EQUINE GASTRIC ULCER SYNDROME (EGUS): ADULT HORSES

A.M. Merritt, DVM, MS, ACVIM (hon)
**EGUS is NOT just one disease**, but reference to a group of problems with differing etiologies that result in ulceration of either (or both) the non-glandular (squamous) or glandular mucosa.
PRIMARY
NON-GLANDULAR
(SQUAMOUS) MUCOSAL
ULCER DISEASE
EQUINE STOMACH
Primary Squamous Ulcer

Normal

Lesions

Photos courtesy of Dr. Chris Sanchez
Essentially, acidic damage to a vulnerable mucosa, similar to gastro-esophageal reflux disease (GERD) in humans
EQUINE GASTRIC ULCER SYNDROME
1° Squamous Mucosal Ulcer Disease

Common Complaints, Adults in Training

• Poor appetite; failure to finish grain
• Reluctance to train/decreased performance
• Poor body condition, rough hair coat
• Low-grade colics
Primary Squamous Mucosal Ulcer Disease

Cause?

- Specific effects of intensive exercise on GI status?
When the diet includes forage *ad libitum*
pH PROBE IN PROXIMAL STOMACH
Walk
Trot +
Gallop
Lorenzo-
Figueras
and Merritt. AJVR 2002; 63:1481

t Turn on
Treadmill Motor

pH proximal stomach

Fed
Fasted

Time blocks

(n=3)
Non-glandular or Squamous
Duodenum

HIGH DENSITY LIQUID
pH 1-2

Increased intra-abdominal pressure

revised from Schummer et al., 1979
Primary Squamous Mucosal Ulcer Disease

Cause?

- Specific effects of intensive exercise on GI status?
- “Stress”?

Remember: “Classic” stress ulcer affects glandular, but not non-glandular, mucosa.
Primary Squamous Mucosal Ulcer Disease

Cause?

• Specific effects of intensive exercise on GI status?

• “Stress”? Is tensing of abdominal muscles part of the stress response in horses??
Primary Squamous Mucosal Ulcer Disease

Recent studies in Australia by Lester et al. indicate that more ulceration occurs in horses where:

1. The housing setup minimizes animal to animal contact
2. The training location is urban vs. rural
3. There is more talk than music on the barn radio.
Primary Squamous Mucosal Ulcer Disease

Cause?

- Specific effects of intensive exercise

- “Stress”? Initially, most prevalence studies suggested that the lesions are markedly reduced or disappear, without anti-ulcer medication, if the horse is taken out of training.

However....
The prevalence and anatomical distribution of equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark

N. LUTHERSSON*, K. HOU NIELSEN†, P. HARRIS‡ and T. D. H. PARKIN§

Fig 2: Anatomical location of EGUS severity score ≥2 in the 5 different work type groups in the population examined (presented as percentage horses affected in each of the work type groups). Numbers at the top of each bar represent the number of horses in each category. ■ = Ulcers in glandular region only; □ = Ulcers in nonglandular region only; ○ = Ulcers in both regions.
Primary Squamous Mucosal Ulcer Disease

Cause?

• Specific effects of intensive exercise on GI status?

• “Stress”?

• High concentrate diet: large amount of intra-gastric VFA production that potentiates HCl corrosiveness?
Intra-gastric microbial fermentative activity of non-structural CHO
In vitro effects of hydrochloric acid and various concentrations of acetic, propionic, butyric, or valeric acids on bioelectric properties of equine gastric squamous mucosa

Frank M. Andrews, DVM, MS; Benjamin R. Buchanan, DVM; Sionagh H. Smith, DVM, PhD; Sarah B. Elliott, BS; Arnold M. Saxton, PhD

Conclusions and Clinical Relevance—The VFAs, especially acetic acid, in the presence of HCl at a pH of ≤ 4.0 appear to be important in the pathogenesis of NG mucosal ulcers in horses. (Am J Vet Res 2006;67:1873–1882)
Risk factors associated with equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark

N. LUTHERSSON*, K. HOU NIELSEN†, P. HARRIS‡ and T. D. H. PARKIN§

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

<table>
<thead>
<tr>
<th>Variable</th>
<th>Single level model</th>
<th>Mixed effects model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Odds ratio</td>
<td>95% CI</td>
</tr>
<tr>
<td>Available forage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hay or haylage (ref)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Straw only</td>
<td>5.7</td>
<td>2.0–16.7</td>
</tr>
<tr>
<td>Water available in the paddock?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes (ref)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>2.5</td>
<td>1.2–5.1</td>
</tr>
<tr>
<td>Starch intake</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1 g/kg bwt/meal (ref)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1–2 g/kg bwt/meal</td>
<td>2.6</td>
<td>1.3–5.2</td>
</tr>
<tr>
<td>≥2 g/kg bwt/meal</td>
<td>3.2</td>
<td>1.3–7.7</td>
</tr>
</tbody>
</table>

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

Cause?

