creation and propagation of squamous mucosal ulcers remains controversial. Increasing the frequency of feeding is often recommended to reduce ulceration. This common recommendation stems from research indicating that feed deprivation results in lower intragastric pH, and that alternating 24 hour periods of feed deprivation and free choice access to hay results in induction of squamous mucosal ulceration in normal adult horses. [25]

Environment

Gastric squamous mucosal ulceration was induced in horses simply by confining horses normally maintained at pasture to a stall for 7 days.[25] These authors suggested that this effect was most likely attributable to altered feeding behaviour.

Physiological stress

Stress plays an important role in the induction of gastric ulcers in humans. Similar mechanisms may be important in the initiation of glandular ulcers in sick adult horses and newborn foals, but are unlikely to play a direct role in squamous mucosal ulceration.

Psychological stress

The role of psychological stress is not known, but some authors have considered that this form of stress is unlikely to be important in the pathophysiology of ulcer disease. Several studies have reported that squamous ulcer prevalence may be greater in horses perceived to be 'nervous' or 'anxious', although differences were not significant. It has been hypothesised that anxiety could be associated with persistent tension of abdominal muscles which could mimic the reduction in gastric volume seen during exercise (*AM Merritt, personal communication*). Gastric acid production in response to a variety of stimuli, including insulin-induced hypoglycaemia and offering by sight and smell only a bucket of grain without allowing the horses to consume it was measured in horses with gastric cannulae.[31] Teasing with feed led to a transient, but non-significant increase in gastric acidity.

While positive steps have recently been made in understanding the pathophysiology of ulceration, there remains a lack of critical data that can be used to formulate effective changes in management. Currently anti-ulcer medication remains the sole management focus, but prolonged medication is costly for the average owner and labour-intensive for the trainer. We recently had the opportunity to screen a large population of thoroughbred racehorses in Western Australia as part of a study that contrasted the efficacy of two commercial anti-acid preparations.[32] The overall point prevalence of squamous mucosal ulceration in this population was also high, but varied widely between stables, ranging between 22 and 94%. The population reflected diversity within the training population in terms of location (metropolitan, semi-rural, and rural), stable design (closed, semi-open, walk in/walk out), visual contact with other horses, and access to turnout (none, < 2 hours daily, and > 2 hours daily). Insufficient numbers of properties sampled precluded statistical analyses of stable factors, but feeding practices, access to pasture turnout, and exercise frequency or intensity did not appear to vary widely between stables. There were no significant effects of age or gender on initial ulcer severity.

Objectives

The aim of the present study was to conduct an expanded epidemiological examination of thoroughbreds in Western Australia in order to identify specific risk factors associated with gastric squamous mucosal ulceration. The ultimate goal would be to identify management factors that could be manipulated in order to decrease the prevalence of disease.

Methodology

The study was approved by the Murdoch University Animal Ethics Committee (AEC R1151/05) and included informed owner consent.

Case selection

thoroughbred trainers from several regions in the southwest of Western Australia were solicited to take part in the proposed study. All available horses in each stable were subjected to gastric endoscopy irrespective of whether the trainer suspected ulcer disease or whether or not the horses were receiving anti-ulcer medication. There was a recommended drug withdrawal period of 4 days prior to racing after the sedation used in the procedure. This precluded collection of data from approximately 150 horses throughout the study.

Gastroscopy procedure

Endoscopy of the stomach was performed using a 3-meter flexible videoendscope (VES EV G300, Xion, Germany) in the free-standing, sedated horse. The procedure occurred in the training stable. The duration of fasting prior to endoscopy varied between 6 and 14 hours. Feed was not withheld in horses maintained full time at pasture. Water was not withdrawn prior to the procedure. Horses were sedated with a combination of xylazine (200 mg) and butorphanol (5mg) both given intravenously. The response to sedation was assessed subjectively as either under-sedated, adequately sedated, or over-sedated. Horses were additionally restrained with the aid of a nose twitch (Figure 1). The squamous mucosa was examined in its entirety in every horse. The procedure took on average 8 minutes per horse. Examinations were recorded on videotape; ulcers were scored at a later time.

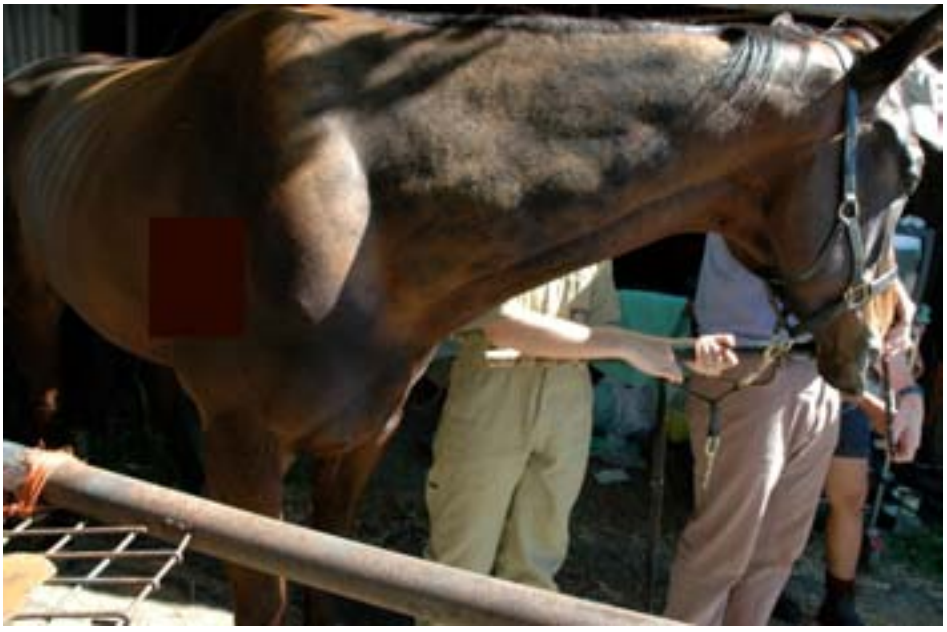

Figure 1 - Passage of the videoendoscope

Ulcer scoring

The gastric squamous mucosa was scored using a simplified system that ranged from 0 to 3.[12] Examples are included as figure 2. Lesions were graded as follows: Grade 0, normal - intact mucosal epithelium with or without reddening or hyperkeratosis; Grade 1, mild - single or multiple small ulcers; Grade 2, moderate - single or multiple large ulcers; and Grade 3, severe - extensive ulceration with coalescing of ulcerated areas. For the purposes of statistical analysis animals were grouped as follows: horses graded as 0 or 1 were classified as not having ulcers and those graded with an ulcer score of grade 2 or 3 were classified as having ulcers.

