

DSLD'S DEVASTATION

DSLD CAN STRIKE A HORSE AT ANY AGE, FROM BEING PRESENT AT BIRTH TO NOT DEVELOPING UNTIL LATE IN LIFE. AND IT'S NOW CONSIDERED A SERIOUS, BODY-WIDE PROBLEM.

Degenerative suspensory ligament desmitis (DSLD) is a progressive and devastating lameness that's gaining attention.

In this condition, there is failure of normal healing, with the ligament in involved legs becoming progressively thicker and more spongy. Once thought to be a problem only of the suspensories, recent research has discovered this is actually a bodywide problem.

What Is It?

In the simplest sense, DSLD is a failure of tendons and ligaments to maintain, remodel and repair themselves in a normal fashion. Over time, the normal rope-like structure of these tissues becomes distorted by accumulation of a large amount of proteoglycans, molecules of proteins linked to sugars. The blood supply to the tendons and ligaments progressively shrinks. The exact cause for this failure and accumulation of proteoglycans has not yet been determined. However, DSLD has been proven to be a systemic disease, meaning it's a body-wide problem.

Onset is often in early adulthood. The earliest symptoms are often vague and nonspecific, such as unexplained stumbling and vague lameness that may shift from leg to leg (see our table for symptoms of ligament/tendon injury vs. DSLD).

Stiffness, especially when rising, is common. It's not unusual for a DSLD horse to have a long history of lameness and neurological exams and to be misdiagnosed in these early stages. The horse may have asymptomatic periods interspersed with times when symptoms are again obvious. As the disease progresses, changes in the fetlocks become obvious. It may strike the front legs first or the hind legs, may be worse in one leg but always involves more than one.

The first change is often swelling, followed by obvious thickening of the suspensory, usually in the suspensory branches. The joint pouches of the fetlock joint may be enlarged. Heat may be obvious. Palpation of the suspensories elicits a pain response and ankle flexion tests are positive, often extremely so and disproportionate to the amount of lameness the horse may be showing.

At this stage, there's still a pattern of "flares" of symptoms alternating with relatively good periods but over time the suspensories become progressively enlarged, painful and develop a mushy consistency on palpation when the leg is held up but may feel abnormally tight when it is bearing weight.

Conformation changes occur, with the fetlocks either dropping down and the horse becoming coon footed, or the opposite may occur, with the fetlocks becoming very upright. When a hind leg is involved, the whole limb may become post legged. Eventually, the condition progresses to the point that the horse is constantly in pain, may even go down and refuse to rise. Euthanasia is often the only option for these horses.

Gait Changes

The gait changes with DSLD. Common abnormalities include toe first landing, development of a “rope walking” like gait, traveling extremely wide behind when the hinds are involved, and hopping like a rabbit at the canter when the hinds are involved. These gait changes sometimes lead to a misdiagnosis of neurological disease. To relieve the strain on their ankles, DSLD horses often dig holes to stand in toe first. When the hind legs are involved, they may sit on fence lines or rocks to rest their legs and may be observed dog sitting.



In the early stages, behavior changes, especially reluctance to work, are common. DSLD horses often have episodes that look like colic/abdominal pain but nothing is found to explain it on examination. Areas other than the fetlocks, such as the back, patellar ligaments of the stifle, and hip joints are commonly painful.

As the disease progresses, many horses show rapid premature aging, with a gaunt head, loss of topline and hind end musculature, abdominal wall weakness and sagging.

In the later stages, many horses seem to age rapidly, with muscle wasting. Extremely stretchable skin, that may even hang loosely in wrinkles, develops in some cases. Some horses develop hard, boxy swellings along the sides and back of their hocks. Flexor tendons may slip out of position at the point of the hock. Many owners report their DSLD horses develop allergies for the first time in their life, and mares frequently abort. Whether these last two are directly linked to the disorder, or secondary problems caused by stress, has not been determined.

Nature vs. Nurture

There are those who believe that DSLD is actually a result of external factors, such as poor shoeing, poor conformation, terrain, even the unique leg strains caused by certain gaits. While it's true that all of those things can contribute significantly to the burden placed on the supporting ligaments and tendons of the upper legs, the most recent finding that the microscopic abnormalities can also be identified in places like the eye and blood vessels clearly shows this is truly a body-wide problem.

Anatomy of Normal Tendon And Ligament

A normal tendon or ligament is composed of long fibers of collagen, a very dense type of connective tissue (like “gristle” in a steak). These fibers are entwined with each other, like the strands of a rope. On dissected specimens, the tendons and ligaments are smooth, white and glistening. There



DSLD is now being recognized as a body-wide, systemic disease.

is little, if any, proteoglycan in the background, either visibly or on microscopic views. With DSLD, there are scattered islands of collagen fibers but large amounts of a gelatinous fluid that is the increased ground substance. Islands of cartilage or bone may be found.

Degenerative Tendinopathies And Desmopathies in People

There is a syndrome in humans involving failure of tendons or ligaments to heal that is usually believed to be triggered by repeated use or overuse of the involved area. The Achilles tendon is frequently involved, but this problem may develop virtually anywhere. The microscopic changes seen bear a striking resemblance to DSLD, including the accumulation of large amounts of ground substance/proteoglycans. As with DSLD, the cause for this failure to heal is unknown, but at least one study believes that it probably does have a genetic predisposition, possibly unmasked by an injury. It may well be that DSLD in some individuals, or breeds, fits this pattern as well.

