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Selenium

The Jekyll and Hyde of the
Livestock and Poultry Industry



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Selenium has a paradoxical position in livestock and poultry production, since it is recognized both as a toxicant and as an essential micro-nutrient. Selenium toxicity to livestock and poultry was recognized during the time of Marco Polo. In 1858 it was described in military horses at Fort Randal, Nebraska Territory. Interest in the role of selenium in agriculture was rekindled during the past decade with the discovery that selenium additions to the diets of animals in certain areas stimulated production.

Toxic amounts of selenium are obtained from selenium-containing forages and grains, overdosage in feeds or medication, water or industrial sources such as the sulfide ores of copper and silver mining. Soil selenium is not a good indicator of the likelihood of selenium toxicity in livestock. Plant analysis is a much more satisfactory guide.

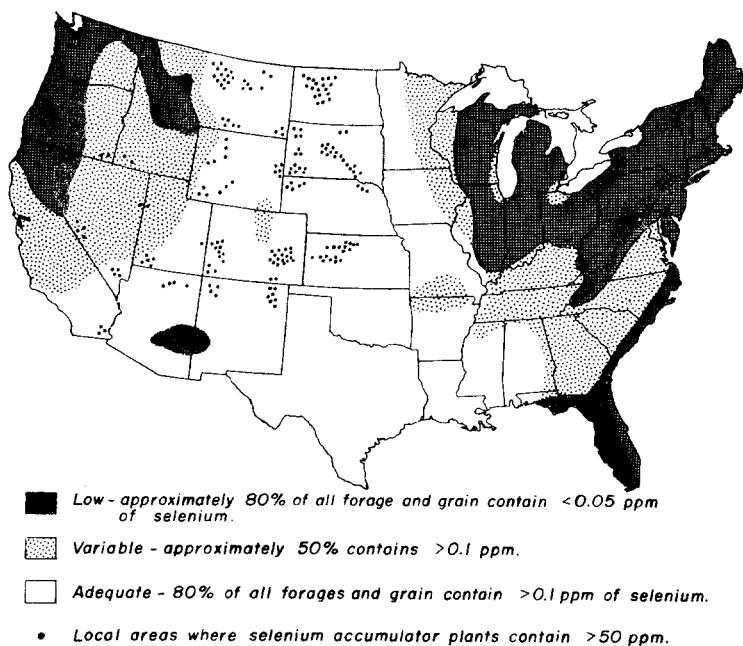
The chemistry of selenium in soils is not completely understood. Selenates, selenites, elemental

selenium and organic selenium have been reported to occur in soils. Selenates and to a lesser extent organic selenium are the selenium forms most available to plants.

Selenium toxicity in livestock in the United States is most commonly found when animals graze plants containing more than approximately 4 to 5 ppm selenium. These plants are most commonly found growing on neutral to alkaline soils derived from sedimentary rocks of Permian and Cretaceous age high in selenium. Such soils are called seleniferous soils. Plants growing on acid soils containing high levels of selenium are much less likely to produce selenium toxicity. In acid soils, the availability of selenium is reduced by the formation of insoluble compounds or complexes of ferric iron and selenite.

The geographic distribution of low, variable and adequate selenium areas in the USA are illustrated in Figure 1. This map was prepared by the staff of the

FIGURE 1



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U. S. Plant, Soil and Nutrition Laboratory, Ithaca, New York. Although a great deal of work was expended in preparing this map, there is a need for more refined sampling within the broad geographical areas of "low" and "adequate" selenium. For instance, North Dakota is listed as a zone in which selenium is "adequate". However, selenium deficiency has been reported in sheep in certain areas of this state.

Selenium Accumulation in Plants

Plants vary greatly in their ability to absorb selenium. Differences in accumulation are most pronounced when plants are grown in media high in available selenium. Plants which absorb large amounts of selenium when grown on seleniferous soils, and which usually are only found on such soils, are referred to as selenium-accumulator or indicator species. Plants which are listed as indicator species include: prince plume (Stanleya)*, milk vetch (Astragalus)*, golden weed (Oenopsis),** and woody aster (Xylorhiza)**.