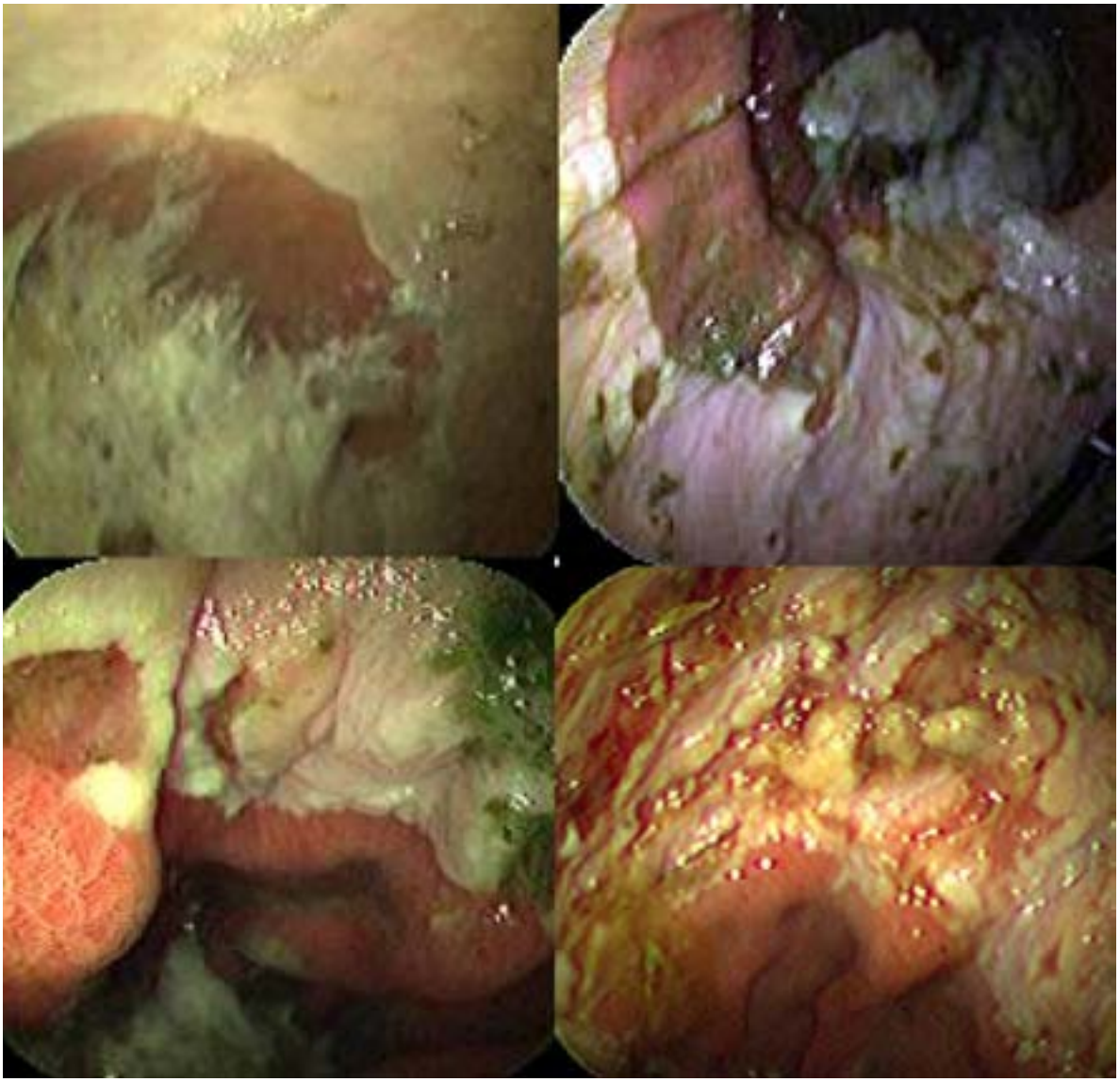

Figure 2 - Clockwise from top left: Grade 0 ; Grade 1; Grade 2; Grade 3

Survey

The following variables were collected via survey of trainers and extraction of information from the following websites: The Australian Studbook (www.studbook.org.au); Racing NSW registration database (www.racingnsw.com.au); and the racing database from the Western Australian Turf Club (http://cris.waturf.org.au/scripts/ws.exe/syturf/findinfo.hts?)

Horse Name
Age
Sire
Dam
Family Group
Gender (Male entire, female, gelding)
Colour
Trainer
Weeks in work
How long at current property (weeks)
Racing to expectation (Below, At, Exceeding)
Location (Metropolitan, Non-metropolitan)
Body condition score (Poor, Normal, Heavy)
Ownership
Stereotypic or abnormal behaviour (Y/N)
Weaving (Y/N)
Head Nodding (Y/N)
Stall Walking (Y/N)
Stall pawing (Y/N)
Self-mutilation (Y/N)
Tail rubbing (Y/N)
Stall kicking (Y/N)
Crib-biting (Y/N)
Wood chewing (Y/N)
Aggression towards horses (Y/N)
Aggression towards humans (Y/N)
Biting (habitual; snapping) (Y/N)
Kicking (Y/N)
Threatening behaviour (ear pinning, pointing rear end) (Y/N)
Mobile aggression (tendency to rush or charge aggressively) (Y/N)
Nervousness (Calm, Normal, Nervous)
Mobile Alarm (exaggerated alarm or panic)
Loads and travels (Good, Poor)
Handling barriers (Good , Poor)
Behaviour at racetrack (Calm, appropriately alert, overly anxious)
Response to sedation (under, normal, over)
Appetite (Excellent, Normal, Poor)
Ease at maintaining bodyweight (Very easy, Normal, Difficult)
Access to pasture (Y/N)
Turnout with other horses (Y/N)
Minutes of pasture turnout per day
Oaten Hay (Y/N)
Wheat Hay (Y/N)
Lucerne Hay (Y/N)
Grass Hay (Y/N)
Oaten Chaff (Y/N)
Wheaten Chaff (Y/N)
Lucerne Chaff (Y/N)
Bran (Y/N)
Number of feeds per day
Oats (Y/N)
Barley (Y/N)
Maize (Y/N)
Lupins (Y/N)
Pellets (Y/N)
Sweet Feed Mix (Y/N)
Salt/mineral block (Y/N)
Canola oil (Y/N)
Supplements
Scheme water (Y/N)
Ground water (Y/N)
Dam water (Y/N)
Rain water (Y/N)
Number of days per week exercise
Number of days per week fast work
Exercise at track on property (Y/N)
Exercise at racetrack (Y/N)
Exercise in bush (Y/N)
Exercise at beach (Y/N)
Exercise on walking machine (Y/N)
Blinkers during training /racing (Y/N)
Number of exercise riders
Swimming (Y/N)
Swimming location
Swimming times per week
Swimming duration (min)
Stall type
Bedding sand (Y/N)
Bedding rubber (Y/N)
Bedding shavings (Y/N)
Bedding straw (Y/N)
Direct contact with other horses (Y/N)
Barrier between horse solid (Y/N)
Barrier between horse mesh (Y/N)
Barrier between horse rails (Y/N)
Barrier between horses none (Y/N)
Stall toys (Y/N)
Balls in stall (Y/N)
Tyres in stall (Y/N)
Witch's Hats (Y/N)
Oil Cans (Y/N)
Radio played in barn (Y/N)
Talk/racing radio (Y/N)
Music radio (Y/N)
Hours of radio per day
Number of workers in stable
Number of years training
Property size
Number of horses in work current
Number of horses in work normal
Horse density (calculated)
Distance to airport
Distance to beach
Distance to major road
Distance to railway
Anti-ulcer medications current (Y/N)
Anti-ulcer medication last 14 days (Y/N)
Anti-ulcer drug
Anti-ulcer dosage
Anti-ulcer frequency of treatment
Anti-ulcer duration of treatment
Other medications current (Y/N)
Medications
Other medications last 14 days (Y/N)
Lameness (Y/N)
Colic (Y/N)
Other health problems (Y/N)
Racing campaigns
Career starts
Career wins
Career places
Career prize money
Starts this campaign
Wins this campaign
Places this campaign
Prize money this campaign
Trials this campaign

Statistical Analyses

Questionnaire data were imported from Excel into SPSS ver 14.0 and Egret for Windows (Cytel Software, Ver 2.0.31) for statistical analyses.

Descriptive statistics for variables were determined and association between categorical variables (such as gender, racing to performance, presence of stereotypic behaviours etc) and the presence of gastric ulceration (score 2 or 3) were analysed with a Chi-square test for independence or a Fisher's exact test and by calculation of odds ratios and 95% confidence intervals.

Associations between continuous variables such as age, weeks in training, distance from airport etc and the presence of gastric ulceration were analysed using a parametric ANOVA or a Kruskal-Wallis ANOVA. For the univariable analyses significance was set at 5%.

Logistic-normal multiple regression was then used to create a multivariable random effects model. Only variables significant at the $P \leq 0.25$ in the univariable analyses were considered eligible for inclusion in the logistic multiple regression analyses. Dummy variables were generated for any categorical variable with more than two levels. Backward elimination was used to determine which factors could be dropped from the multivariable model. The likelihood-ratio test statistic was calculated to determine the significance at each step of the model building. Because of the likely presence of additional variation due to the clustering of individual horses into stables, stables (trainer) were incorporated as a random effect in the model.

The level of significance for a factor to remain in the final model was set at 5%. Two-way interaction terms among the explanatory variables were examined after identification of the reduces set main effects. Each interaction was added to the model and the significance assessed in the same way as for the explanatory variables.

The correlation between factors significant in the univariable analyses but not included in the final model, and factors included in the model, were calculated using Spearman rank coefficients.

Results and Discussion

Data were collected from 402 thoroughbred horses over a 10 months in 2006. There were 37 trainers enrolled in the study, 27 of which had 8 or more horses examined. The trainers and horses were from the following regions in south Western Australia (Figure 3):

- Albany located 420 km south of Perth on the Southern Coast; 13 trainers with a total of 82 horses
- Mandurah/Larkhill situated 60 km south of Perth on the coastal plain; 6 trainers with 96 horses
- Swan Valley located 60 km north east of Perth; 2 trainers with 35 horses
- Bunbury located on the coast 170 km south of Perth; 8 trainers with 76 horses
- Ascot/Belmont situated within the metropolis of Perth; 8 trainers with 113 horses (Figures 4 & 5)

Figure 3 - Region location (image from Google Earth)

Figure 4 - Proximity of Ascot Racecourse to Perth CBD (Google Earth)

Figure 5 - Ascot Racecourse (A), Training Stables (B),
Swan River (C), Great Eastern Highway (D) (Google Earth)

For statistical purposes the trainers and horses were categorised as metropolitan (113 horses from Ascot/Belmont) and non-metropolitan (289 horses).

The overall prevalence of non-glandular ulceration was 53%. This included 20% of horses with grade 1 lesions, 22% of animals with grade 2 lesions, and 11% with grade 3 disease (Figure 6). For statistical purposes horses were categorised as having no or mild disease (grades 0 and 1 – overall 67%) and moderate or severe ulceration (grades 2 and 3 – overall 33%) (Figure 7). The prevalence of moderate and severe ulceration varied over the five locations as follows (see Figure 8):

- Albany 32.9% (82 horses)
- Mandurah/Larkhill 27.1% (96 horses)
- Swan Valley 22.9% (35 horses)
- Bunbury 11.8% (76 horses)
- Ascot/Belmont 55.8% (113 horses)

Results of the univariate analysis are presented in Table 1 and the multivariate analysis in Table 2.

