SELENIUM: ITS ROLE IN LIVESTOCK HEALTH AND PRODUCTIVITY

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ABSTRACT

Selenium (Se) is an essential metalloid trace element that has a very narrow margin of safety between the toxic and deficient doses in animals and humans. This paper highlights about the beneficial and harmful effects of Se in livestock productivity and health besides presenting some information on its relation to Khari disease of buffaloes in Baitadi and Darchula districts and ways to mitigate toxic effects of Se in buffaloes.

Key words: Selenium, deficiency, toxicity, Khari disease

HISTORICAL BACKGROUND

Selenium (Se) is a naturally occurring metalloid element that is essential to human and animal health in trace amounts but is harmful in excess. Selenium was first identified in 1817 by the Swedish chemist, Jons Jakob Berzelius; however, selenium toxicity problems in livestock had been recorded for hundreds of years previously although the cause was unknown. Marco Polo reported a hoof disease in horses during his travels in China in the 13th century. Similar problems were noted in livestock in Colombia in 1560 and in South Dakota (USA) in the mid-19th century where the syndrome was called alkali disease (Fordyce, 2005). While Jons Jakob Berzelius was isolating selenium from red deposits in the lead chambers of a sulfuric acid plant, illness of the workers in the plant was attributed to exposure to high levels of selenium (Oldfield, 1999).

Selenium has chemical and physical properties intermediate between metals and non metals and is similar to those of sulfur, arsenic and tellurium, all of which are in Group VI of the periodic chart of the elements. Like sulfur, selenium can exist in the 2^-, 0, 4^+, and 6^+ oxidation states as selenide (Se^{2-}), selenium (Se^0), selenite (Se^{4+}) and selenate (Se^{6+}), respectively. Selenium behaves antagonistically with copper and sulfur in humans and animals inhibiting the uptake and function of these elements. The volatilization of Se from volcanoes, soil, sediments, the oceans, microorganisms, plants, animals and industrial activity all contribute to the Se content of the atmosphere. Selenium is a bioaccumulator which means that plants and animals retain the element in greater concentrations than are present in the environment and the element can be bioconcentrated by 200-6000 times. Arid environments with alkaline soils, in times of drought or where less irrigation water is predispose to high soil selenium levels and a greater uptake of Se by plants. Such conditions exist in Rajasthan and southern parts of the Haryana states of India where above normal soil selenium levels (Yadav *et al.*, 2005). In parts of China the Se content of early Cambrian aged rock range from 10-40ppm (mg/kg) (Kunli et al., 2004).

The selenium status of human populations, animals, and plants varies markedly around the world as a result of different geological conditions. High selenium concentrations are associated with some phosphatic rocks, organic rich black shales, coals, and sulfide mineralization, whereas most other rock types contain very low concentrations. Globally selenium deficient soils are far more widespread than are seleniferous ones. Animal health is affected by selenium deficiency or excess in the diet, the intake of selenium being dependent on the amount of selenium taken up by plants as bioavailable selenium (Fordyce, 2005).

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Selenium forms a vital constituent of the biologically important enzyme glutathione peroxidase (GSH-Px). This enzyme reduces peroxides in cells thus preventing oxidative injury to cells (Rotruck *et al.*, 1973). Because of this vital role, deficiency of selenium in animals may result in a wide variety of clinical signs. Selenium in the form of selenoproteins is critical in the formation of thyroid hormones and other endocrine systems. Adequate selenium levels have also been shown to be necessary for normal spermatogenesis (Kohrle *et al.*, 2005). In severe deficiency states, myodegeneration occurs resulting in cardiomyopathy, muscle weakness and death (Koller, 1986). This paper gives an overview of selenium related health disorders in livestock, precautions to be taken when submitting biological samples for diagnosis and ways to mitigate the problems associated with either selenium deficiency or toxicity.