Other plants also accumulate selenium, but their geographical distribution is not restricted to seleniferous soils. These plants include: the broom snake weed (Gutierrezia)* or Xanthocephalum, stick leaf (Mentzellia)*, gum weed (Grindelia)*, Indian paintbrush (Castilleja)*, salt brush (Atriplex)*, iron weed (Sideranthus)* or (Aplopappus), hoary aster (Machaeranthera* or Aster). Also included in this group would be the sunflower, common cereals, pigweed and turpentine weed.

Some researchers previously considered that selenium was an essential element for at least some of the selenium-indicator plants. However, essentiality of selenium for plant growth has not been definitely established. In one recent study, selenium stimulated the growth of Astragalus species by ameliorating a toxicity of another element. If selenium is essential for plants it is required in very minute amounts.

Indicator plants are usually not palatable to livestock because of an offensive odor caused by volatile organic selenium compounds produced by the plants. Animals that are introduced into a geographical area and are not acquainted with native seleniferous plants may initially consume them. When available grazing is inadequate, animals may be forced or induced into consuming the seleniferous plants.

The retention of selenium by host animals is dependent upon the body reserve, concentration in feeds, and form of selenium. Approximately 25 to 75 per cent of selenium consumed from natural feeds

* Found in North Dakota.

** Found in states adjacent to North Dakota.

is retained. Selenium is present in milk, its level correlating with the concentration in feed. Consumption of more than physiological quantities of selenium will result in excretion via the urine, bile, pancreatic fluid, expired air, milk and feces.

Unlike the marked differences which occur when various plant species are grown on seleniferous soils, only relatively small differences in selenium accumulation are found when the same species are compared on soils with low contents of available selenium. Changes in growth rates or in species, however, may influence the incidence of selenium deficiency in livestock.

Signs of Selenium Toxicity (Bob-tailed disease, alkali disease, blind staggers)

Selenium toxicity is a geographical disease usually occurring in areas of 20 inches or less of rainfall, shale type formations and volcanic deposits. Local distribution may be quite variable with concentrations in washes or depressions that are known as poison traps. Irrigation may influence the area of concentration.

The acute form of selenium toxicity may occur in all domestic animals but primarily is observed in cattle, horses and sheep and is often incorrectly referred to as blind staggers. The usual source is indicator plants. The signs observed are described as occurring in three stages.

During stage one the involved animal may stray from the herd or flock and wander aimlessly about. When confronted with objects in its path of movement the animal ignores them or stumbles over them, due to impaired vision. Circling may be observed and temperature and respiration may be normal.

As the condition progresses (second stage) the signs become more pronounced, with the front legs becoming weak and most body weight being placed on the hind legs. The neck may be placed on a solid object for additional support. Paralysis appears suddenly and the throat and tongue may be involved. Profuse salivation with the signs of colic such as grunting, grating of the teeth and bloating will appear.

Signs of the third stage include blindness, labored respiration, emaciation, sub-normal temperature, collapse and death.

Selenium toxicity may also appear in the chronic or sub-acute form, which is frequently referred to as bob-tailed disease or incorrectly as alkali disease. The signs observed are due to consumption of moderate quantities of selenium over several weeks or months.

In cattle the signs include ragged, cracked and sloughed hoofs due to interference of hoof growth at the coronary band. Similar lesions may be observed on the horns. Lameness, because of bone erosion at the joints, results in walking on the knees while grazing and is an additional sign observed. The hair coat becomes rough and there is often a loss of hair from the switch of the tail. Five to 10 ppm of selenium may be found in horns, hoofs or hair. Emaciation is also often observed.

Involved horses may lose the hair from the tail and/or mane (bob-tailed disease) and may also exhibit deformed hoofs. Approximately 5 to 45 ppm of selenium can be detected in the hoofs and hair of involved animals.