The prevalence data were further categorized on the basis of location: non-metropolitan (24.2%) and metropolitan (55.8%) and trainer. Based on univariate analysis there was a significant effect of location ($p<0.001$). Horses trained in the city were 3.94 times more likely to have moderate or severe non-glandular ulceration than those trained in other regions. There was also a significant effect of trainer on the prevalence of non-glandular ulcer disease ($p<0.001$). This effect was preserved when the analysis was further restricted to those trainers with 8 or more animals surveyed. The between trainer variability is illustrated in Figure 9. Horses from three trainers (numbers 12, 29 and 30) had no evidence of gastric ulceration; in contrast horses from 8 trainers (15,16, 22, 25, 28, 32, 34 and 35) had more than 50% of horses with scores 2 or 3.

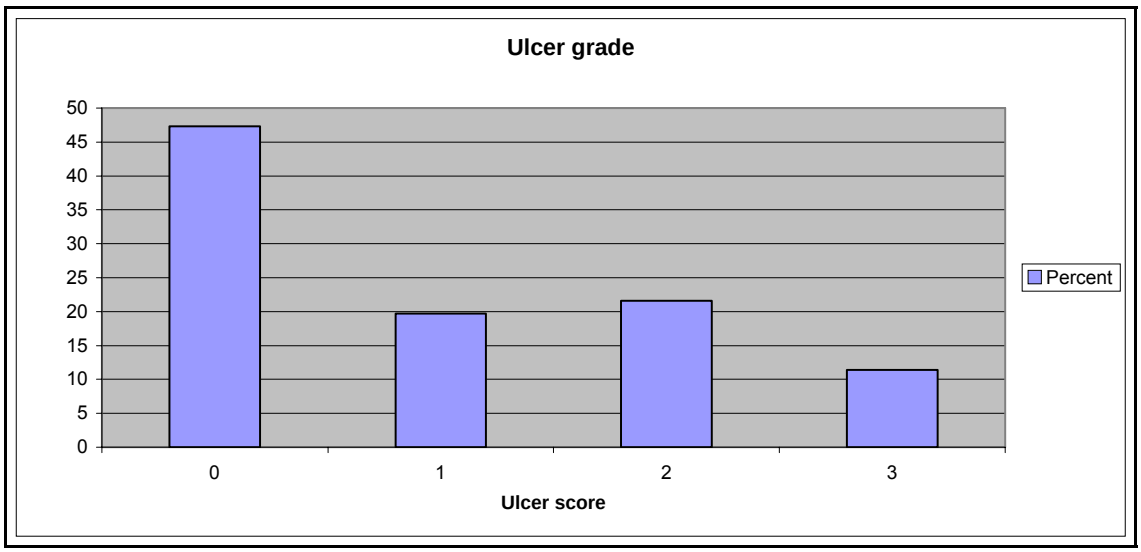

Figure 6 – Distribution of cases based on ulcer score

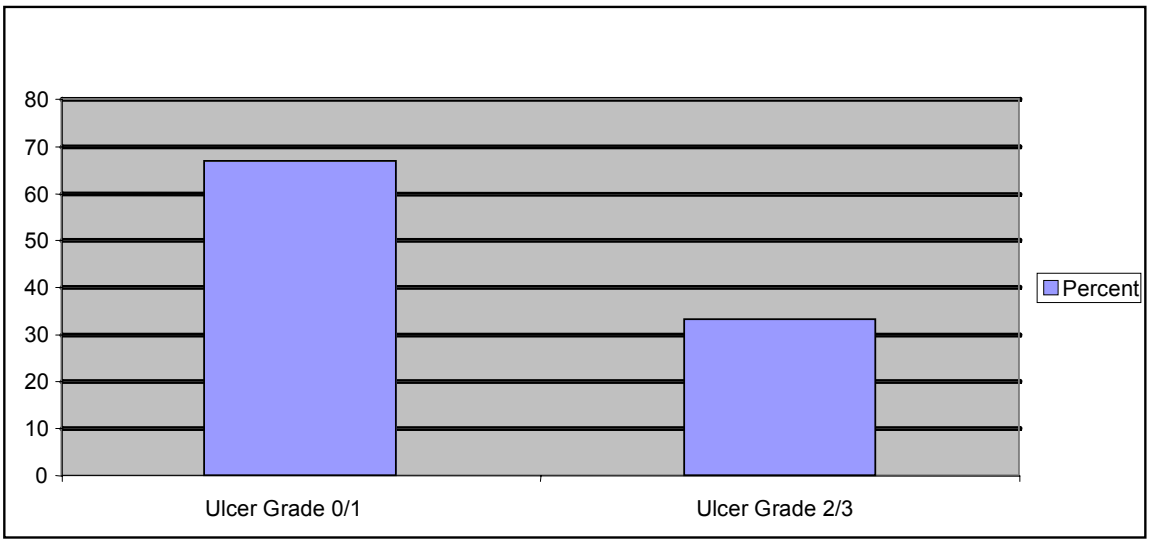

Figure 7 – Overall ulcer prevalence

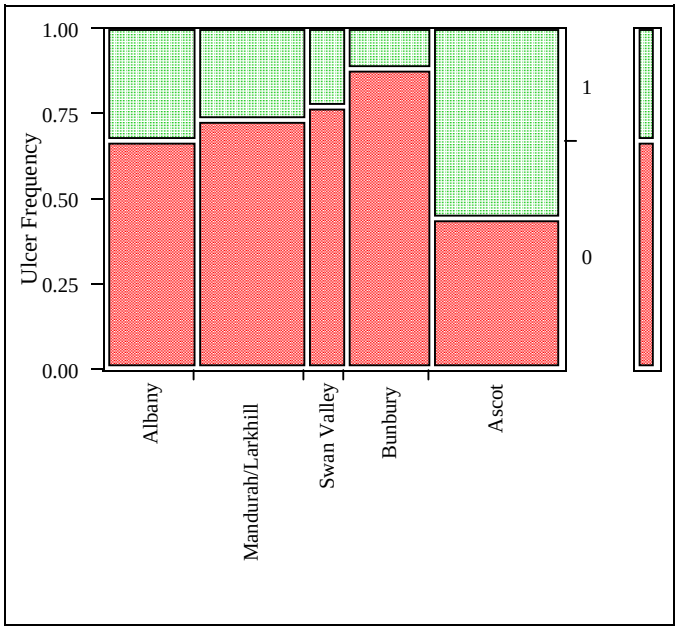

Figure 8 - Mosaic plot of ulcer frequency for each of the five regions studied. 0= no ulcers (grade 0/1); 1=ulcers (grade 2/3) (the width of the columns represents the relative number of horses examined from that location)

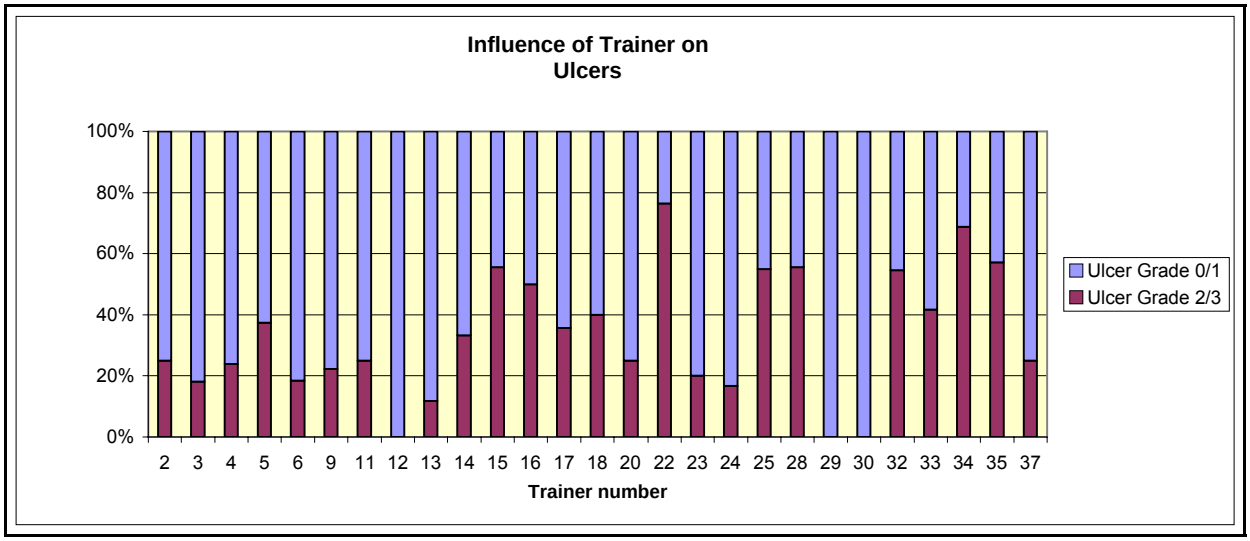

Figure 9 – Prevalence of moderate or severe ulceration according to trainer (trainers with more than 8 horses).

Gender and Age

There were more geldings (250; 62.2%) than females (135; 33.6%) enrolled in the study. There were only 17 entire male horses (4.2%) and these were combined with geldings for statistical purposes. There was no effect of gender on the prevalence of non-glandular ulceration (p=0.95). The mean age of animals enrolled in the study was 4.34 ± 0.08 years. There was no effect of age on ulceration (p=0.92).