SELENIUM TOXICITY IN ANIMALS

The toxic effects of selenium were first discovered in the 1930s when livestock ate certain plants of some wild vetches of the genus *Astragalus*, which accumulated toxic amounts of selenium from the soil (Moxon, 1937). The identification of selenium accumulator plant species (some *Astragalus species)* in the environment is an indication that grasses and other forages will also have accumulated selenium, and therefore is a risk to livestock grazing them. Soil and herbage levels of Se exceed 5 mg/kg and 3 mg/kg dry matter respectively in 90% of toxic fields (Roger *et al.*, 1990). Addition of too much selenium to feeds, improper uses of selenium containing injectable or feeding livestock forages or feed grown in soils rich in selenium can result in selenium poisoning (Hatch, 1982).

Tiwari *et al.* (2006) compared the acute toxicosis caused by organic selenium (selenomethionine) found in plants with that caused by the supplemental, inorganic form of selenium (sodium selenite) and reported that in an acute oral exposure, selenium from selenomethionine is twice as bioavailable, but can be slightly less toxic than sodium selenite. They also found that sodium selenite, but not selenomethionine administration resulted in decreased liver vitamin E concentration. Lambs receiving 2, 3, and 4 mg/kg body weight as sodium selenite and 4, 6, and 8 mg/kg body weight as selenium methionine had visible evidence of reduced feed intake, depression, reluctance to move, and tachypnea following minimal exercise. Major histopathological findings in animals of the high dose groups included multifocal myocardial necrosis and pulmonary alveolar vasculitis with pulmonary edema and hemorrhage. Some investigators (Casteignau *et al.*, 2006) reported on selenium toxicosis due to errors in dosage of selenium in swine feed that resulted in an initial episode of diarrhea followed by dermatological and neurological signs; the most obvious sign being marked hind limb paresis. Cutaneous lesions consisted of diffuse alopecia, multifocal skin necrosis and coronary band necrosis of the hooves. Central nervous system lesions comprised of a severe bilateral polio-encephalomalacia of the ventral horns. In general therefore, elemental Se is relatively non toxic whereas organic Se found in plants and grains is more toxic to livestock.

Several hundred deaths have been reported in sheep from acute/subacute selenium intoxication following grazing of seleniferous plants growing on reclaimed phosphate mines in southeastern Idaho (Fessler *et al.*, 2003). Natural selenium toxicosis was reported from seven states of the USA. Over supplementation with selenium was reported as a cause of toxicosis in 15 states (Edmondson, *et al.*, 1993). Chronic selenosis is often reported in India in winter season with the symptoms of hair loss, cracks on skin, hooves and horns, leading to elongation and sloughing of hooves, lameness, ataxia and recumbency (Gupta *et al.*, 1982). In an experimental study with buffalo calves, adverse effects appeared when the whole blood selenium concentrations increased above 2 μg/ml, with mortality occurring when blood levels exceeded 3.4 μg/ml (Deore *et al.*, 2005).

Circumstances in which selenium poisoning occur are quite variable. The Se levels of dairy cows are highest in the winter when indoor feeding of Se-rich concentrates is practiced in Norway (Ropstad *et al.*, 1988). Sugarcane foliage from seleniferous areas can accumulate high levels of selenium ranging from 7.9 to 67.5 mg/kg. These selenium levels were 6-14 times higher than those from non-seleniferous areas (Dhillon and Dillon, 1991). Researchers from India (Ghosh *et al.,* 1993) reported Selenium toxicities in grazing buffaloes of the sub-Himalayan areas of West Bengal showing gangrenous syndrome of the extremities, skin cracks or sloughing, detachment of hooves, emaciation and eventually recumbency and death.

Selenium poisoning should be generally suspected based upon a variety clinical signs including weight loss, poor growth rates, lameness, defective hoof growth, horizontal ridges or cracks in the hoof wall, hair loss, infertility and acute deaths especially when errors are made in mixing of selenium into animal feeds or overdosing injectable selenium products. A garlicky odor on the animals' breath may be detected.