In sheep, hoof lesions, emaciation, anemia, loss of wool, and decreased appetite are common signs. Bone deformities, blindness and fetal abnormalities have been reported in lambs. The signs are often intensified by consumption of weeds because of drought. Toxic levels of selenium are 5 ppm or greater in the ration.

Erosion of the joints of the long bones accompanied by lameness, hoof cracks, and deformities, loss of body hair and emaciation are signs of toxicity in swine.

Animals may be born with or develop the signs of selenium toxicosis in early life, as selenium passes the placental barrier and will also be in the milk of the nursing young.

Deformed chick embryos accompanied by low hatchability are common signs of selenium toxicity in poultry. Fertility is not usually adversely affected. Egg production and feed efficiency are adversely affected.

Selenium Deficiency Is Becoming a More Frequent Problem

Selenium deficiency in livestock and poultry has become a more frequent problem in recent years. It is not unusual to find areas within a distance of 25 to 50 miles where toxicity signs are observed in one location while selenium deficiency signs are observed in the other. Deficiency areas in the United States include the Pacific Northwest, including western Montana, the northeastern one fourth of the United States and central and southern Florida. There is some transportation of selenium via feeds from high concentration areas to deficiency areas.

Selenium is an essential element to all domestic animals and poultry for the normal functions of the

heart, muscles, liver, kidney and pancreas. Deficiency signs are closely aligned to those of vitamin E deficiency.

White muscle disease (WMD) is the most frequent recognized sign of selenium deficiency in domestic livestock. There is a close relationship to vitamin E deficiency and WMD but at present it is not well defined.

In addition to white muscle disease, swine may exhibit the signs of mulberry heart disease and liver degeneration. Vomition and gastric ulceration have also been reported as signs of selenium deficiency. Sudden deaths with no previous signs of illness often are reported as in selenium deficiency areas.

In poultry the signs of selenium deficiency include unthrifty birds, weepy skin, and subcutaneous edema, accompanied by easy bruising and muscle hemorrhage (exudative diathesis).

Selenium deficiency has recently been associated with general conditions of unthriftiness, poor gains, and greater susceptibility to disease for all domestic animals and birds. In some cases selenium medication has been reported to correct such poorly defined conditions as ill thrift, infertility, and chronic scours, and has been reported to increase weight gains and wool yields.

Prevention

Toxicity due to selenium may be prevented by identification of the selenium source and prevention of its consumption by animals or birds. Providing adequate forage will aid in preventing the consumption of selenium accumulator plants. The feeding of arsenic or arsenic acid may aid in decreasing the toxic signs of selenium toxicity.

The selenium deficiency may be prevented by the intramuscular or oral administration of selenium. Selenium is usually administered in combination with vitamin E. White muscle disease in lambs has been successfully prevented by the intramuscular administration of one milligram selenium as sodium selenite with 68 I.U. of vitamin E as d-alpha-tocopheryl during the first week of life. The therapeutic dose is twice the prophylactic dose. Sodium selenite at 0.1 ppm in the ration will prevent WMD in lambs, or 10 milligrams given orally or subcutaneously to ewes one month before lambing will aid in its prevention. The subcutaneous administration of sodium selenite to weaned piglets at 0.06 milligrams per kg body weight will prevent WMD in swine.

Sodium selenate and/or sodium selenite may now be added to feeds to prevent deficiency signs.

breath and sweat are other signs. Not all signs may be observed simultaneously.

Selenium Can Be Toxic to Humans

Usually selenium toxicity in humans is due to industrial contacts such as dust and fumes. Some shampoos contain selenium and those we use may cause toxicity.

The toxic signs in humans include: pallor, nervousness, depression, gastro intestinal disturbances and dermatosis. A coated tongue and a garlic odor of the

Selenium Requirements and Toxicity in Livestock and Poultry - ppm of Ration

Species	Requirement	Non-toxic level	Toxic level
Cattle	0.10	2.0	8.0
Sheep	.010	----	10.0
Swine	0.10	2.5	7.0
Poultry	0.15	5.0	15.0

Adapted from Patrias, George: Feedstuffs, Vol. 41, P. 24, March, 1969.