# **Top Medicine Studies of 2012 (AAEP 2012)**

By Nancy S. Loving, DVM

Updated: Monday, February 4, 2013 8:00 AM Posted: Monday, February 4, 2013 7:40 AM

# Originally published on TheHorse.com

Each year equine veterinarians attending the American Association of Equine Practitioners (AAEP) Convention flock by the thousands to one of the meeting's headline events: the Kester News Hour. Stephen Reed, DVM, Dipl. ACVIM, of Rood & Riddle Equine Hospital in Lexington, Ky., presented a summary of many recent practical and applicable equine medicine studies to a packed audience at the 2012 convention, held Dec. 1-5 in Anaheim, Calif.

# Endocrinology, Metabolic Syndrome, and Laminitis

First, Reed addressed a commonly diagnosed disease in older horses: pituitary pars intermedia dysfunction (PPID), which is often referred to as equine Cushing's disease. He reported on a retrospective study in which researchers examined the medical records of 217 horses diagnosed with PPID from three veterinary teaching hospitals from 1993 to 2004. Notably, there was a proportional increase in PPID diagnoses from 2.5 per 1,000 horses in 1993 to 3.72 horses per 1,000 in 2002.

The most common findings veterinarians noted in these horses were hirsutism (excessive hairiness) in 84% and laminitis in 50%. In most cases the time from the onset of clinical signs to diagnosis was 180 days. Reed noted that about half of the horses diagnosed with PPID survived 4  $\frac{1}{2}$  years following a confirmed diagnosis. Treatment success varied with the medication: a combination of cyproheptadine and compounded pergolide yielded 60% success; pergolide alone resulted in 40% success; and cyproheptadine alone in 29% success. These medications were all that were available prior to the FDA approval of pergolide (Prascend, released in December 2011).

Veterinarians can diagnose PPID using a variety of tests, each of which poses a unique challenge. Historically, veterinarians considered the overnight dexamethasone suppression test the gold standard, but currently, more veterinarians are sampling for plasma ACTH (adrenocorticotrophic hormone). Reed mentioned another evaluation test for PPID:  $\alpha$ -MSH ( $\alpha$ -melanocyte-stimulating hormone). Of 126 horses with a mean age of 21.5 years slated for euthanasia, researchers identified 109 as normal, based on testing for ACTH,  $\alpha$ -MSH, and cortisol; 17 had clinical hirsutism. Following euthanasia, researchers measured each horse's pituitary gland and evaluated it for pathology.

In summary, Reed noted, "All the currently available tests were good at correctly identifying the problem in end-stage cases of PPID." He advised of another test that may prove useful in future: the TRH (thyroid-releasing hormone) stimulation test that allows blood sampling at five and 20 minutes following TRH administration.

In the third paper Reed addressed researchers looked at the use of digital cryotherapy (ice therapy) for laminitis treatment. The investigators used an oligofructose model, giving a large bolus of the carbohydrate to induce laminitis. At the first sign of lameness at a walk, investigators placed one forelimb in ice and kept the other at ambient temperature. Then researchers scored the severity of laminitis based on pathology following euthanasia. Researchers could complete a reasonable pathologic analysis on only four of the nine horses' non-iced legs due to severe damage. The iced legs showed much less evidence of pathologic injury. Reed was excited by the researchers' findings that demonstrated support for limb icing following the onset of clinical signs in acute laminitic cases.

## **References:**

- McFarlane D, Breshears MA, Cordero M, et al. Comparison of plasma ACTH concentration, α-MSH concentration, and overnight dexamethasone suppression test for diagnosis of PPID. J Vet Intern Med 2012.
- Van Eps A, Pollitt C, Underwood C, et al. Digital hypothermia applied after the onset of lameness prevents lamellar failure in the oligofructose laminitis model. *Am Coll Vet Intern Med Equine Research Forum* May 2012.
- McGowan TW, Pinchbeck GP, McGowan CM. Prevalence, risk factors and clinical signs predictive for equine pituitary pars intermedia dysfunction in aged horses. Equine Vet J 2012;44:1-5.
- Rohrbach BW, Stafford JR, Clermont RSW, et al. Diagnostic frequency, response to therapy, and long-term prognosis among horses and ponies with pituitary pars intermedia dysfunction, 1993-2004. J Vet Intern Med 2012;26:1027-1034.
- Karikoski NP, Horn I, McGowan TW, et al. The prevalence of endocrinopathies laminitis among horses presented for laminitis at a first-opinion/referral equine hospital. Domestic Anim Endocrinology 2011;41(3):111-117.

