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Swine Toxicity & Selenium in Grains

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SWINE TOXICITY & SELENIUM IN GRAINS

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I. USA Experiences with Selenium—Micronutrient & Poison

Selenium was identified as an essential micronutrient over 50 years ago but the poisonous effects of selenium on range livestock, known as “alkali disease” in the central USA have been described for over 80 years. There is speculation that the defeat of General Custer in the battle of the Little Big Horn in Montana in 1876 may have been caused by lameness in horses affected by selenium poisoning. An army surgeon in South Dakota reported selenium toxicity in horses due to grazing on seleniferous plants. High selenium soils are derived from Cretaceous shales in the central states, including the Dakotas, Montana, and Wyoming (Franke, 1934) (Eisler, 1985).

The soil is the ultimate source of selenium which is ultimately incorporated into plant tissue proteins consumed by animals. The uptake of selenium by plants does not necessarily correspond directly to the soil's total selenium content. The knowledge of high and low selenium soils provides a useful base for assessing potential areas of deficiency and toxicity. The availability of selenium for plant uptake depends upon the chemical forms of selenium including: selenides, elemental selenium, selenites, and selenates, in increasing order of bioavailability. Selenium is more bioavailable from alkaline soils from high selenium source materials. Selenium is less bioavailable from low selenium source parent materials with poor aeration, low pH, and soils are subjected to high rainfall.

Selenium is an essential mineral and is incorporated into enzymes which regulate normal body processes. Selenium and vitamin E are anti-oxidants which help prevent cellular peroxidation, controlling cell damage that may lead to cancer. Two specific human diseases have been associated with selenium deficiency. Keshan disease results in an enlarged heart and poor heart function. Kashin-Beck disease results in degeneration of the cartilage and bone joints. Recent findings have shown that severe selenium deficiency in vitamin E deficient hosts can increase the mutation rates of RNA viruses, suggesting that selenium deficiency may increase the risks of measles, influenza, hepatitis and acquired immune deficiency syndrome (Combs, 2007).

In early laboratory studies, it was found to prevent liver degeneration in rats. Later, selenium was found to prevent clinical diseases in chicks, calves, lambs, and small pigs. Selenium deficiencies in swine were being reported in some areas of the Midwest, mainly Indiana, Ohio, and Michigan. Typical symptoms of selenium deficiency include muscular dystrophy, pale muscles, small hemorrhages in heart muscle, and necrosis of the liver (Cromwell, 2008).

Selenium is routinely used as a livestock feed supplement in areas where selenium is naturally deficient in feeds and forages. In the USA, the selenium deficient soils tend to be derived from sandy areas in the southeast, sedimentary rocks in the northeast or from more recent volcanic ash in the Pacific Northwest (Figure 1) (Oldfields, 1995). Michigan, Indiana, Ohio and Kentucky seem to be the major hog producing states that are deficient in selenium (Cromwell, 2008).

Selenium is a component of the enzyme glutathione peroxidase which activates glutathione which detoxifies peroxides, and provides protection of cellular membranes against peroxide damage. A selenium enzyme has been shown to have a function in thyroid metabolism. The dietary requirement for selenium ranges from 0.3 ppm for weanling pigs to 0.15 ppm for finishing pigs and sows (National Research Council, 1998). Research has indicated that the organic form such as selenium enriched yeast is more biologically effective than inorganic forms such as sodium selenite in reproducing sows and boars, but both forms can produce glutathione peroxidase (Kim et al., 2001). Selenium in natural foods or organic forms has been shown to be less toxic than purified or formulated forms of selenium for rats as the minimum toxic levels were higher (1.4 ppm selenium) for organic sources of selenium (Eisler, 1995).

Other research addressing selenium nutrition, metabolism and toxicology are summarized in Table 1. Toxicity studies with sensitive rat species indicated that high protein diets made the rats less vulnerable to the toxic effects of selenium. The protective compounds in the linseed oil were two cyanogenic glycosides compounds that could be converted to cyanide. Cyanide protects the animal from selenosis by forming a compound with the selenium that can be readily excreted in the urine (Kephart, 1999). However, the protein source must be analyzed for selenium content, since selenium intake must be minimized after selenosis. Metal intake also affects selenium toxicity since insoluble complexes are formed with arsenic, copper, cadmium, and mercury, increasing excretion. International organizations are recognizing the importance of mercury and selenium ratios by proposing that both be monitored as bioindicators of ecosystem bioaccumulative risks.