• Specific effects of intensive exercise on GI status?

• “Stress”?

• High concentrate diet: large amount of intra-gastric corrosive VFA production?

• Meal feeding?
EQUINE PROXIMAL GASTRIC pH OVER 3 DAYS

Fed 12 hrs, fasted 12 hrs, fed 24 hrs, fasted 24 hours

No fasting; Hay free choice

Husted et al., Equine Vet. J. 2009
Risk factors associated with equine gastric ulceration syndrome (EGUS) in 201 horses in Denmark

N. LUTHERSSON*, K. HOU NIELSEN†, P. HARRIS‡ and T. D. H. PARKIN§

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

<table>
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<tr>
<td>Available forage</td>
<td></td>
<td></td>
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<tr>
<td>Hay or haylage (ref)</td>
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<tr>
<td>Straw only</td>
<td>4.5</td>
<td>1.6–12.5</td>
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<tr>
<td>Water available in the paddock?</td>
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<tr>
<td>Yes (ref)</td>
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<td></td>
</tr>
<tr>
<td>No</td>
<td>2.4</td>
<td>1.2–5.1</td>
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<tr>
<td>Starch intake</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1 g/kg bwt/meal (ref)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>≥1 g/kg bwt/meal</td>
<td>2.4</td>
<td>1.3–4.6</td>
</tr>
<tr>
<td>Interval between forage feeds</td>
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<td></td>
</tr>
<tr>
<td>&lt;6 h (effectively ad lib.)</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>&gt;6 h</td>
<td>3.9</td>
<td>1.5–10.4</td>
</tr>
</tbody>
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Crib-Biting and Squamous Ulcers?
Evaluation of Intra-Abdominal Pressure in Horses That Crib

Valeria Albanese¹, DVM, Amelia S. Munsterman¹, DVM, MS, Diplomate ACVS & ACVECC, Fred J. DeGraves², DVM, PhD, and R. Reid Hanson¹, DVM, Diplomate ACVS & ACVECC

¹ Auburn University, Auburn, Alabama and ² Western Kentucky University, Bowling Green, Kentucky

Veterinary Surgery Proc., 2013

**Figure 3** Mean intra-abdominal pressures (IAP) in cribbing horses (orange) compared to noncribbing horses (blue) during the 2-hour study. X-axis corresponds to time (minutes), Y-axis to IAP (mmHg).
Primary Squamous Mucosal Ulcer Disease

Goals of Therapy

1. Increase overall intra-gastric pH to >4.0 by either suppressing gastric acid secretion or buffering the contents. The longer acting the effect, the better.
Rx: 4 mg/kg OD for 21-28 days
Px: 1 mg/kg OD
Table 2—Odds of moderate or severe gastric ulceration as a function of antiulcer treatment during the preceding 2 weeks in 798 racehorses in active race training

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No treatment</td>
<td>Reference</td>
<td>NA</td>
<td>NA</td>
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<tr>
<td>Buffer</td>
<td>1.69</td>
<td>0.70–4.06</td>
<td>0.24</td>
</tr>
<tr>
<td>Sucralfate</td>
<td>0.59</td>
<td>0.22–1.60</td>
<td>0.29</td>
</tr>
<tr>
<td>H₂ receptor antagonist</td>
<td>1.43</td>
<td>0.75–2.74</td>
<td>0.27</td>
</tr>
<tr>
<td>Compounded omeprazole</td>
<td>1.54</td>
<td>0.54–4.37</td>
<td>0.42</td>
</tr>
<tr>
<td>Proprietary omeprazole</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Low dosage*</td>
<td>0.18</td>
<td>0.08–0.40</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>High dosage†</td>
<td>0.18</td>
<td>0.10–0.32</td>
<td>&lt; 0.001</td>
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*Administered at a dosage of 2 mg/kg (0.9 mg/lb), PO, every 24 hours. †Administered at a dosage of 4 mg/kg (1.8 mg/lb), PO, every 24 hours.

