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# AAEP Convention Highlights

In Part 4 of our 2018 AAEP Convention coverage, brought to you by Zoetis, we cover treatment options.

**A**t the 2018 AAEP Convention, held in San Francisco, California, there were many presentations and moderated discussions that veterinarians could attend. In this article, we have synopses of several of those presentations and discussions, brought to you by Zoetis.

## Vitamin E Supplementation in Horses

A variety of neuromuscular diseases develop due to vitamin E deficiency. Clinical signs associated with dietary deficiency of this fat-soluble vitamin include an abnormal stance, ataxia,

muscle weakness and muscle atrophy, to name a few.

Equine motor neuron disease (EMND), vitamin E deficient myopathy (VEM), and dystrophy/equine degenerative myeloencephalopathy (eNAD/EDM) are syndromes associated with vitamin E deficiency.

Vitamin E is a potent anti-oxidant, and it has multiple forms, but alpha-tocopherol (a-TOH) is the most significant bioavailable form that horses need for immune function as well as neuromuscular and reproductive health.

Pasture is a rich source of vitamin E for horses; however with drought conditions, seasonal effects in northern

climates, and increasing urbanization, horses are more regularly faced with loss of pasture. Carrie Finno, DVM, PhD, DACVIM, from the University of California, Davis, explained that once pasture is cut and harvested, vitamin E oxidizes immediately, making that forage vitamin E deficient. Ideally, horses are provided with fresh pasture at least six months of the year.

Measurement of appropriate vitamin E status in the horse relies on measurement of alpha-TOH in serum. Horses grazing on pasture for at least 12 hours a day tend to have levels of 3-4 ug/ml. Levels of alpha-TOH >2 ug/ml are considered acceptable and within

“normal” range. Even in horses that seem “normal” in previous studies, serum alpha-TOH levels were measured as only 1.52 ug/ml, which is abnormally low. It takes a while until muscle atrophy is visibly apparent, despite the deficiency. Performance issues are also associated with low vitamin E levels.

Finno stressed that it is important to avoid hemolysis of the blood sample otherwise concentrations of alpha-TOH might falsely decrease by as much as 33%. Remove serum as soon as possible after collection in a red top tube. Also, alpha-TOH is light sensitive and must remain chilled for submission to the lab.

Supplements for vitamin E come in different types, including powder, pellet or as a liquid. It also comes in two forms:

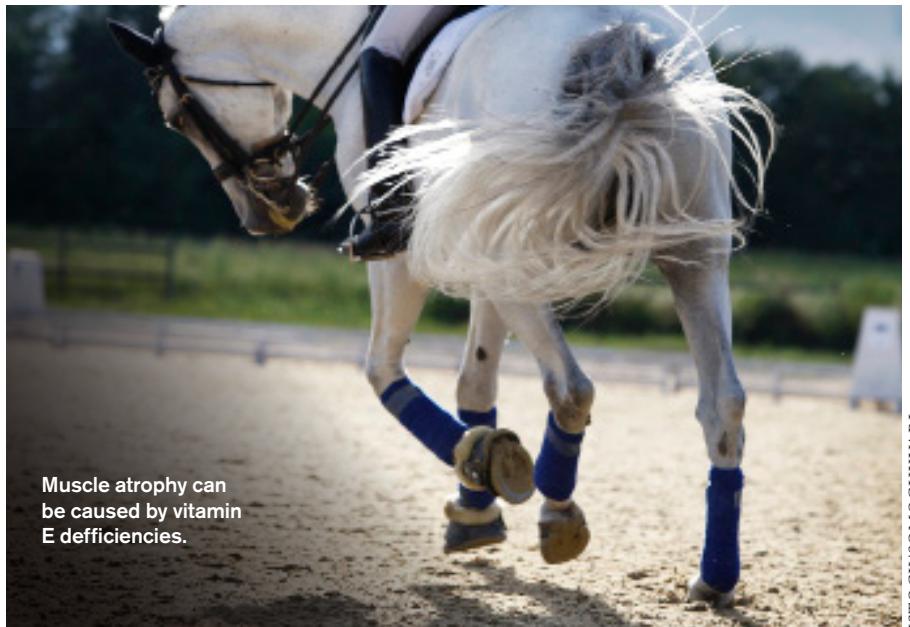
- Natural form—this is referred to as RRR alpha-TOH, which is the bioavailable form
- Synthetic form

Of note is that all vitamin E in commercial grains is a synthetic form, and Finno stresses that no amount of synthetic product will increase a horse’s vitamin E level.

It takes 8-10 weeks to appreciate vitamin E elevations in a deficient horse if consuming a natural powdered or pelleted formulation. In contrast, it takes less than one day to elevate vitamin E to a normal range if vitamin E is supplemented in a natural liquid form.

Liquid is the best form to give to a diseased horse, especially since the liquid form increases vitamin E concentrations in the CSF within two weeks.

If an owner decides, due to expense, to switch from the liquid natural form to the powdered or pelleted natural form, Finno recommended overlapping treatment with both, then slowly withdrawing the liquid form. This avoids a



Muscle atrophy can be caused by vitamin E deficiencies.

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precipitous drop in serum alpha-TOH levels.

If a horse is withdrawn entirely from vitamin E supplementation, sufficient serum levels of alpha-TOH are depleted by 4-6 months as its storage in fat and muscle is used up. It is recommended that serum alpha-TOH can be checked at the same time that a Coggins’ test is drawn in the spring.