Training

The mean number of weeks in training at the time of examination was 12.3 ± 0.33 weeks, with a range of <1 to 32 weeks. The time that an animal had been in training was significantly associated with the prevalence of ulcers (p<0.001). This variable retained significance in both the univariable and multivariable models. The odds of a horse developing moderate or severe ulceration increased by 1.69 times for every week that the horse was in training. The increase in prevalence over time is illustrated in figure 9. In contrast to the length of time in training there was no effect of total time at the property on ulcer prevalence (p = 0.56). The number of days of exercise per week was not associated with disease (p=0.28), but the number of days of fast work was correlated (p=0.007). Horses that had a greater number of fast days of work per week were at increased risk of disease. As expected this variable was significantly correlated with the number of weeks in work (Pearson correlation coefficient (PCC) = 0.332), the latter of which was retained in the final model. The number of exercise riders was not associated with ulcer disease (p=0.66). The number of years that the trainer had been training and racing horses was also not associated with an increased risk of ulceration (21.5 years for trainers of horses without gastric ulcers and 23.3 years for trainers of horses with gastric ulcers, p = 0.15).

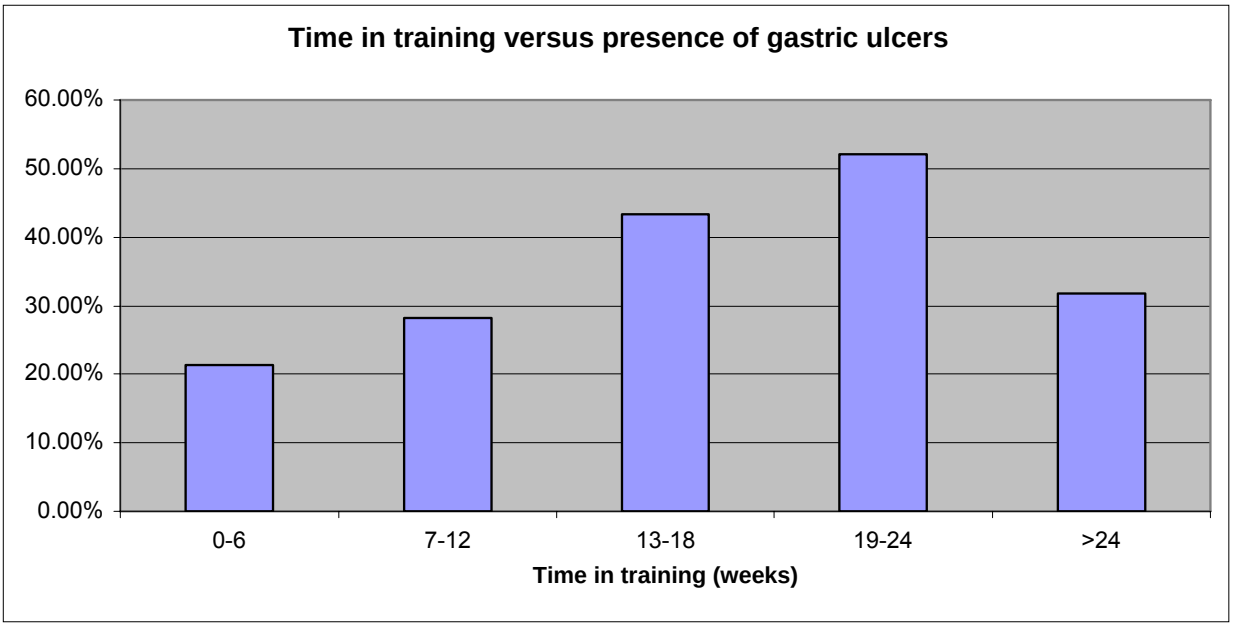

Figure 10 - Change in ulcer prevalence (ulcer score 2 or 3) over time in training

The site of training varied across the study population and many horses were exercised at more than one site. Exercise at a track on the property where the animal resides was strongly associated with a reduced prevalence of ulcer disease (protective factor). This was significant in the univariable and multivariable models ($p<0.001$). Animals trained at a track on the home property were 3.27 times less likely to have squamous ulcer disease than those that were not. Exercise on a bush track was also linked to reduced ulceration ($p<0.001$) but this factor was not included in the final multivariable model. Exercise at a racetrack was positively correlated with the development of moderate or severe ulceration ($p=0.001$), but again this variable was not retained in the final model. Most animals stabled in the city were exclusively trained at the local racetrack. Horses that were exercised at a beach or on a walking machine were not at any greater risk of disease, although numbers were small in these categories.

Blinkers were only used on a small number of horses in the study (34; 8%) and their use was not associated with ulcer disease ($p=0.30$). Approximately 25% of the horses studied were swum as part of their training. The majority of these animals were swum in purpose-constructed pools with very small numbers exercised in rivers and dams. On a univariate basis swimming was significantly associated with ulcer disease ($p=0.007$) and these animals were 1.8 times more likely to have ulcers than those that did not swim. Of further interest were significant effects of swimming frequency ($p=0.004$) and duration (0.042) with increased risk of disease associated with more frequent and/or longer swimming events. None of these variables were retained in the final model.

Performance

Horses were categorised based on the trainer's assessment of performance. For statistical assessment animals that were performing below their expected level were compared with those performing at or above their expected level. There were 100 horses in the study that were performing unsatisfactorily according to the trainer. On the basis of univariate analysis this variable was significantly associated with moderate or severe non-glandular ulceration ($p=0.015$) with under-performing horses 1.78 times more likely to have ulcer disease than those performing at or above expected level. This variable was not retained in the final multivariable model. Performing to trainer expectations was however significantly correlated with several of the factors retained in the final model including the number of weeks that the horse had been in training (PCC = 0.332) and difficulty in maintaining body condition (PCC = 0.206).

None of the following racing variables were significantly associated with moderate or severe gastric ulceration: number of campaigns, career starts, total career prize money, career wins, career places, current rating, number of wins in current campaign, wins or places in the current campaign, or prize money earned in the current campaign. The number of starts in the current campaign was a significant factor (p=0.03), but again this was significantly correlated with the number of weeks in work and was subsequently dropped from the final model.

Body Condition and Dietary Factors

Trainer interpretation of appetite and the ease (or difficulty) in maintaining adequate body condition, along with body condition score were all significantly associated with moderate or severe non-glandular ulceration on univariate analysis. Approximately 10% of horses surveyed had a poor appetite; many were slow eaters or failed to complete a concentrate meal. These horses were 2.92 times more likely to have disease than those with a normal appetite. Trainers reported that it was difficult to maintain body condition on 17% of horses. This was highly significant in the univariable and multivariable models. These horses were 3.42 times more likely to have ulcer disease than those that easily maintained adequate body condition. These two factors were highly correlated with a Pearson coefficient of 0.445. It was therefore not surprising that one of the two variables was removed from the final model. There was also a significant correlation within the final model between difficulty in maintaining body condition and the number of weeks in work; this again would be an expected finding. Although not maintained in the final model the body condition score was significantly correlated with appetite and ease of maintaining body weight.

Most trainers fed 2 or 3 concentrate meals daily, with a small number feeding 4 times daily. Meal frequency was not associated with disease (p=0.48). Univariate analysis identified several dietary ingredients as being positively associated with ulcer disease. These included the feeding of oaten chaff (p=0.03), lucerne chaff (p=0.003), or oats (p=0.01). Feeding maize (corn) (p=0.001) or wheat hay (0.03) was negatively associated with the development of moderate or severe gastric ulceration. None of these factors were accepted into the final model. Several of these dietary variables were positively or negatively correlated with being exercised at a track on the property or pasture turnout, from which we suggest that diet may in part be determined by location.

Diet is commonly quoted as a risk factor for non-glandular ulcer disease in horses. The feeding of high-energy low-roughage diets is commonplace in most thoroughbred training establishments. The most common carbohydrate source in the present study was oats, which was fed to around 90% of horses surveyed. This was commonly supplemented with smaller amounts of maize (corn) and/or barley. Commercial pellets (57%) and sweet feed mixes (47%) were also commonly used. Chaff was a common fibre source with most horses receiving either oaten (50%) or wheaten chaff (45%). Many received smaller quantities of lucerne chaff. Oaten and lucerne hay were also used although rarely exceeded 2 flakes/day. Just over half of the horses in the study had some access to pasture, although the time and pasture quality varied.

The univariate analysis identified several dietary components of interest, including the feeding of wheaten hay, oaten chaff, lucerne chaff, oats and maize. None of these factors were retained in the final multivariate model. These results would indicate that dietary factors are not important in the genesis of squamous mucosal ulceration, however it should be emphasized that diet is relatively consistent across most training establishments.

One of the most consistent signs ascribed to gastric ulcer disease is alteration to appetite. In the present study there were significant correlations between body condition score, appetite, and ease of maintaining bodyweight. The latter variable was included in the overall model. It would seem plausible that ulceration leads to a reduced appetite, which in turn makes maintenance of body condition difficult in the face of growing energy demands associated with increases in the level of

exercise. This would lead to the poor body condition in affected horses. These effects are closely linked to the number of weeks in training.