#### Foals

Neonatal foals are quite susceptible to gastric ulcers due to any kind of stress situation and/or illness. Both historically and currently, clinicians have been/are eager to medicate with anti-ulcer meds at the initial presentation of a sick foal. Therefore, Reed described a study in which researchers examined the use of histamine-type 2 receptor antagonists (such as omeprazole).

He noted that anti-ulcer medications, specifically omeprazole, increase neonatal foals' risk of developing diarrhea. Increases in *Clostridium dificile* are associated with diarrhea in human patients, but this was not seen in 1,600 foal diarrhea cases. Reed stressed, "In foals treated with anti-gastric ulcer medications, the study showed that hospital-associated diarrhea increased twofold while at the same time there was no reduction in foal ulcers." Clinicians should evaluate each neonate case very carefully to confirm ulcers before initiating anti-ulcer medication treatment.

Reed then took up the topic of the *Lawsonia* organism's effects in weanlings. The proliferative inflammation of the small intestine and colon (enterocolitis) leads to classic signs of increased thickening of the small intestinal mucosa and subsequent poor body condition. In addition, practitioners should be aware of the potential for necrotizing enteritis associated with *Lawsonia*; this can cause acute, severe gastrointestinal

signs such as endotoxemia and death.

Moving on, Reed asked, "What is the prevalence of antimicrobial-associated diarrhea (AAD) in weanlings?" He described a study in which scientists reviewed records of 5,251 weanling horses from three referral practices--all were treated with antibiotics for non-gastrointestinal-related problems. Although the incidence of AAD was very low (0.6%) among these horses, nearly 19% of the diarrhea cases, had a fatal outcome. Reed stressed that drugs from any antimicrobial class can cause AAD due to the tendency for usual intestinal organisms to become opportunistic, intestinal pathogens.

# **References:**

- Elfenbein JR, Sanchez LC. Prevalence of gastric and duodenal ulceration in 691 nonsurving foals (1995-2006). *Equine Vet J* 2012;44(Suppl 41):76-79.
- Furr M, Cohen N, Axon J, et al. Treatment with histamine-type 2 receptor antagonists and omeprazole increase risk of diarrhoea in neonatal foals treated in intensive care
  units. *Equine Vet J* 2012;44(Suppl 41):80-86.
- Page AE, Fallon LH, Bryant UK, et al. Lawsonia intracellularis-associated necrotizing enterocolitis in 4 weanling horses. J Equine Vet Sci 2012;32:S-37.
- Vannucci FA, Pusterla N, Mapes SM, et al Evidence of host adaptation in Lawsonia intracellularis infections. J Equine Vet Sci 2012;32:S44.
- Barr BS, Waldridge BM, Morresey PR, et al. Anti-microbial associated diarrhoea in three equine referral practices. *E Vet Jour* 2012; July 11(E pub ahead of print). Neurologic and Muscle Diseases

Reed next described MADD (multiple acycl-CoA dehydrogenase deficiency), which veterinarians also refer to as seasonal pasture myopathy. This is a highly fatal form of non-exertional rhabdomyolysis seen in autumn and spring, and it resembles an atypical myopathy veterinarians have observed in Europe.

Based on case histories, veterinarians diagnosed six North American horses with seasonal pasture myopathy. They tested urine and muscle samples, looking for organic acids that might suggest abnormal lipid metabolism. In affected muscles they noted degeneration as well as lipid accumulation in the myofibrils. In atypical myopathy cases in Europe, researchers believe an enzyme deficiency causes the biochemical deficit; in a similar disease that affects humans, there appears to be a genetic predisposition. But, with the variety of horse breeds affected, Reed suggested that MADD is less likely to have a genetic basis. Instead, researchers have identified that horses consuming a particular toxin, hypoglycin A--present in the seeds of the box elder tree--develop the myopathy.

