



# U of M Horse Newsletter

Providing research-based information to Minnesota Horse Owners

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## Overo Lethal White Syndrome (OLWS)

The overo coat pattern features white markings that do not cross the back of the horse between its withers and its tail. An overo may be either predominantly dark or white. OLWS is a recessive disorder (two copies of the mutation are necessary to produce disease) that appears to have been selected for in horses where white spotting is a desirable trait. The defective gene has been found in American Paint Horses, American Miniature Horses, Half-Arabians, Thoroughbreds, and crop-out (horses with excessive white markings) Quarter Horses.

Horses heterozygous for the mutation most commonly have an overo-type white coat color pattern with the highest association in frame overos (> 94% heterozygotes, frame overos typically have white patches centered in the body and neck). However, there certainly are many exceptions within horses of overo lineage where horses without white coloration carry the mutation (up to 20% incidence of heterozygotes in broodstock lacking excessive white patterning).



Photo Credit: Stephanie Valberg, University of Minnesota

OLWS foals are homozygous for the mutation and are born alive with blue eyes and a white coat. Small black markings on the head, mane and tail can occur in OLWS foals. These foals initially

appear normal except for their unusual coloring. After a varying period of time, signs of colic emerge due to the foal's inability to pass feces. The OLWS foal has an underdeveloped, defective intestine caused by a failure of the embryonic cells that form nerves in the gastrointestinal system. Oddly enough, these cells also play a role in determining skin color.

There is no treatment for OLWS, and surgery to bypass the intestinal damage has never been successful due to the extensive nature of this type of lesion. Euthanasia is advised for all OLWS foals with colic. There are some completely white foals that do not have OLWS. Although owners call these "living lethals" they actually are heterozygous for the OLWS mutation or have a separate viable white coat color gene.

Testing for the OLWS gene is performed at the Veterinary Genetics Laboratory at the University of California, Davis ([www.vgl.ucdavis.com](http://www.vgl.ucdavis.com)). This test is recommended for all frame overos and their descendants. Mares or stallions that have previously produced an OLWS foal are in all likelihood heterozygous for the mutation. Carriers should not be bred to each other to avoid the birth of OLWS foals.

The horses at greatest risk of carrying the defective allele are overos, particularly of American Paint Horses and American Miniature Horse breeding. A small number of Tobiano and breeding stock horses also carry the defective gene, and a very small number of carrier horses have been detected in other breeds. These other carriers include Pinto horses, which indicate that as other breeds import overo color patterning, they can also import the lethal gene.

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### Upcoming Events

#### Equine Forage, Nutrition and Facilities Management Hands-On Field Day

Tuesday, August 24th  
5:00 to 9:00 pm

Equine Center & Plot Area  
St. Paul, MN

Topics include: Pasture Management, Supplements for Grazing Horses, Mud Management, Manure Management, Grass ID, and Grazing Plot Tour

#### Fall Horse Owner Programs

Saturday, October 2nd  
9:00 am to 12:30 pm

Equine Center  
St. Paul, MN

Topics include: Dentistry, Nutrition Roundtable, Elderly Horse Care, Research Update, Fly Control, How Do You Roll?, and Q & A with Presenters.

Saturday, November 6th  
1:00 to 4:30 pm

Apollo High School  
St. Cloud, MN

Cost to attend each program is \$20/person  
Program Sponsors are ADM Alliance Nutrition and Purina Mills

Register for all programs at [www.extension.umn.edu/horse](http://www.extension.umn.edu/horse)



## Injection of the Coffin Joint to Treat the Navicular Area

Navicular syndrome is a common cause of lameness in horses. Horses diagnosed with this syndrome are often treated with coffin joint injection of the corticosteroid triamcinolone acetonide (TA) in combination with sodium hyaluronate (HA). Although injection of the coffin joint relieves lameness in many of these cases, it has been suggested that direct injection of corticosteroid into the navicular bursa is a more specific treatment which results in a better outcome. Injection of the navicular bursa is a more difficult procedure than injection of the coffin joint though, often requiring radiographic guidance to place the needle in the bursa. Previous studies have demonstrated that there is no direct communication between the coffin joint and the bursa so it is unknown whether TA will diffuse from the coffin joint to the bursa. Additionally, HA is a thick solution and could potentially block diffusion. However, we suspected that TA would diffuse from the coffin joint to the navicular bursa in normal horses and that this diffusion would not be hindered by the addition of HA. The overall goal of our study was to determine whether injection of TA into the coffin joint was a viable treatment option for horses with navicular syndrome.

In the first part, 11 horses with no evidence of foot pain or navicular syndrome were enrolled in the study. One forelimb coffin joint was injected with TA and the opposite forelimb coffin joint injected with TA + HA. One hindlimb served as a control limb (to determine if diffusion of the corticosteroid was through the bloodstream). Six hours after

injection of the coffin joints, navicular bursal fluid samples were taken from each forelimb and one hindlimb control. The fluid was analyzed for the presence of TA.

Triamcinolone acetonide was found in all navicular bursas sampled, including the hindlimb control bursas. However, the levels of corticosteroid in the forelimb navicular bursas were significantly higher than the hindlimb controls. These results indicate that while some of the drug was absorbed into the bloodstream and able to move into remote (non-injected) joints, the majority of the corticosteroid diffusion was local. There was no difference in TA levels between forelimbs showing that addition of HA did not alter diffusion of the corticosteroid.

In the second part, 11 additional horses with clinical and radiographic signs of navicular syndrome were identified. The possibility existed that changes in the foot (thickened joint lining, scar tissue, etc) would prevent movement of corticosteroid in the diseased horses. Both front coffin joints were injected with TA and the same fluid samples were taken as in the first study.. Foot radiographs were also scored for severity of navicular changes.

Triamcinolone acetonide was found to move from the coffin joint into the navicular bursa in all of the horses with navicular syndrome. The severity of radiographic changes was not significantly associated with amount of TA found in the front limb bursal fluid samples.

Unfortunately, no studies have been performed that show what level of corticosteroid is necessary to control pain in the equine joint or bursa. Levels of TA were high

enough to suggest this type of treatment is likely to work. This study has demonstrated that injection of the coffin joint with TA appears to be an effective way to deliver corticosteroids to the navicular bursa in horses clinically affected by navicular syndrome.

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Figure 1. Needle being placed to obtain bursal fluid



Figure 2. Radiograph confirming needle placement