The USA Center for Disease Control (CDC, 2005) has performed human biological monitoring since 1999, including monitoring for thirteen metals in blood and urine. The most recent biological monitoring of lead and mercury levels (2002) has indicated a decrease in metal bioaccumulation exposures, presumably due to decreasing emissions and effective environmental pollution controls. Unfortunately, background levels of selenium have not been evaluated. A study is recommended to biologically monitor selenium exposures and bioaccumulation in USA populations from dietary sources such as pork, beef, milk, grains, and inhalation exposures. Ecosystem impacts as a result of selenium waste emissions from coal fired power plants, oil refineries, metal recyclers, and phosphate mining projects, are well documented (Hamilton, 2003, Hagelstein, 2007).

Background concentrations of selenium in human blood range from 0.03 to 0.20 ppm. In human bone selenium concentrations range from 1.0 to 9.0 ppm and other human soft tissues contain about 0.10 ppm selenium. A study addressing the minimal blood selenium concentrations determined that symptoms of selenium deficiency occur if serum concentrations are less than 0.02 ppm and symptoms of selenium excess occur at serum concentrations greater than 0.11 ppm (Emsley, 2001). Other studies have reported high blood selenium concentrations, up to 0.90 ppm from subjects in Poland and Spain. Residents of South Dakota and Wyoming have reported blood selenium concentrations up to 0.40 ppm.

A 2005 study in Brazil, reported selenium hair concentrations of pregnant and non pregnant females of 0.61 and 2.46 ug selenium/gram of hair, respectively. Other studies report selenium hair concentrations ranging from 0.44 to 1.04 ug selenium/gram of hair and a reference range for hair analysis in children has been reported as 0.95 to 1.70 ug selenium/gram hair (Doctor's Data Inc. St. Charles, Illinois). Biological samples of selenium in toenails have reported values ranging from 0.17 to 1.7 ug selenium/gram of nail. Unfortunately, most of these biological monitoring studies did not report toxic effects or symptoms of selenosis in the individuals sampled.