Hematological changes in selenosis cases may include decreased fibrinogen levels and prothrombin activity, increased serum alkaline phosphatase, alanine aminotransferase (ALT), aspartate aminotranferase (AST), and succinic dehydrogenase and reduced glutathione levels. Serum or liver Se levels exceeding 2ppm is indicative of acute toxicity. In chronic cases hair analysis with >5 ppm selenium is confirmatory. Forage analysis in which the Se levels exceed 5ppm should be considered hazardous to livestock health. Selenium accumulator plants may contain as much as 15,000ppm of Se. Livestock grazing plants growing in soils containing in excess of 0.5ppm are at risk of developing selenosis (Rosenfeld, 1964). The Environmental Protection Agency (EPA) has set a chronic eco-toxicity threshold of 5 µg/L in water.

SELENIUM DEFICIENCY IN LIVESTOCK

Only after identifying the beneficial role of Se and vitamin E in preventing dietary hepatic necrosis and exudative diathesis in rats and chicks, was selenium's nutritional value recognized in 1957 (Mayland, 1994). Selenium is necessary for growth and fertility in animals, neutrophil and lymphocyte function, and antibody production. Clinical signs of Se deficiency include dietary hepatic apoptosis in rats and pigs; exudative diathesis, embryonic mortality, poor response to antigens, pancreatic fibrosis in birds and white muscle disease (nutritional muscular dystrophy) in ruminants and other species (Ammerman, 1975; Swecker, 1997). Clinical signs of selenium deficiencies in animals, birds and humans include reduced appetite, growth, production, and reproductive fertility, a general unthriftiness, and muscular weakness. Retained placenta is reported in selenium deficient cows, while 'mulberry heart' disease is noted in pigs. Selenium deficiency in animals is very common and widespread around the globe affecting much of South America, North America, Africa, Europe, Asia, Australia, and New Zealand (Fordyce, 2005). Survey of state veterinarians and state veterinary diagnostic laboratories revealed that selenium-deficiency diseases were diagnosed in 46 states and were reported to be an important livestock problem in regions of 37 states of the USA (Edmondson, *et al.*, 1993).

LABORATORY ANALYSIS OF SELENIUM

There have been few publications addressing the need of proper storage of samples intended for determination of selenium level (Olivas *et al.*, 1998 and Palacios and Lobinski, 2007). Organic selenium reportedly declined drastically in a matter of a month when stored at different temperatures and in different containers. Palacios and Lobinski (2007) reported that 75% of selenium was lost after 30 days of storage. Their results demonstrated the oxidative degradation of selenoproteins and glutathione peroxidase (GSHPx) during storage of serum. In the author's experience, hay samples containing relatively high levels of Se

and stored at room temperature with exposure to light, when tested 6 months later had no detectable toxic levels of Se (Khanal *et al.*, 2008).

Blood and liver samples are the samples of choice for ante mortem and postmortem diagnosis of both Se deficiency and toxicity (Tiwari *et al.*, 2006). A whole blood selenium concentration is a better and more sensitive indicator of selenium status than hair selenium or glutathione peroxidase activities (Deore *et al.*, 2003). Controversy exists today as to the most suitable methods of assessing selenium levels *in vivo*. Assays of glutathione peroxidase (GSHPx) appear to be one of the promising modes of assessing Se status (Brody, 1999). Liver is the most suitable tissue for this purpose. Red blood cells (RBC) selenium levels have been used for measuring intracellular selenium levels. RBC and tissue selenium levels reflect long term selenium status, while plasma and urinary selenium levels are sensitive indicators of the amount of selenium consumed in foods the previous day or two, and do not accurately reflect tissue levels. Whole blood or serum selenium status measured at the herd level provides best consistency (Waldner *et al.*, 1998).

MITIGATION MEASURES FOR SELENIUM DEFICIENCY AND TOXICITY

The foremost approach to preventing selenium related health disorders is determining the prevalence of selenium in the environment. This may require soil and forage analysis if such information has not been determined previously. Farmers can be trained to identify Se accumulator plants so that their animals can be moved to safer areas. In Se deficient areas supplementing selenium will be necessary. In areas where Se levels are high in the soils and forages, it is necessary to avoid pastures high in Se or adopt measures to counter the toxic effect of selenium.