Aggression

There were several variables collected describing aggression. These related to aggression not only directed towards humans, but also towards other horses and other types of animals. Demonstrations of threatening behaviour (ear pinning or pointing rear end) or charging (mobile aggression) were also recorded. None of these factors were significantly associated with ulcer disease; in fact horses that were aggressive towards humans were less likely to have squamous mucosal ulceration (26.1% versus 34.0%). Many aggressive characteristics (mobile aggression, biting, kicking, and demonstration of threatening behaviour) were significantly correlated with demonstration of stereotypical or abnormal behaviour (see below). For example, horses that were biters were 29.9 times more likely to have a stereotypic behavioural trait than a non-biter.

Nervousness and Anxiety

There were several questions pertaining to nervousness and anxiety within individual horses. These included an overall trainer assessment of nervousness, aversion (readily shying at strange objects), mobile alarm (exaggerated alarm or panic attacks), problems in loading and travelling, problems at barriers, and over-anxiety at racetracks. The only variable that was significantly associated with squamous mucosal ulceration was aversion ($p=0.006$). Several trainers reported that aversion at strange objects was at varying degrees common to all horses. This factor was not retained in the final model. The response to the standard dose of sedative was not correlated with gastric ulceration.

Environment

A significant effect of location (horses from metropolitan locations had a higher prevalence than those from non-metropolitan stables) was described above. Other highly significant environmental factors associated with ulcer disease prevalence were access to pasture ($p<0.001$), turning out with other horses ($p=0.001$), and duration of pasture turnout ($p=0.001$). Each of these were negatively correlated with disease; horses with access to pasture were 3.11 times less likely to have ulcer disease, horses turned out with other horses were 4.20 times less likely, and horses without ulcers had a mean of around 4 hours turn out per day compared with approximately one hour for those with disease.

Direct contact appeared to be protective against ulceration ($p<0.001$). Horses that had direct (touching) contact with others were 2.5 times less likely to have moderate or severe ulcer disease. This variable was not included in the final model; this was most likely due to the logical and expected high correlation with the variable “turnout with other horses”. There was also a strong negative correlation between playing of a radio and direct contact with other horses. There appears to be limited contact permitted between horses in stables where a radio is played; this was a feature of several metropolitan and regional stables. Steel mesh or solid metal, timber or brick barriers separating animals were more commonly associated with ulceration than rails only. This again may reflect an ability, or inability, of horses to have touching contact with other animals. Mesh barriers had a significant association with non-glandular ulceration based on univariate analysis ($p<0.001$), but this factor was not retained in the final model. The variable was significantly correlated with several variables included in the overall model, which may explain why it was removed. Mesh barriers were more commonly used in barns without pasture access and where the radio was played. This housing type was more common in metropolitan areas.

Stall toys are often used to relieve boredom in stalled horses. On univariate analysis there was no overall significant effect on ulceration of presence of toys in the environment, but witch’s hats were positively associated with disease ($p=0.008$) and oilcans were negatively associated ($p=0.002$). Witch’s hats were negatively correlated with exercise at a track on the property, indicating that they were likely more commonly used on smaller properties. Oilcans were used by a small number of trainers and in the final model, where trainer was used as a random effect, the effect was lost.

The number of workers in the stable or property was not an important factor. Property size ($p<0.001$) and stocking density (horses/hectare) ($p<0.001$) were both significantly associated with ulcer disease however both were removed from the final multivariate model. Horses with ulcers were more likely ($p < 0.001$) to have come from a smaller property (mean property size of 5.77 hectares) and from a property with a higher stocking density (mean 46.2 horses/hectare) than horses without severe ulcers (mean 15.52 hectares and 20.1 horses/hectare respectively).

As described above, playing of a radio in the barn was strongly associated with gastric ulceration ($p<0.001$). This was also true for playing of talk radio ($p<0.001$) and music ($p<0.001$). Horses that were exposed to talk radio were 3.6 times more likely to have ulcers than those that did not. Music increased the odds of ulceration by 2.8 times. The length of time that the radio was played was also significant ($p<0.001$) with horses with ulceration having a mean time of 4.1 hours of radio playing compared with 2.1 hours in horses without ulceration. The effect of radio was strong enough to remain in the final model. Playing a radio increased the odds of moderate or severe ulcer disease by 2.89 times. There were several significant variables after univariate analysis that were excluded from the final model and were strongly correlated with playing of a radio. Most significant were location in a metropolitan stable, swimming as part of the training routine, having mesh dividers between yards, not having access to pasture, drinking scheme and not ground water, not having direct contact with other horses, and not exercising in a bush environment. Most of these variables were more commonly found in urban training environments.

Health and Medications

None of the variables recorded were significantly associated with ulceration. Currently receiving an anti-ulcer medication or other medication were not significantly associated with disease. Gastrophilus *spp*. (Bot fly) larvae were not associated with an increased risk of ulceration.

Influence of sire and maternal family

There were 163 different sires with progeny in the study. This precluded meaningful statistical analysis however a plot of ulcer prevalence in the offspring of sires with more than 8 progeny demonstrated wide variability (Figure 11). There was a similar problem with investigation of the maternal family line although there was less variability in ulcer prevalence when compared to the sire line (Figure 12).

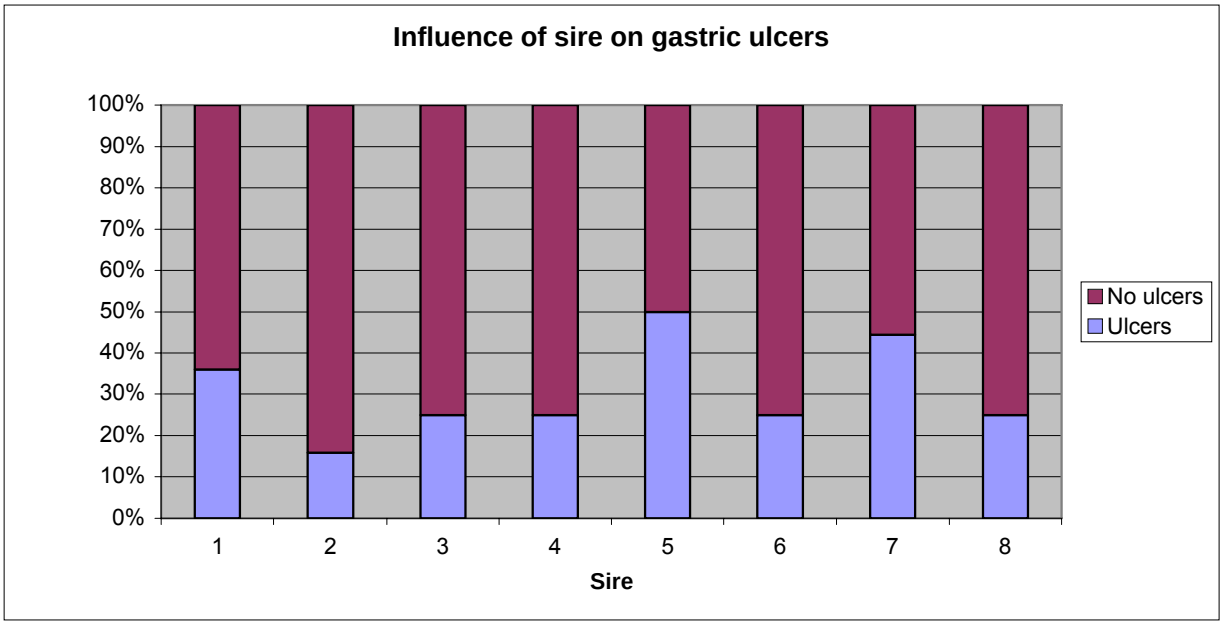

Figure 11 - Influence of sire on ulcer prevalence (sires with more than 8 progeny)

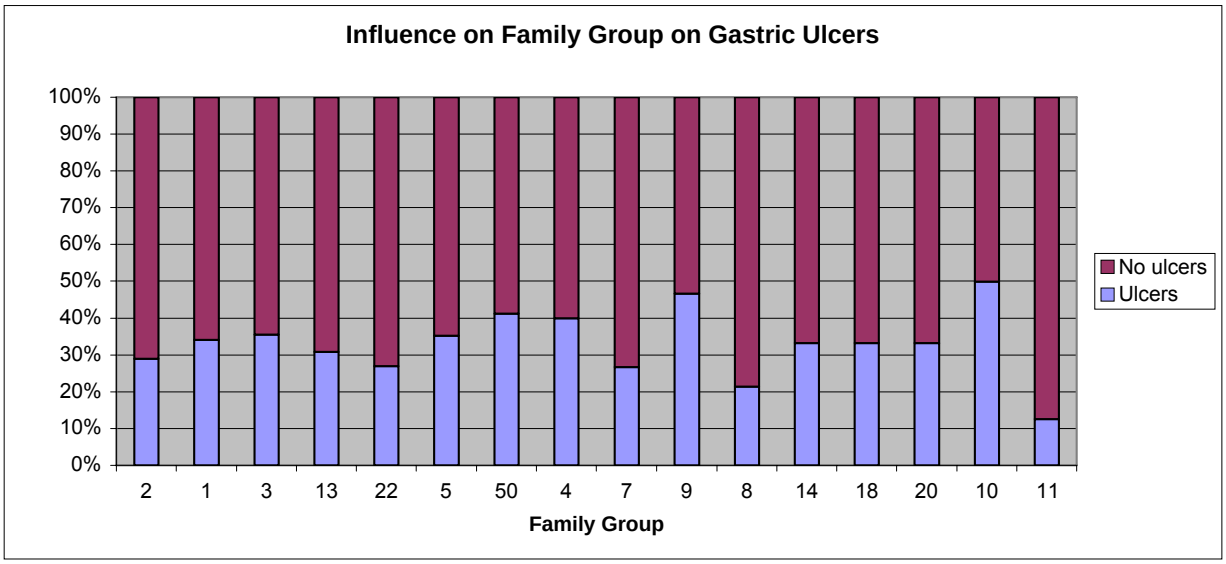

Figure 12 - Influence of maternal family on ulcer prevalence (n>8)