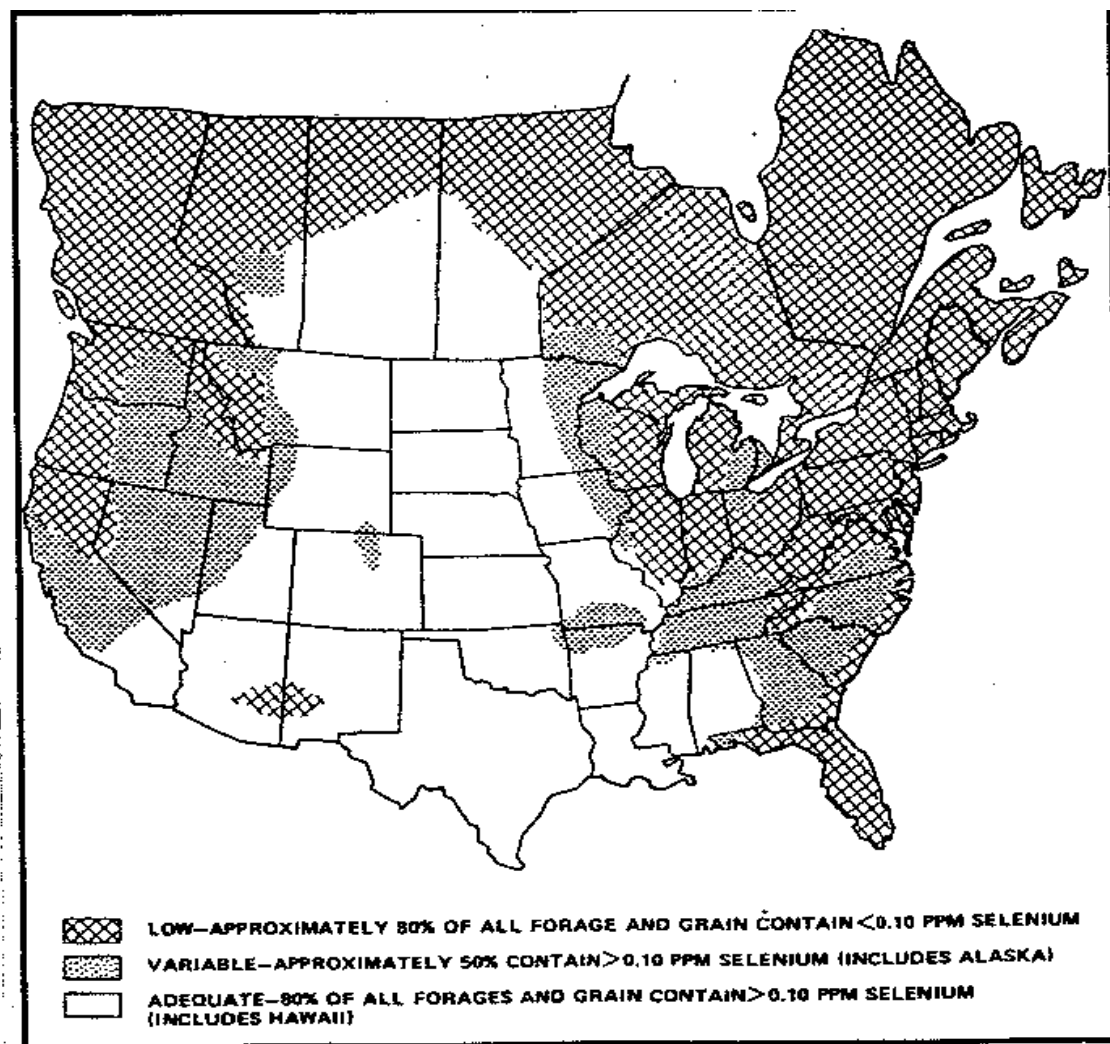
A 2001 study in Italy reported human toxic effects due to dietary exposure to 300 ug selenium/day. Toxic effects included thyroid hormone synthesis impairment, immune system impairment, hepatotoxicity, gastrointestinal disturbances, dermatological disorders, and hair loss. It has been reported in a few human articles that a consistent intake of 1,000 ug/day selenium over a long period of time will produce selenosis in humans (Mahan, 2008).

The recommended minimum dietary intake ranges from 10 ug selenium/day for infants to about 70 ug/day for male adults or pregnant females. The recommended maximum or upper tolerable intake ranges from 350 to 500 ug selenium/day for adults. The dietary difference between the recommended minimum daily intake and the upper tolerable intake of selenium is less than a tenfold difference. Figure 2 illustrates the recommended minimum and upper tolerable daily dietary intakes of trace minerals for humans. Note the narrow margin of safety in dietary selenium intakes. Careful monitoring of dietary selenium intakes is prudent. (Smolin, 1999, Research Pub Med Abstracts, January 2006).

