

Selenium Toxicity in the Western United States' Pork Industry



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Over the past year, selenium toxicity was identified in several swine and some cattle herds in the Western United States. Extension agents, local producers and State Veterinarians worked closely with Don Mahan of The Ohio State University to identify the problem and help producers in their efforts to reverse it through diet modifications.

Mahan believes undiagnosed selenium toxicity may have been plaguing pork producers across the Western states for a few years. Producers that have struggled with animal health, reproductive and lameness issues that do not seem to respond to conventional treatments should discuss the possibility of selenium toxicity with their herd veterinarian. Producers also should enlist a qualified nutritionist to ensure that rations are not high in selenium, and balanced for all other nutrients.

The following question and answer section is useful for producers to understand selenium toxicity, how to identify it as a problem in their herd and how to address it.

WHAT ARE THE MAJOR SYMPTOMS OF SELENIUM TOXICITY?

The first sign normally is the loss of hair and cracking of the hoofs, particularly at the coronary band (junction of hoof and foot). This may be followed by reduced feed intake and growth rate of pigs. Upon continued feeding of a high-selenium diet, the pigs will walk erratically (swaggering). Sows fed such diets for a long time period will experience fewer and reduced estrus and many will not conceive. Sows will show evidence of reduced feed intake, lower litter size, increased mummies and stillbirths and lack of milk production. Some of the pigs born from such sows will be deformed, particularly in the cartilage forming tissue, and become hairless while nursing the sows. Although these reproductive signs also may be indicative of other diseases, further examination of these signs is warranted. Selenium toxicity may be acute or chronic.

TOXICITY?

Acute selenium toxicity occurs rapidly upon the injection or feeding of a high level of selenium (inorganic or organic) to animals. Acute dietary selenium toxicity is generally considered to start at approximately 5.0 ppm (5mg /kg or 2.2 mg/lb – equivalent to 5 grams per ton) in a diet. Toxicity could occur within three and six weeks after feeding such a diet. Normally, the pig rids itself of excess selenium through the urine and feces, but once these avenues are overwhelmed, selenium builds up in the tissue causing tissue damage and toxicity. Occasionally, a foul smell (similar to rotten eggs) is noted in the breath of animals which have been given excess selenium.

Chronic selenium toxicity results when the amount of selenium consumed per day by the animal is lower, but has been consumed over a longer period of time and produces the same signs as the acute form of toxicity. Consequently, the time to achieve a toxic level in the pig is much longer, making an early diagnosis and intervention more difficult. It is most probable that the cases recently reported in the West were caused by chronic selenium toxicity.

HOW DID EXCESS SELENIUM ENTER THE ANIMALS' DIETS?

It is believed that selenium entered the pigs' diets through feedstuffs of plant origin, including grains and grasses. Plants do not require selenium for growth and development, the amount of selenium in a plant is in direct relationship to the amount of selenium in the soil. Selenium is retained when the plant when selenium replaces sulfur in an amino acid for selenium or in plant tissue that normally contains sulfur.

Normally, sulfur is used by the plant in the synthesis of the amino acid methionine. When the plant uptakes selenium from soil, it binds it to methionine, resulting in a modified amino acid called selenomethionine. The amount of selenomethionine produced by the plant depends on the relative ratio of sulfur to selenium in the soil and the rate of absorption of selenium by the plant. Thus, in selenium-rich soils, high protein seeds would contain more selenium. The animal cannot synthesize methionine or seleniomethionine and depends on its diet for its supply and for tissue synthesis. The animal can use either form for normal protein synthesis.

WHAT IS UNIQUE ABOUT THE WESTERN USA?

Soils do not have the same selenium content. Some areas in the western region of the Midwest have soils with high selenium contents. Consequently, the grains and forages that are grown in these regions may have high selenium content. In contrast, states such as Ohio, Minnesota and Indiana have lower soil selenium contents. Grain-producing areas along the east coast (such as in Florida and North Carolina) and along the west coast (such as in Oregon and Washington) with high levels of rainfall and sandy, well-drained soils do not retain many minerals and consequently, soil selenium is low or deficient. In addition, soils that are alkaline, dry and with a high pH are more likely to have selenium of a higher bioavailability for plant absorption.