Dr. Jeannette Mero has been studying DSLD for a number of years, and in 2000 founded the nonprofit organization DSLD Research Inc.

Careful and thorough postmortem examinations performed by Dr. Jaroslava Halper, from the Veterinary School at the University of Georgia, found that the same abnormalities seen in suspensories from DSLD horses can also be found in the flexor tendons, patellar tendons of the stifle, the nuchal ligament in the neck, arteries and the sclera (“whites”) of the eyes. This study confirms that DSLD is a systemic, body-wide disease, not a local problem with the suspensory ligaments. Dr. Halper also had the opportunity to study a skin biopsy from a DSLD horse with the loose skin symptom and reported there was almost complete absence of elastic fibers in the skin.

Diagnosis

Until recently, the only way to definitively diagnose DSLD was by postmortem examination of the legs. However, with the finding that the nuchal ligament shows the same changes as the legs, nuchal ligament biopsy has emerged as a possible definitive test that can be done on the live horse. Anyone interested in information on this test should contact Dr. Halper by calling 706-542-5830 or contact her by e-mail through her web page at

<http://www.vet.uga.edu/vpp/halper/halper.html>.

Otherwise, a presumptive diagnosis is made by history combined with physical examination, response to flexion tests and ultrasound appearance of the suspensory ligaments. One odd characteristic of DSLD is that the ligaments will begin to enlarge before they show ultrasound evidence of “holes” and disrupted fiber patterns that most vets will be looking for to confirm a diseased tendon or ligament. For this reason, measurement of the size/thickness of the suspensory body and suspensory branches is also important.

Dr. Mero has put together an examination and ultrasound protocol for DSLD, with detailed guidelines related to where along its length the suspensory should be measured, in what planes to measure and suggested cut offs for normal suspensories.

The hallmark of DSLD compared to other tendon/ligament problems is the progressive enlargement of the ligament over time. Dr. Mero’s protocols and examination forms can be found at www.dsld.org, as well as sample ultrasound images from DSLD horses and some photos of postmortem specimens.

PROGNOSIS, TREATMENT

The long-term prognosis for a horse with DSLD is extremely poor. Although there can be long periods where the degeneration seems to stabilize and the horse is reasonably comfortable, this is a progressive condition that will usually end up with euthanasia. While opinions differ on riding DSLD horses during their relatively asymptomatic periods, a rider’s weight is extra burden on the weak legs. Mares should not be bred, both

because of the likely genetic component and because the weight of the pregnant uterus is a severe strain on them.

Some specialized shoeing techniques have been developed and been successful in stabilizing the disease for prolonged periods of time in some non-Peruvian horses but do not work well for the Peruvians horses.

Most owners are having the best results with keeping their horses barefoot, with frequent attention to keeping the feet meticulously balanced and the toes well backed up. These horses also typically do best when kept on as much turnout as possible.

Some people find sports boots or support wraps help their horses be more comfortable. NSAIDs are typically used when horses are having a flare, as well as the usual intensive cooling/icing and wrapping to control swelling. Some horses are maintained on MSM and/or devil's claw.

Preliminary results with a new treatment are showing promise. Research in people with degenerative ligament and tendon injuries found excellent pain relief and improved clinical outcomes when they were treated with a topical nitric oxide, either nitroglycerin or isosorbide compounds.

The healing process in normal tendons is highly dependent on production of nitric oxide at all stages of healing and absence of nitric oxide leads to exaggerated inflammatory responses.

Because of this, a trial is underway following the response of DSLD horses to supplementation with Jiaogulan and a nutritional supplement that supplies the raw nutrients needed for the body to manufacture nitric oxide. So far, reports have been positive, with reductions in pain, sometimes quite dramatic, being seen in the same time frame as in laminitic horses treated with Jiaogulan, within one to three days.

Genetics Issue?
DSLD has been found in a wide variety of breeds, but it's particularly common in the Peruvian Paso. In non-Paso breeds, the disease usually develops after the age of 15. With the recent finding that DSLD is a body-wide disorder, and many reports from owners that the problem has run in family lines, there is an extremely high index of suspicion that the disease is genetic. In fact, Dr. Gus Cothran is hot on the trail of genes that may be linked with DSLD and is focusing on areas where genetic changes have been described in association with human diseases that bear a strong resemblance to equine DSLD, such as the Ehlers-Danlos syndrome.
Ehlers-Danlos is actually a constellation of several different types of connective tissue disorders, with different genetic changes, so pinning this down could take some time if the situation in horses is similar. There are many clinical similarities between E-D and DSLD. In addition to the abnormal ligaments and tendons, human patients may have changes in their intestinal-tract walls, which leads to bouts of abdominal pain, eye changes, arterial changes and odd progeria (premature aging) or very loose skin. The hope is that the genetic work will eventually lead to a diagnostic test for DSLD. For the moment, most involved with this disease feel very strongly that affected horses should not be bred.