See Table 1 for remainder of key.
What about compounded omeprazole??

NOT RECOMMENDED!

Potency and bioavailability are unreliable
Primary Squamous Mucosal Ulcer Disease

Goals of Therapy

1. Increase overall intra-gastric pH to >4.0 by either suppressing gastric acid secretion or buffering the contents. The longer acting the effect, the better

2. Modify feeding practice/housing arrangement
Equine vet. J. (2009) 41 (7) 625-630

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

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Compared to coastal bermuda hay fed at same amount/kg/d

Both groups also received a pelleted concentrate

MEAL FED!

Horses were subjected only to light exercise

Buffering effects of the high Ca and protein content??
DEVICES TO PROLONG HAY INGESTION

Greater and more sustained salivary secretion
Primary Squamous Mucosal Ulcer Disease

FEEDING STRATEGIES AIMED AT REDUCING INCIDENCE

• Less grain (supplement with oils for extra calories?)

• Forage always available (no meal feeding)

• More alfalfa (luzerne) hay

• Supplemental forage in “nibble bag”
NSAID INDUCED GLANDULAR DISEASE
NSAID-INDUCED GLANDULAR ULCERS
IF ON LONG-TERM NSAID THERAPY CONSIDER

CORN OIL supplementation of diet; 10 ml/50 kg daily
NON-NSAID ASSOCIATED GLANDULAR MUCOSAL DISEASE
HORSE STOMACH

Non-glandular or Squamous

Most frequent site of non-NSAID related glandular ulceration

Adapted from Nickel et al., 1979
PYLORIC GLANDULAR MUCOSAL DISEASE

(Murray et al.; JVIM 2001;15:401)

162 Horses

Clinical signs that precipitated a request for gastroscopy included:

- Diminished appetite (50)
- Recurrent or intermittent colic (33)
- Acute colic (22)
- Other colic (12)
- Poor body condition or recent weight loss (19)
- Signs related to behavior or attitude (16)
- Other signs (18)
- No signs (18)

Thirty-one horses had more than 1 sign.
Fig 7. Grade 4 lesion in the mucosa immediately adjacent to the pylorus (P) in a horse. Underlying fibrosis is indicated by the tissue being stiff and rigid when probed with a biopsy forceps.

Murray et al.; JVIM 2001;15:401
Thanks to Dr. Carsten Dicks
EQUINE STOMACH
Antral Mucosa and Pylorus

Normal

Gastritis
EGUS - 2º SQUAMOUS DISEASE - ADULTS

- Severe pyloric gland disease
- EGUS related stricture
- Non-EGUS related stricture

Evidence of Physical Obstruction of Gastric Outflow

Proximal to Duodenal Diverticulum!!
No pathogenic *Helicobacter* has yet been found in equine gastric tissue
Risk Factors?

Butcher et al., ACVIM Proc. 2012 (abstract):
- feeding of unprocessed grains
- infrequent feeding of a complete diet
- lack of haylage feeding
- no grass turnout
Current evidence implicates a common risk factor of meal feeding of diets that contain moderate to large amounts of non-structural carbohydrates in the pathogenesis of both glandular and non-glandular (squamous) ulcers.

Verification of this implication by additional epidemiological studies is of utmost importance.
NON-NSAID ASSOCIATED GLANDULAR MUCOSAL ULCERS

TREATMENT CONSIDERATIONS

1. Increase the intra-gastric pH?
   There are evidence-based indications that this may be effective, especially when the lesions appear to be more acute
   BUT
   Even oral OME at 4 mg/kg may be ineffective in promoting healing
NON-NSAID ASSOCIATED GLANDULAR MUCOSAL ULCERS

TREATMENT CONSIDERATIONS

1. Increase the intra-gastric pH?

2. Adjunct? Biopsy of intra-gastric lesions and duodenal mucosa may provide some guidance
   - Eosinophilic infiltrate: corticosteroids (CS)
   - Neutrophilic infiltrate: antibiotics? (AB)
   - Plasmacytic/Lymphocytic infiltrate: AB?, CS?
NON-NSAID ASSOCIATED
GLANDULAR MUCOSAL ULCERS

TREATMENT CONSIDERATIONS

There is much to be learned!!

Very dependent upon identifying specific etiologies