In general, selenium deficiencies in animals are corrected by giving injections, dietary supplements, salt licks and drenches. While correcting selenium deficiencies, administration of organic selenium such as selenium methionine was found to result in higher tissue, serum, and whole blood selenium concentrations than by the administration of equivalent doses of selenite (Tiwari *et al.*, 2006). The level of dietary selenium needed to prevent deficiency depends on the vitamin E status and species of the host. Assuming normal vitamin E status of the animal, concentrations of 0.04 - 0.1 mg/kg (dry weight) of selenium in feedstuffs are generally adequate for most animals with a range of 0.15-0.20 mg/kg for poultry and 0.03-0.05 mg/k for ruminants and pigs (WHO, 1987). International standards for Se requirements for cattle are in the range of 0.1 to 0.18 mg/kg dry matter. However there is considerable variation with the recommended level for cattle in different parts of the world; Ireland recommends are 0.24 -0.48 mg/kg dry matter (Rogers 1990), while in the USA the maximum allowable supplementation of Se is 0.3 mg/kg. In Finland, where soil selenium levels are very low, sodium selenate fertilizers had been used to produce crops with adequate amounts of selenium

Although there are no specific treatments to correct selenium toxicities in animals, recognition of seleniferous plants, proper land management and selective grazing may help prevent selenosis. Animals having a blood selenium level >1.5 μg/ml is indicative of impending selenium toxicosis and such animals should receive corrective measures to alleviate Se toxicity (Deore *et al.*, 2002). Administration of reduced glutathione (GSH) intravenously at 5 mg/kg of BW reportedly arrested the toxic signs, prevented mortality and lowered glutathione peroxidase (GSH-Px) activity (Deore *et al.*, 2005). Se toxicity in buffaloes was reportedly treated successfully in India by Arora et al (1975) and Arora (1985), using a daily oral dose of a trace mineral mixture consisting of five sulfates (1 kg magnesium sulfate, 166 gm ferrous sulfate, 24 gm copper sulfate, 75 gm zinc sulfate and 15 gm cobalt sulfate). A daily dose of 30 gm of this Pentasulfates per adult animal was given until recovery was noted (21-50 days). Of 517 buffaloes treated, 430 (83%) were cured in

21-50 days. Feeding high protein diets and a balanced mineral mix that contains sulfur and copper can reportedly reduce selenium toxicity (Fessler *et al.*, 2003).

It has been reported that application of gypsum up to 1 ton/hectare reduced selenium content in sugarcane tops from 15.16 to 5.08 mg/kg in a field experiment (Dhillon and Dhillon, 1991). Of late, phyto-remediation using kenaf and canola plants has been tried to clean up soil and water contaminated with selenium (Wood, 2000). By doing so, water and soil can be detoxified and canola enriched moderately with selenium levels can be fed to livestock in deficient areas.

Crisman *et al* (1994) demonstrated that horses that spent more than 50% of their time on pasture had a significantly lower (p<0.001) selenium concentration (0.124 ± 0.002 µg/ml) as compared to horses that spent less than 50% of time on pasture (0.144 ± 0.005 µg/ml). This finding is similar to the finding of Khari disease (chronic selenosis) in water buffaloes of western Nepal where the incidence is higher in stall fed buffaloes. Affected water buffaloes are reportedly showing improvement after being allowed to graze on pasture (Khanal, *et al.*, 2006).

CONCLUSION

The majority of livestock producing areas of the world are subject to the multifaceted effects of selenium on livestock health and disease. Selenium deficiency is more of a problem geographically than is Se toxicity due to seleniferous soils. Khari disease syndrome in lactating buffaloes of Darchula and Baitadi, in which Selenium toxicity was implicated as one of the multiple causative factors, has been showing very promising result to Pentasulfates-owing to its antagonistic effect to toxic levels of Selenium. The geochemistry of livestock-producing areas should be well understood to mitigate selenium related disorders in animals.

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