Stereotypic or Abnormal Behaviour

Multiple factors in this category were positively associated with ulcer disease. These included head nodding, crib-biting, and stall kicking. Several other behavioural traits had p-values between 0.05 and 0.25, including weaving, stall walking, and stall pawing. Demonstrating any stereotypic or abnormal behavioural trait was also significantly related to ulceration (p=0.047). Crib-biting was the most significant trait and was retained in the final model. These animals were 7.58 times more likely to have moderate or severe ulcer disease than those that did not crib bite. Animals with abnormal behaviour were more likely to demonstrate aggression towards people.

Stereotypies are defined as repetitive behavioural traits that become fixed in both form and orientation.[33] Examples include weaving, head nodding, crib biting, wood chewing and stall walking. Stereotypic or abnormal behaviour was reported in approximately 25% of the horses in the present study. This value may underestimate the true prevalence, as several trainers appeared to accept some behavioural traits as normal. A study of 225 young horses in the United Kingdom reported that 34.7% of the animals developed abnormal behaviour, many of which were true stereotypies.[34] It could be argued that several of the traits measured in the present study were not true stereotypies, but rather altered behaviour in response to anxiety or specific diseases. Given the similar physical characteristics between crib-biting and wind-sucking it is plausible that trainers may have incorrectly differentiated the conditions. Some investigators suggest that the two vices are one and the same.[35] Consequently data for both behavioural traits were pooled and described as crib-biting. Crib-biting involves grasping a horizontal object with the upper incisors, contracting the ventral neck muscles leading to arching of the neck and caudal retraction of the larynx. Air is drawn into the cranial oesophagus in response to the creation of a negative pressure gradient between the oesophagus and the pharynx. Air is not swallowed as was once thought.[36] Wind-sucking describes an identical physical event to crib-biting, but without the need to grasp an object with incisors. Crib-biting was the most common stereotypic trait in the present study and was noted in over 8% of horses. Previous studies have reported that crib-biting occurs in approximately 5% of captive domesticated horses.[17] It is widely acknowledged that this oral stereotypy occurs in response to boredom, but recent data also indicate a link between the feeding of high grain and low roughage diets and the onset of cribbing.[34]

Crib biting was strongly associated with gastric ulceration. This was apparent in both the univariate and multivariate analyses. Horses that demonstrated this behaviour were more than 7.5 times more likely to have moderate or severe non-glandular ulceration than those that did not. This association is

not new; a study of crib-biting and non crib-biting juvenile horses also concluded that the behavioural trait was associated with gastric ulceration. [17] What remains unclear is whether the crib-biting causes gastric ulceration, occurs in response to the ulceration, or if crib-biting and gastric ulceration are occurring independently in response to other common factors. There is no physiological basis to suggest that crib-biting causes non-glandular ulceration of the stomach. Some have hypothesised that crib-biting may provide relief in horses with ulcers by inducing saliva flow which could theoretically provide buffering capacity to protect from acid injury.[17] The impact of crib-biting on saliva production has not been investigated. Univariate analysis identified a significant impact not only of crib-biting, but also head nodding and stall kicking on the presence of moderate and severe non-glandular ulceration. Furthermore weaving, stall walking, stall pawing, and wood chewing all returned p-values between 0.05 and 0.25 and were therefore entered into the multivariate analysis. It is tempting to conclude that other common factors, such as boredom or diet, could be the primary initiator of both stereotypic behaviour and non-glandular gastric ulceration. We were unable to document significant correlation between crib-biting and any of the variables included in the final multivariable model.

The most common stereotypies after crib-biting were stall walking (7%), wood chewing (6%), stall pawing (5%), stall kicking (4%), head nodding (3%), and weaving (3%). There were no animals that exhibited self-mutilation, and only a single horse that appeared to compulsively rub its tail and perineum.

Final model

Factors included in the final model were number of weeks in work, whether the horse was a crib-biter, whether the trainer reported difficulty in maintaining the bodyweight of the horse, if a radio was played in the barn, whether or not the horse was turned out with other horses, and whether or not the horses were exercised at a track on the property.

Table 1 – Results of the univariate analysis

Variable		N	Moderate or Severe (%)	P-value	Odds Ratio (95% CI)
Gender	Male	152	32.9	0.95	1.0
	Female	200	33.2		1.01 (0.66-1.56)
AGGRESSION					
Aggression to horses	Yes	58	36.2	0.590	1.18 (0.66-2.1)
	No	344	32.6		1.0
Aggression to humans	Yes	46	26.1	0.280	1.0
	No	356	34.0		1.46 (0.73-2.92)
Biting	Yes	42	33.3	0.970	1.01 (0.51-2.0)
	No	360	33.1		1.0
Kicking	Yes	35	34.3	0.870	1.06 (0.51-2.20)
	No	367	33.0		1.0
Threatening behaviour	Yes	52	34.6	0.080	1.08 (0.58-2.0)
	No	350	32.9		1.0
Aggression to other animals	Yes	22	31.8	0.900	1.0
	No	380	33.2		1.06 (0.42-2.67)
Mobile aggression	Yes	56	30.4	0.640	1.0
	No	346	33.5		1.16 (0.63-2.13)
NERVOUSNESS					
Nervousness	Yes	69	39.1	0.240	1.38 (0.81-2.35)
	No	333	31.8		1.0
Aversion	Yes	46	15.2	0.006	1.0
	No	356	35.4		3.05 (1.33-7.04)
Mobile alarm	Yes	39	41.0	0.270	1.46 (0.75-2.87)
	No	363	32.2		1.0
Problems loading and travelling	Yes	90	34.3	0.870	1.0
	No	312	33.2		1.09 (0.37-3.21)
Problems with barriers	Yes	11	18.2	0.340	1.0
	No	369	35.0		2.42 (0.51-11.36)
Anxiety at track	Yes	58	37.9	0.400	1.28 (0.72-2.28)
	No	344	32.3		1.0
STEREOTYPIC BEHAVIOUR					
Variable	**Response**	**Number**	**Moderate or Severe Ulceration (%)**	**P-value**	**Odds Ratio (95% CI)**
Weaving	Yes	11	54.5	0.13	2.49 (0.75-8.33)
	No	391	32.5		1.0
Head Nodding	Yes	14	64.3	0.012	3.83 (1.26-11.67)
	No	388	32.0		1.0
Wind Sucking	Yes	33	73.3	0.000	6.47 (2.79-14.97)
	No	369	29.8		1.0
Stall Kicking	Yes	16	56.3	0.040	2.72 (1.0, 7.46)
	No	386	32.1		1.0
Self mutilation	Yes	0	-		
	No	402	33.1		
Stall Walking	Yes	29	44.8	0.160	1.71 (0.8, 3.68)
	No	373	32.2		1.0