WHAT IS UNIQUE ABOUT THE CURRENT CASES OF SELENIUM TOXICITY?

The analysis of feed ingredients in diets consumed by affected pigs in Western states confirmed the presence of high levels of selenium. Animal tissue assays also confirmed high selenium in affected pigs.

Mahan and others hypothesize that the high price of feedstuffs has stimulated production of feed stocks from relatively dry lands in the West that had historically been idle or less used. This land may have had a buildup of selenium in its soil reserve and the resulting grains and products harvested contributed more selenium than that that can be tolerated by animals, particularly when fed for a prolonged time period. Because it is currently cheaper to transport grain from the Western United States and the Western Corn Belt than from the Eastern Corn Belt, much of the grain fed to those animals could have originated from previously land where selenium levels are higher than normal. Producers should always test their grain supply, purchased or grown on farm, for its selenium content.

ARE PIGS EQUALLY AFFECTED?

As indicated previously, the chronic form of the selenium problem takes a longer time to develop. In fact, a recently completed regional trial using grains and protein sources grown in several areas of the United States including South Dakota and Nebraska produced no toxicity levels when fed to pigs from 50 to 250 lbs. of body weight. The diets were found to contain approximately 0.80 ppm selenium. Therefore, feeding diets with less than that amount of selenium to grower/finisher pigs should not produce toxicity problems in market pigs and in fact have been fed to swine raised in that area for many years.

In contrast to market pigs, high dietary levels of selenium would be expected to have more of an adverse affect on older animals including breeding females. For example, grower pigs fed a high dietary selenium level which produces no toxicity issues from 50 to 250 pounds will exhibit indigenous blood selenium levels that are higher than normal (more than 0.4 ppm in plasma). These pigs might develop chronic toxicity if retained in the breeding herd and continued on a high -selenium diet. Consequently, the development and degree of selenium toxicity depends upon the original dietary selenium content fed, the resulting selenium blood plasma status of the pig entering the breeding herd, the levels of dietary selenium fed during gilt development, gestation and lactation diets.

In breeding females, for example, selenium toxicity could be present in both the adult sow and her progeny because organic selenium (found in the grains and in some premixes) is readily transferred to the fetus and also to mammary tissue and thus into to the milk supply.

Note that lactation diets often contain higher levels of protein in the form of soybean meal that can potentially have higher selenium levels. The other feedstuffs that are added to a diet may also influence the development of toxic selenium levels. For example, alfalfa meal that is sometimes fed to breeding animals may contribute more selenium to the diet if the feedstuff is grown in Western states.

Generally the water supply does not contain excess selenium but if pigs are allowed access to waste water or water from a mining area it could contain a supply of selenium that could produce toxic effects since urine is a major route of excretion of selenium for the pig.

ARE ANIMALS WITH SELENIUM TOXICITY EXPECTED TO RECOVER?

Adult hogs showing advanced signs of toxicity (weakness, lack of strength, lameness, emaciation) generally will not recover. However, hogs with minor chronic signs, may respond to diets having reduced levels of selenium and it may be possible to overcome the selenium toxicity.

HOW CAN SELENIUM TOXICITY BE CONFIRMED?

Producers should test all potential feeds and feed ingredients for selenium levels and enlist the assistance of a qualified nutritionist to ensure the diet is balanced for all nutrients. Producers also should work with their herd veterinarian to discuss the possibility and identify the signs of toxicity early on, as well as all other contributing factors.

HOW CAN SELENIUM TOXICITY BE RESOLVED ON YOUR FARM?

Selenium toxicity can be addressed through the pig's diet. Rations should be monitored for selenium content and those with high selenium levels should not be used, particularly for breeding stock.

This report was developed by Don Mahan, Ph.D., The Ohio State University on request from the Pork Checkoff.



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