Selenium deficiency masks itself as different diseases.

By Kristin M. Patton, D.V.M.

LIKE A MASKED MARAUDER WAITING TO DECIMATE YOUR HERD, selenium deficiency can hide, disguising itself as different diseases.

But knowledge and prevention are your keys to stopping the hidden problem before it starts.

What is Selenium?

Selenium is part of the process that protects cell membranes, including muscle cell membranes, from damage due to free radicals. It can be found in plant and animal tissues, and is important for normal immune function, fertility, and muscle health.

How Much?

Talk to your veterinarian about local selenium levels. In general, though, mares should get 1 milligram per head per day or 4.4 to 6 milligrams per head per week in the form of a trace mineral containing 15 to 30 ppm of selenium.
to peroxides formed during normal cell functions. Additionally, selenoproteins are directly involved in the biosynthesis of DNA and thyroid regulation. Newer studies have shown that selenium is important in cancer prevention and in the treatment of AIDS and cancer patients.

Since muscle cells are highly active, selenium deficiency usually shows up as muscle degeneration.

Genetic diseases such as HYPP, glycogen branching enzyme deficiency, exercise-induced rhabdomyolysis and equine polysaccharide storage myopathy can be exacerbated if the muscles aren’t getting enough selenium. Horses that aren’t getting enough selenium can develop necrosis of any muscles, including the heart and diaphragm, which can lead to respiratory or heart failure and death.

With all the essential roles selenium plays in the body, it would be easy to oversupplement selenium on the theory that if some is good, more would be better. But in the case of selenium, too much can be as bad as not enough. Too much selenium can lead to severe cell injury resulting in malformed hooves, poor thrift and even death.

**White Muscle Disease**

*White muscle disease is the result of a selenium deficiency that occurs in foals from birth to 11 months of age, with most cases showing up within the first two months of life. The disease shows up in one of two ways: Either foals die within hours or days from massive cardiovascular collapse or they show marked muscle weakness and are sometimes unable to eat.*

Under a microscope, the muscle degeneration shows up as pale, tan-white-gray streaks of chalky, gritty muscle. Skeletal muscles and heart muscles can be affected, unlike many other muscle diseases in horses that do not affect the heart.

Foals with the acute form of the disease, called acute fulminating white muscle disease, suddenly die or have an increased heart rate, difficulty breathing and a moist cough. Foam might come from the nostrils. These foals progress to being down and unable to rise with repeated thrashing and struggling that can be misinterpreted as signs of colic.

Foals that have the subacute form of the disease often have secondary diseases, such as aspiration pneumonia, that mask the selenium deficiency. The foal often will not be able to rise without assistance, or if it does, it will be extremely weak and stiff. Because the foal’s neck hurts, it may carry the neck oddly, which is called wry neck, and touching it may cause more pain. The large muscle groups of the legs and back can also be painful if touched.

Veterinarians use blood tests to check selenium levels, and a muscle biopsy might be performed to confirm a diagnosis. Radiographs can help in evaluating foals’ lungs for aspiration pneumonia and urinalysis can help determine whether the kidneys are excreting muscle pigments.

**Masseter Myopathy**

*Adult horses can show acute or subacute white muscle disease, similar to foals. Usually the disease shows up in certain muscle groups in adult horses, such as the cheek muscle – the masseter muscle – and the muscles of the neck. Wry neck is common.*

The masseter muscle, or cheek muscle, closes the jaw during eating. Horses with masseter myopathy can have difficulty eating or opening their mouths. They can show weight loss, have difficulty moving or atrophy of the muscle.

Masseter myopathy appears similar to tetanus, tempromandibular joint disease, fractured jaws, eclampsia and some neurological diseases.

As in foals, adult horses with masseter myopathy will have elevated muscle enzymes and will show the same changes in a muscle biopsy.

**Prevention and Treatment**

Selenium deficiency treatment can be unrewarding if the animal is already affected with the disease. Intramuscular injections of selenium and oral supplements are the usual treatment course.

Prevention is paramount for the entire herd, especially in areas that are known to be selenium-deficient. You need to have your herd levels checked by measuring whole blood selenium levels and glutathione peroxidase activity.

If you aren’t sure whether your area is selenium-deficient, you can ask your veterinarian or county Extension agent.
Selenium toxicity can occur with oversupplementation of selenium or when horses graze plants in areas with high selenium content.

The areas known as the “poison belt” that have high soil selenium include Wyoming and South Dakota in the United States and areas of western Canada.

Two disease syndromes are seen in horses that eat high selenium forage, a sudden form and a longer lasting form. The sudden or acute form of toxicity causes intestinal signs or heart failure leading to death in almost all of the horses affected. The longer lasting or chronic form causes weight loss, rough hair coat and loss of hair, especially loss of mane and tail hair, and hoof problems. The coronary band might separate and the hoof might fall off or grooves might form in the hoof wall that are parallel to the coronary band.

Selenium toxicosis is diagnosed by testing for high levels of selenium in the blood or hair as well as evaluating feed selenium levels.

To learn more about the dangers of oversupplementation, see “A Matter of Moderation” in the April 2006 Journal.

Too much selenium is as bad as too little. It can lead to hoof sloughing, among other problems.

Kristin M. Patton, D.V.M., Ph.D., DACVP, is an assistant professor at Oregon State University in the college of veterinary medicine and a special contributor to The American Quarter Horse Journal. To comment, write to aqhajrn1@aqha.org.

What about me?

If you aren’t sure whether your area is deficient in selenium, you can consult a soil map or talk to your veterinarian or county Extension agent.

In the United States, the Pacific Northwest, the Atlantic states and the upper Midwest are known to have areas of deficiency. If you own cattle, sheep or goats, those animals also need supplementation, and if you eat only locally grown foods, you’ll want to consider talking to your doctor about adding selenium as well.