Recent outbreaks of equine herpesvirus in the United States have caused widespread concern. Reed discussed a study in which the researchers were interested in understanding the epidemiology of herpes outbreaks, specifically seeking to determine the period of time that infected horses shed the virus. Of 104 horses in an outbreak, 20 exhibited neurologic signs, veterinarians took daily nasal swab samples from the neurologic cases. Two-thirds of the horses tested positive for virus on swabs from Day 0 to 3. But, most importantly, Reed said, "The last day of detected shedding for all horses was disease Day 9." The quantity of viral DNA on the swabs was not associated with severity of disease or the horse's age. Whether a horse developed signs of equine herpes depended on if he had a fever and/or was introduced to susceptible horses, for example, as in a boarding facility. Reed emphasized, "The take-home message is that biosecurity measures should be observed for 14 days beyond the last onset of clinical signs of the last horse in the outbreak."

# **References:**

- Kydd JH, Slater J, Osterrieder, N, et al. The third International Havemeyer Workshop on Equine Herpesvirus type 1. Equine Vet J 2012; 44: 513-517.
- Burgess BA, Tokateloff N, Manning S, et al. Nasal shedding of equine herpesvirus-1 from horses in an outbreak of equine herpes myeloencephalopathy in Western Canada J Vet Intern Med 2012;26:384-392.
- Sponseller BT, Valberg SJ, Schultz NE, et al. Equine multiple acyl-CoA dehydrogenase deficiency (MADD) associated with seasonal pasture myopathy in the Midwestern United States. *J Vet Int Med* 2012;26:1012-1018.
- Valberg SJ, Sponseller BT, Hegeman AD, et al. Seasonal pasture myopathy/Atypical myopathy in North America associated with ingestion of Hypoglycin A within seeds of the box elder tree. *E Vet J* 2012 E pub.

## Respiratory

Reports from the 9th International Conference on Equine Infectious Disease in October 2012 filled the latter stretches of Reed's literature summaries. Researchers have historically believed that *Streptococcus equi* subspecies *equi* is a stable organism, but they recently determined it has a dynamic and flexible genome.

In another study Reed described, researchers reported that *S. equi*-related guttural pouch empyema (a collection of pus within the guttural pouch, which is an amplified Eustachian tube running from the ear to the pharynx) is most likely caused by failure to clear the organism from the pouch rather than a retropharyngeal (behind the pharynx) abscess breaking into the guttural pouch. Also, several clones of the organism might reside in the guttural pouches of horses that are strangles carriers.

Another paper Reed relayed centered on purpura hemorraghica (a condition of hemorrhage and edema that is triggered by an allergic immune reaction to the M protein from *S. equi* bacteria) following vaccination for strangles. He noted that SeM protein blood titers are very high following natural exposure and that antibodies might be detectable in the blood for more than a year. The take-home message is that veterinarians should consider vaccinating for strangles only if the horse's titer has been checked; in fact, it might be necessary to wait two years following infection in order to avoid vaccine-related complications such as purpura.

The final report that Reed presented dealt with the association of clinical signs seen with nasopharyngeal cicatrix (scar formation) via endoscopic exam. From 2003 to 2008, researchers compared 118 cases to 121 controls. Clinical signs were fairly benign; i.e., horses exhibited clear nasal discharge, respiratory noise, or exercise intolerance if a circumferential lesion (one that is fully encircling) was present. The horse might experience respiratory distress if there was greater than 50% obstruction. Reed emphasized that veterinarians should include nasopharyngeal cicatrix in a differential diagnosis if a horse demonstrates any or several of these signs. The underlying cause is unknown but might be associated with an irritant, infection, or allergen.

## **References:**

- Harris SR, Webb K, Aanensen DM, et al. Diversity microevolution and within-host niche adaptation of Streptococcus equi. J Equine Vet Sci 2012;32:S-12.
- Bustos CP, Munoz AJ, Picos JA, et al. Different strains of *Streptococcus equi equi* isolated from a guttural pouch empyema. *J Equine Vet Sci* 2012;32:S-19.
- Boyle AG, Smith MA, Boston RC. Risk factors for high serum SeM titers after natural outbreaks of *Streptococcus equi equi* in horses. *J Equine Vet Sci* 2012;32:S-19.
- Holcombe SJ, Hurcombe SD, Barr BS, et al. Dysphagia associated with presumed pharyngeal dysfunction in 16 neonatal foals. Equine Vet J 2012;44(Suppl 41):105-108.
- Norman TE, Chaffin MK, Bisset WT, et al. Association of clinical signs with endoscopic findings in horses with nasopharyngeal cicatrix syndrome: 118 cases (2003-2008) J Am Vet Med Assoc 2012:240(6);734-739.

Disclaimer: Seek the advice of a qualified veterinarian before proceeding with any diagnosis, treatment, or therapy.