Stall pawing	Yes	19	47.4	0.180	1.88 (0.75, 4.74)
	No	383	32.4		1.0
Tail rubbing	Yes	1	100	0.33	
	No	401	32.9		
Wood chewing	Yes	24	45.8	0.17	1.78 (0.77-4.08)
	No	378	32.3		1.0
Any stereotypic or abnormal behaviour	Yes	101	39.2	0.047	1.50 (1.00-2.40)
	No	301	29.5		1.0
DIET AND APPETITE					
Variable	**Response**	**Number**	**Moderate or Severe Ulceration (%)**	**P-value**	**Odds Ratio (95% CI)**
Poor appetite	Yes	41	56.1	0.001	2.92 (1.52-5.62)
	No	361	30.5		1.0
Body Condition	Thin	46	53.8	0.020	2.52 (1.13-5.61)
	Adequate or Heavy	356	31.6		1.0
Difficulty in maintaining condition	Yes	67	53.7	0.000	2.85 (1.67-4.87)
	No	335	29.0		1.0
Oaten Hay	Yes	287	34.1	0.480	1.19 (0.74-1.89)
	No	115	30.4		1.0
Wheat Hay	Yes	20	10.0	0.030	1.0
	No	382	34.3		4.69 (1.07-20.41)
Lucerne Hay	Yes	161	37.3	0.150	1.37 (0.90,-2.08)
	No	241	30.3		1.0
Grass Hay	Yes	21	23.8	0.350	1.0
	No	381	33.6		1.62 (0.58-4.52)
Oaten Chaff	Yes	202	38.1	0.030	1.58 (1.04-2.41)
	No	200	28.0		1.0
Wheaten Chaff	Yes	183	30.6	0.330	1.0
	No	219	35.2		1.23 (0.81-1.87)
Lucerne Chaff	Yes	345	35.9	0.003	3.0 (1.42-6.3)
	No	57	15.8		1.0
Bran	Yes	123	30.9	0.540	1.0
	No	260	34.1		1.15 (0.73-1.82)
Oats	Yes	366	35.0	0.010	3.3 (1.3-8.8)
	No	36	13.9		1.0
Barley	Yes	113	34.5	0.700	1.09 (0.69-1.73)
	No	289	32.5		1.0
Maize/Corn	Yes	234	26.5	0.001	1.0
	No	168	42.3		2.03 (1.33-3.10)
Lupins	Yes	51	33.3	0.970	1.01 (0.54-1.89)
	No	351	33.0		1.0
Commercial pellets	Yes	229	33.2	0.960	1.01 (0.66-1.54)
	No	173	32.9		1.0
Commercial sweet feed	Yes	188	33.5	0.870	1.04 (0.68-1.57)
	No	214	32.7		1.0
Mineral block	Yes	59	33.9	0.890	1.04 (0.58-1.87)
	No	343	32.9		1.0
Canola oil	Yes	73	41.1	0.110	1.53 (0.91-2.58)
	No	329	31.3		1.0
Scheme water	Yes	183	50.8	0.000	1.0
	No	217	18.4		4.57 (2.92-7.17)
Ground water	Yes	189	18.5	0.000	1.0
	No	213	46.0		3.75 (2.38-5.92)
Rain water	Yes	28	17.9	0.080	1.0
	No	374	34.2		2.39 (0.89-6.45)

EXERCISE AND PERFORMANCE					
Exercise at track on property	Yes	155	20.6	0.000	1.0
	No	247	40.9		2.66 (1.67-4.24)
Exercise at racetrack	Yes	288	38.2	0.001	2.45 (1.46-4.09)
	No	114	20.2		1.0
Exercise at beach	Yes	49	32.7	0.950	1.0
	No	353	33.1		1.02 (0.54-1.93)
Exercise in bush	Yes	184	22.8	0.000	1.0
	No	218	41.7		2.42 (1.56-3.75)
Walking Machine	Yes	84	38.6	0.410	1.31 (0.69-2.51)
	No	318	32.4		1.0
Swimming	Yes	104	44.0	0.007	1.88 (1.18-3.0)
	No	298	29.5		1.0
Blinkers	Yes	34	41.2	0.300	1.47 (0.72-3.0)
	No	368	32.3		1.0
Racing at Expected Level	Below Expectation	100	43.0	0.015	1.78 (1.12-2.83)
	At or Above Expectation	302	29.8		1.0
ENVIRONMENT					
Location	Metropolitan	113	55.8	0.000	3.94 (2.49-6.24)
	Non-metropolitan	289	24.2		1.0
Access to Pasture	Yes	210	21.4	0.000	1.0
	No	192	45.8		3.11 (2.01-4.78)
Turned out with other horses	Yes	85	12.3	0.001	1.0
	No	317	37.1		4.20 (1.94-9.09)
Direct contact with other horses	Yes	130	20.0	0.000	2.50 (1.50-2.46)
	No	272	39.3		
Stall Toys	Yes	190	33.2	0.980	1.01 (0.66-1.53)
	No	212	33.0		1.0
Stall Balls	Yes	55	30.9	0.710	1.0
	No	347	33.4		1.12 (0.61-2.07)
Witch's Hat	Yes	95	44.2	0.008	1.88 (1.17-3.02)
	No	307	29.6		1.0
Toy Cans	Yes	51	13.7	0.002	1.0
	No	351	35.9		3.52 (1.54-8.06)
Radio	Yes	204	45.6	0.000	3.31 (2.13-5.15)
	No	198	20.2		1.0
Talk radio	Yes	141	51.8	0.000	3.60 (2.31-5.60)
	No	261	23.0		1.0
Music	Yes	149	47.7	0.000	2.80 (1.82-4.30)
	No	253	24.5		1.0
HEALTH AND MEDICATIONS					
Anti-ulcer medications current	Yes	34	44.1	0.150	1.67 (0.82-3.41)
	No	368	32.1		1.0
Anti-ulcer medications last 14 days	Yes	35	40.0	0.360	1.39 (0.68-2.83)
	No	367	32.4		1.0
Gastrophilus larvae	Yes	54	29.6	0.560	1.0
	No	348	33.6		1.2 (0.64-2.25)
Medications – not anti-ulcer	Yes	34	47.1	0.070	1.91 (0.94-3.87)
	No	368	31.8		1.0
Colic	Yes	14	28.6	0.720	1.0

	No	388	33.2		1.25 (0.38-4.05)
Lameness	Yes	104	36.5	0.390	1.23 (0.77-1.96)
	No	298	31.9		1.0

Continuous Data

	Mean in horses with ulcers	Mean in horses without ulcers	P value
Age	4.33	4.35	0.92
Weeks in work	13.95	11.55	0.001
Pasture minutes per day	74.14	236.4	0.001
Number of meals	2.2	2.16	0.48
EXERCISE AND PERFORMANCE			
Number of work days per week	6.08	6.03	0.28
Number of fastwork days per week	2.05	1.82	0.007
Number of riders	2.11	2.15	0.66
Number of stable workers	4.88	4.64	0.32
Number of trainer years	21.47	23.34	0.15
Property size	5.77	15.531	0.000
Horses per hectare	46.22	20.069	0.000
Career starts	12.62	10.92	0.35
Career money	45061	46120	0.92
Career places	3.72	3.95	0.70
Career wins	2.29	2.51	0.63
Length of time at property	16.25	14.75	0.56
Money	7346.8	5778.28	0.48
Places	0.69	0.87	0.27
Radio hours per day	4.11	2.13	0.00
Rating	64.7	66.4	0.30
Starts	2.3	1.54	0.03
Wins	0.43	0.47	0.82
Trials	0.78	0.48	0.002
Number of times swimming per week	1.31	0.76	0.004
Swimming duration per session (minutes)	3.3	2.4	0.042

Table 2 – Results of the multivariate analysis

	Coefficient	Std.Error	p-value	Odds Ratio	Lower (C.I.)	Upper (C.I.)
%GM (constant)	-1.943	0.3667	<0.001	0.1433	0.06981	0.294
Weeks in work	0.06569	0.01873	<0.001	1.068	1.029	1.108
Wind sucking	2.025	0.4978	<0.001	7.576	2.856	20.1
Difficult to maintain body condition	1.23	0.3209	<0.001	3.421	1.824	6.417
Radio in the barn	1.025	0.276	<0.001	2.787	1.622	4.787
Turnout_with other horses	-1.208	0.4798	0.01183	0.2989	0.1167	0.7654
Exercise track on property	-1.186	0.2702	<0.001	0.3054	0.1798	0.5186

Deviance with 395 df = 405.6
Likelihood Ratio Statistic at 7 df = 151.7 p-value < 0.0010

Conclusions

The overall prevalence of gastric squamous mucosal ulceration in the present study was lower than that reported in most other studies on racing thoroughbreds. This likely reflects the methodology and large variance across stables and regions. The decision to categorise animals with mild disease as normal is controversial but not without precedent.[10] The association between ulcer severity and the presence of clinical signs is poorly defined and in our clinical practice treatment is usually reserved for animals with moderate or severe disease. The prevalence rates quoted in this study must be viewed in light of this classification.

There were significant effects of both trainer and location on ulcer prevalence. This confirmed our pre-study suspicion that the prevalence of ulceration would vary considerably within regions of Western Australia. Only 11.8% of thoroughbreds examined in the Bunbury region had moderate or severe non-glandular ulceration compared with 55.8% of horses housed around the metropolitan racetrack. In contrast to some other studies we were unable to document an effect of gender or age on ulceration.

The formation of gastric squamous ulcers is clearly multi-factorial and likely includes elements of physiological and psychological stress. The fact that several stables were essentially free of disease may be interpreted to suggest that no single factor is critical for ulcer formation. Consistent with the findings of other studies the frequency and severity of ulcer disease was strongly associated with the length of time in training. In contrast disease was not correlated with the length of time at the training facility indicating the possible importance of exercise and diet.

We were however unable to identify any specific dietary factors that were positively or negatively associated with ulcer disease. Access to pasture is commonly cited as a protective factor in gastric ulcer disease; it is often assumed that the consumption of pasture may provide a nutritive protective effect. Any benefit from turnout may be due to relief from boredom rather than any direct benefit from the consumption of pasture. Direct contact with other horses appears to be a critical factor in protecting against ulcer disease; this is reflected by a significant effect of turnout with other horses in the final model. Furthermore solid barriers, as opposed to rails, were associated with an increased risk of ulcer disease. A radio in the barn could also be considered as a form of psychological stress to horses. This factor was retained in the final model. On a univariate basis talk radio, typically racing radio, created a greater risk of disease than music. Stereotypic behaviour was significantly associated with ulceration; this may also indirectly reflect boredom or psychological stress within the animal's environment. It would seem unlikely that the behavioural trait itself would have a direct link to ulcer disease.