Table 1. Selenium Nutrition & Toxicology

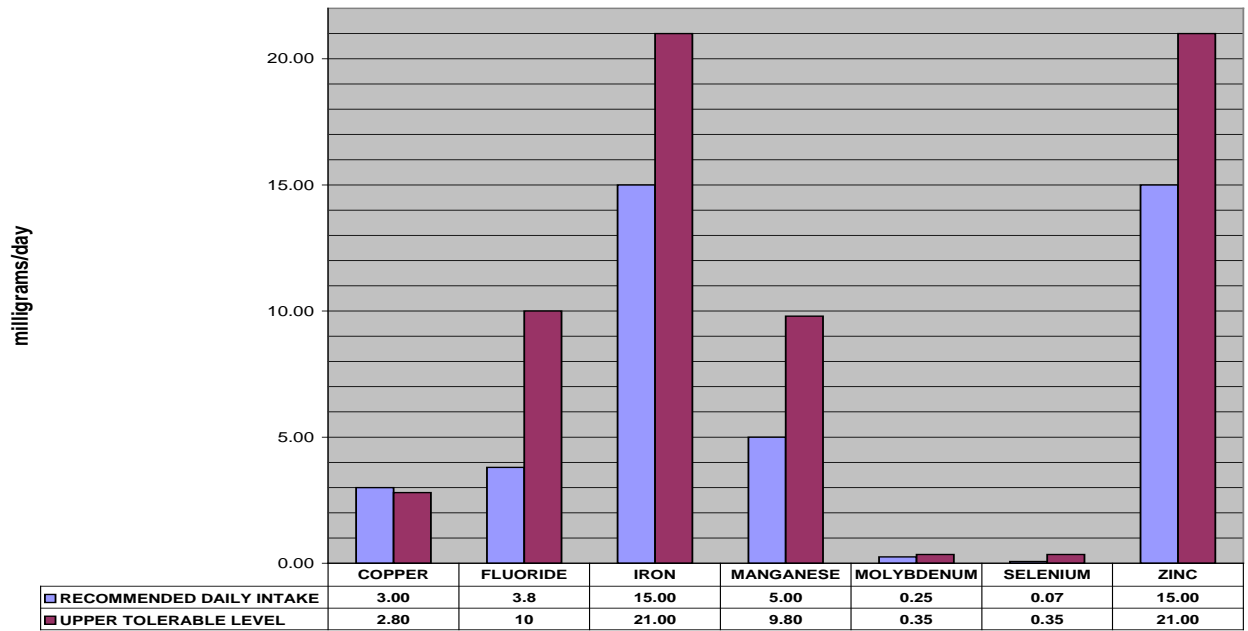
- Selenium and Mercury Ratios—
 - Viral Cardiomyopathy related to Mercury/Selenium Ratio Imbalances
 - Bioindicators for Ecosystem Risk Assessments
 - Swedish Study: Increased Mercury in Lake Fish & Decreased Selenium (Fly Ash) Input
- Swine Selenium Toxicity Dependent Upon Adequate Intakes—
 - Protein (without selenium)
 - Minerals
 - Vitamins
- Selenium Toxicity Affected by Metal Intake—
 - Arsenic, copper, cadmium, mercury form insoluble metal complexes with selenium increasing excretion, decreasing storage, and decreasing harmful effects of the metal compounds.
 - Urinary arsenic and selenium levels are similar.
- Selenium Recommended Human Minimum and Maximum Intake Levels—
 - Narrow Margin of Safety in Dietary Intake

Figure 1. USA and Canada Map of Selenium Distribution in Forage and Grain



North America Regional Distribution of Forage and Grains Containing Low (<0.10 ppm) to Adequate (>0.10 ppm) Selenium Concentrations

Figure 2. Human Trace Mineral Intakes—Recommended Minimum and Maximum Levels



II. Case Study of Swine Selenosis

A. Background

A USA Swine operation first noticed symptoms of toxicity in swine in 2004. The operation has variable number of pigs ranging from 100 to 340 sows and the business plans are to sell 100 suckling pigs every week. The other local operations are usually less than 100 sows. Diagnostic tests were run to determine the cause of the toxic symptoms, but all laboratory tests for bacterial and viral swine diseases were negative. The grains also tested negative for mycotoxins. Finally, the liver of an aborted fetus was analyzed and found to have high concentrations of selenium. The drinking water supply was also tested and did not contain selenium greater than the maximum contaminant level (0.05 ppm selenium).

B. Discussion

The toxic symptoms exhibited by the two northwest USA swine herds discussed in this report are listed in Table 2 and the biological sampling analyses and swine reference values are presented in Table 3. The most affected populations include the sows and piglets and the initial symptoms of selenosis were staggering, tremors, loss of hair, vomiting, lack of appetite, cracks and swelling at the coronary band, and hoof sloughing (Figure 3). The case study swine producer recommended that other producers observe all the different symptoms by the individuals pigs and consider the health impacts of all symptoms on the herd. In other words, each pig may have a different symptom such as loss of hair, staggering, or hoof problems, but the diagnosis of herd selenosis should be evaluated by symptoms exhibited collectively.