Finno reported that every individual horse’s response is different, so it is important to acquire a baseline serum sample before supplementation begins, then compare that to serum samples following supplementation.

Too much supplementation of vitamin E can be fraught with problems, such as bleeding disorders, effects on B-carotene, and interference with metabolism of some drugs. Dosage can be adjusted based on monitoring serum samples.

## **Magnesium With or Without Boron for Headshaking**

Trigeminal-nerve-mediated headshaking is an extremely frustrating syndrome for horse owners and their veterinarians. The neuropathic pain creates itching, tingling and electric-shock-like sensations that cause a horse to jerk its head suddenly, snort, rub or strike its face, or shake its head. This

makes it difficult, at best, to ride the horse successfully.

Other causes can create somewhat similar signs, such as simple pain, a mass or sinusitis.

The incidence of trigeminal-mediated headshaking is 1-4.6%, with three-fourths of cases in geldings, especially in Quarter Horse or Thoroughbred breeds. The worst cases occur seasonally in spring and summer and might decrease with shortening daylight hours in fall and winter.

Studies have shown that while normal nerve firing of the trigeminal nerve requires 25 mA (milliamps) of light, the trigeminal nerve of a horse with idiopathic headshaking fires at just 2.6 mA.

A number of remedies are tried: face masks with ultraviolet light protection, nose nets, melatonin, cyproheptadine, an Equitens unit, or caudal compression of the infraorbital nerve, for example.

An owner-based survey showed that 43% of horses supplemented with oral magnesium oxide (5-40 mg/kg) experienced some resolution of the headshaking. Intravenous magnesium sulfate achieved reduction in 19%. At the 2018 AAEP Convention in San Francisco, Shara Seldon, PhD, at the Department of Veterinary Medicine and Epidemiology at the University of California, Davis, presented a study using magne-

sium supplementation with or without boron. Her paper was co-authored with Drs. Monica Aleman, Laos Costa and John Madigan.

Boron is a trace mineral applicable to wound healing, arthritis and bone growth, and it also increases blood magnesium by increasing its ionized form.

In the headshaking study, 12 geldings—six with headshaking, six as controls—aged 6-13 years were part of a crossover study with three supplements:

- Pelleted feed (3/4 cup of Equine Senior) with ¼ cup canola oil and two tablespoons of applesauce
- Magnesium citrate added at 24.2 mg/kg per day to the above base feed
- Boron (40 mg/kg) added to the magnesium-supplemented base feed.

The horses were fed hay for a week, then one of the three supplements listed above. Then after a one-week washout, they were fed another of the supplements; and this was repeated for a third round so that each horse received each supplement.

Headshaking frequency of each horse was compared to dietary intake of the hay-only diet and to each supplement. Evaluation of the horses in a round pen measured the number of headshaking events per minute at all gaits since headshaking is known to increase with gait speed.

The results are encouraging using supplements compared to the hay diet only:

- With pelleted feed, headshaking decreased by 44%;
- With magnesium in the pelleted feed, headshaking decreased by 52%;
- With both magnesium and boron in the pelleted feed, headshaking decreased by 64%.

Supplementing with both magnesium and boron in a pelleted diet is



Supplementing headshakers with magnesium and boron showed promise in one study.

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yet another method to potentially help managing trigeminal-mediated headshaking.

### **Intra-articular Polyacrylamide Gel**

Scott McClure, DVM, presented on the use of polyacrylamide gel (PAAG) for intra-articular treatment of equine osteoarthritis (OA). He explained that there is no such thing as an FDA-approved veterinary device and that PAAG gel is considered a device.

There are many forms of PAAG, such as those used in contact lenses, diapers, electrophoresis, etc. The material is inert, non-reactive and non-immunogenic. Differences in materials are based on the percentage amounts of polyacrylamide, the cross-linkers and the temperature at which the materials were made.

A 2.5% PAAG used in a goat model demonstrated no adverse events; nerve endings remained normal, synovial membranes thickened, and the goats' soundness improved with treatment. The 2.5% PAAG formulation was then used to treat 43 horses that were minimally affected with osteoarthritis with lesions less than six months old. At one month, 59% improved; by 24 months, the PAAG gel was still present in the injected joints, and 82.5% of the treated

horses were not lame.

Use of 4% PAAG in 512 human knees achieved improvement by six weeks; the effects lasted for 12-18 months.

McClure and his team administered 4% PAAG into normal fetlocks of six horses on Days 7, 28 and 56, comparing results to a control group. Initial responses included mild effusion and turgidity of the joint that lasted for 21-29 days. White blood cells increased in low numbers at one week in the joints. Within seven days, PAAG was identified on the surface of the synovia and was seen within the interstitial spaces of the synovial membrane by Day 28.

A field trial study administered 4% PAAG to horses with naturally occurring osteoarthritis to see whether horses could improve by at least one grade of lameness by 45 days post-injection, or whether the horses could experience a combined reduction of the following: pain score, range of motion and joint swelling. The trial involved 36 joints in 35 horses treated one time. Most of the horses in the study were middle-aged warmbloods. At 45 days, 82% had success; at 90 days, 75% still showed improvement.

By 16 months following treatment with 4% PAAG, the synovia appeared relatively normal and no detrimental effects had occurred. **EM**

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