Interpretation of the final analyses should be done with some caution. Some key variables were clearly removed from the final model because they were highly correlated with other factors that remained. For example, appetite was not included in the final model despite being highly significant on a one-way analysis. Appetite was however highly correlated with difficulty in maintaining body condition and the number of weeks in work, both of which were included in the final model. A poor appetite in response to gastric ulceration may actually be a critical response that leads to problems in maintaining body condition and reduced athletic performance. These problems are obviously accentuated with the length of time in work.

With most epidemiological studies it is hoped to uncover variables of interest that can be managed to reduce disease prevalence or severity. In this study we have identified several signs that confer a high risk of disease, e.g., difficulty in maintaining body condition and any display of stereotypic behaviour, particularly crib biting or wind sucking. It would also be appropriate where possible to address aspects of the horse's environment that may promote stress. Horses are herd animals and direct contact with others would seem reasonable, ideally under turnout conditions. Playing of the radio

could be considered as a form of stress for some animals, but again care in interpretation is needed as playing of the radio occurred more commonly in metropolitan stables than rural environments. Training at off-site racetracks appears to increase of ulceration, or more importantly training on site either on a track or in the bush is protective against disease. It is also important to consider that merely being located in the metropolitan region is not a primary risk factor, but aspects common to barns in this environment may potentiate ulcers. For example horses in these environments usually train at the local racetrack, have limited contact with other animals, and rarely have an opportunity for turnout.

References

1. Anon: Recommendation for the diagnosis and treatment of equine gastric ulcer syndrome (EGUS), *Equine Vet Educ* 122-134, 1999.
2. Becht JL, Byars TD: Gastroduodenal ulceration in foals, *Equine Vet J* 18:307-312, 1986.
3. Traub JL et al.: Phenylbutazone toxicosis in the foal, *Am J Vet Res* 44:1410-1418, 1983.
4. Hammond CJ, Mason DK, Watkins KL: Gastric ulceration in mature thoroughbred horses, *Equine Vet J* 18:284-287, 1986.
5. Roy MA et al.: Prevalence of ulcers of the squamous gastric mucosa in standardbred horses, *J Vet Intern Med* 19:744-750, 2005.
6. Murray MJ et al.: Gastric ulcers in horses: a comparison of endoscopic findings in horses with and without clinical signs, *Equine Vet J Suppl* 7:68-72, 1989.
7. Vatistas NJ et al.: Cross-sectional study of gastric ulcers of the squamous mucosa in thoroughbred racehorses, *Equine Vet J Suppl* 29:34-39, 1999.
8. Vatistas NJ, Synder JR, Carlson GP: Epidemiological study of gastric ulceration in the thoroughbred racehorse:202 horses (1992-1993) *Proceedings, Am Assoc Equine Pract* pp 125-126, 1994.
9. Begg LM, O'Sullivan CB: The prevalence and distribution of gastric ulceration in 345 racehorses, *Aust Vet J* 81:199-201, 2003.
10. Jonsson H, Egenvall A: Prevalence of gastric ulceration in Swedish standardbreds in race training, *Equine Vet J* 38:209-213, 2006.
11. McClure SR, Glickman LT, Glickman NW: Prevalence of gastric ulcers in show horses, *J Am Vet Med Assoc* 215:1130-1133, 1999.
12. Murray MJ et al.: Factors associated with gastric lesions in thoroughbred racehorses, *Equine Vet J* 28:368-374, 1996.
13. Orsini JA, Pipers FS: Endoscopic evaluation of the relationship between training, racing, and gastric ulcers, *Vet Surg* 26:424-428, 1997.
14. Rabuffo TS et al.: Associations between age or sex and prevalence of gastric ulceration in standardbred racehorses in training, *J Am Vet Med Assoc* 221:1156-1159, 2002.
15. Bertone J: Prevalence of gastric ulcers in elite heavy use western performance horses, *American Association of Equine Practitioners* pp 256-259, 2000.
16. Dionne RM et al.: Gastric ulcers in standardbred racehorses: prevalence, lesion description, and risk factors, *J Vet Intern Med* 17:218-222, 2003.
17. Nicol CJ et al.: Study of crib-biting and gastric inflammation and ulceration in young horses, *Vet Rec* 151:658-662, 2002.
18. Vatistas NJ et al.: Induction and maintenance of gastric ulceration in horses in simulated race training, *Equine Vet J Suppl* 29:40-44, 1999.
19. Lorenzo-Figueras M, Jones G, Merritt AM: Effects of exercise on gastric volume and pH in the proximal portion of the stomach of horses, *Am J Vet Res* 63:1481-1487, 2002.
20. Lorenzo-Figueras M, Jones G, Merritt AM: Effects of various diets on gastric tone in the proximal portion of the stomach of horses, *Am J Vet Res* 63:1275-1278, 2002.
21. Widenhouse TV, Lester GD, Merritt AM: Effect of hydrochloric acid, pepsin, or taurocholate on bioelectric properties of gastric squamous mucosa in horses, *Am J Vet Res* 63:744-749, 2002.
22. Murray MJ: Gastric ulceration in horses: 91 cases (1987-1990), *J Am Vet Med Assoc* 201:117-120, 1992.
23. Dukti SA et al.: Prevalence of gastric squamous ulceration in horses with abdominal pain, *Equine Vet J* 38:347-349, 2006.
24. Murray MJ: Equine model of inducing ulceration in alimentary squamous epithelial mucosa, *Dig Dis Sci* 39:2530-2535, 1994.
25. Murray MJ, Eichorn ES: 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, 1996.

26. Coenen M: [The occurrence of feed-induced stomach ulcers in horses], *Schweiz Arch Tierheilkd* 132:121-126, 1990.
27. Nadeau JA et al.: Evaluation of diet as a cause of gastric ulcers in horses, *Am J Vet Res* 61:784-790, 2000.
28. Nadeau JA et al.: Effects of hydrochloric, acetic, butyric, and propionic acids on pathogenesis of ulcers in the nonglandular portion of the stomach of horses, *Am J Vet Res* 64:404-412, 2003.
29. Nadeau JA et al.: Effects of hydrochloric, valeric, and other volatile fatty acids on pathogenesis of ulcers in the nonglandular portion of the stomach of horses, *Am J Vet Res* 64:413-417, 2003.
30. Andrews FM et al.: *In vitro* effects of hydrochloric acid and various concentrations of acetic, propionic, butyric, or valeric acids on bioelectric properties of equine gastric squamous mucosa, *Am J Vet Res* 67:1873-1882, 2006.
31. Sandin A et al.: Effect of nervous excitation on acid secretion in horses, *Acta Physiol Scand* 168:437-442, 2000.
32. Lester GD, Smith RL, Robertson ID: Effects of treatment with omeprazole or ranitidine on gastric squamous ulceration in racing thoroughbreds, *J Am Vet Med Assoc* 227:1636-1639, 2005.
33. Mason GJ: Stereotypies: a critical review, *Animal Behaviour* 41:1015-1037, 1991.
34. Waters AJ, Nicol CJ, French NP: Factors influencing the development of stereotypic and redirected behaviours in young horses: findings of a four year prospective epidemiological study, *Equine Vet J* 34:572-579, 2002.
35. Delacalle J et al.: Nd:YAG laser-assisted modified Forssell's procedure for treatment of cribbing (crib-biting) in horses, *Vet Surg* 31:111-116, 2002.
36. McGreevy PD et al.: Radiographic and endoscopic study of horses performing an oral based stereotypy, *Equine Vet J* 27:92-95, 1995.

Risk Factors for Gastric Ulceration in Thoroughbred Racehorses

by Guy D. Lester, Ian Robertson and Cristy Secombe

RIRDC Publication No. 08/061

Gastric ulceration is an important health concern to the performance horse industries. Economic impacts of this disease are difficult to quantify but include costs associated with diagnosis, medication and labour in administering treatment. Perhaps of even greater economic significance are the costs attributable to reduced athletic performance. Superimposed on any economic impacts are the emerging industry concerns regarding the welfare of performance horses. The reported high incidence of disease across the world is of concern given the common lay opinion that peptic ulcer disease in humans is commonly correlated with emotional stress.

This report identifies specific factors that may be involved in the development and maintenance of gastric ulcers in racing thoroughbreds.

The primary target group of the report is the thoroughbred racing community. It is, however, likely that risk factors will be common to other industries where animals are performing high levels of exercise. The information will also guide attending veterinarians with respect to identifying horses at risk of ulceration.

This report is part of RIRDC's Horse R&D Program which aims to assist in developing the Australian horse industry and enhancing its export potential.

This publication can be downloaded from our website—
www.rirdc.gov.au
RIRDC books can be purchased from:
www.rirdc.gov.au/eshop

Gastric ulcers are more prevalent in working racehorses kept in boxes compared to those grazing with other horses

Contact RIRDC:
Level 2
15 National Circuit
Barton ACT 2600
Ph: 02 6271 4100

PO Box 4776
Kingston ACT 2604