At the height of the symptoms, there were no litters produced and no income for about a year. All the swine had weight loss and all ages were affected. Especially impacted were the sows and their piglets. About one third of the approximately 300 swine had to be culled. The culled sows were sent to the slaughter house due to feet, leg, and reproductive problems or were euthanized onsite and buried. The surviving pigs were not medicated except to give the most affected ones B-complex vitamin shots.

Feeding adjustments included incorporating local barley (0.05 ppm selenium) into the pig feed, using no premix with selenium, and trying to use less soybean meal. Some of the symptoms did go away with lowering the levels in the feed.

Another USA swine operation described losing a mature sow after a seizure and weanling pigs that started staggering and later died. Sows were analyzed for bacterial and viral diseases such as leptospirosis and parvovirus which were negative. Their blood mineral content indicated high concentrations of selenium and zinc, but low concentrations of iron and manganese. Feeding adjustments included using a corn based diet.

C. Biological Monitoring Results

The case study reported 2007 biological monitoring samples from the swine including liver samples which were nearly twice the reference value of 2.5 ug selenium/gram or 2.5 ppm. The maximum blood sample was also twice the reference level of <0.10 ppm selenium, and hair samples (maximum 0.85 ug Se/gram hair), are presented in Table 3. The swine producer observations of swine selenosis symptoms included the following:

- Initial and individual symptoms exhibited are variable. For example, in a litter of nine piglets, only two piglets may have cartilage deformities.
- All selenosis symptoms exhibited by individuals in the herd must be considered to diagnose disease.
- The background or baseline blood selenium content of the herd is important information in diagnosing selenosis due to feed selenium content. At feed selenium concentrations of only 1.0 ppm for three days and the blood titer of 0.26 ug selenium/gram, the producer observed “teetering”, one early symptom of selenosis.
- The fat and meat consumed from swine with selenosis symptoms had a different flavor, described by the case study producer as “stinky feet”.

Mineral content of all feeds must be analyzed to formulate the proper mineral intake for adequate swine nutrition. The case study experience has suggested less than 5.0 ppm selenium content in the final feed formulation or in grazing forage to prevent acute selenosis. Researchers have suggested that prolonged exposures to high levels of selenium could produce the same symptoms of chronic selenosis. In these cases, the chronic dietary level should be reduced to less than 1.0 ppm total selenium in the swine diet (Kim et al., 2001, Mahan, 2008).

Table 2. Toxic Symptoms Exhibited by Swine Herds

Reproductive	Skeletal/Skin	Organ Indicators	Nervous System
No Estrus Cystic Ovaries Poor Milk Production Small Litters Deformed Piglets Abortions	Hoof—Separation, Ridges, Sloughing Coronary Band— Swelling, Cracks Swollen Joints Edema Sloughing Skin Hair Loss	Livers Enlarged Urinary Bleeding Salivating Vomiting Abscesses Ulcerations Weight Loss	Trembling Staggering Teeth Grinding Fighting Head Tilting Seizures

Figure 3. Observe Sows, Piglets and Hoofs for Selenosis Symptoms



Table 3. Biological Monitoring of Swine

Samples	Reference Values	Case Study Samples
Blood	>0.10 ppm symptoms	0.15 to 0.26 ppm
Liver	1.0 to 2.5 ug/g	3.0 to 4.9 ug/g (n=6) 6.3 ug/g Fetus
Hair	0.27 to 0.60 ug Se/g hair	0.71 to 0.85 ug Se/g hair
Urine	> 0.1 mg/L	
Muscle	>0.4 ppm	

D. Feed and Grain Analysis

Grain analyses from several different laboratories for September and October, 2007 were provided by the swine producer (Table 4). The concentrations of selenium reported were less than 1 ppm in the dry swine feed and alfalfa pellets, 10 to 112 ppm selenium in the swine premix and 600 to 1420 ppm selenium in the mineral supplements.

One study fed selenium in the form of sodium selenate or organic selenium yeast to finishing pigs for twelve weeks at concentrations ranging from 0.3 to 10 ppm. In the selenium enriched yeast diets, the correlation coefficients between dietary and hair selenium was 0.90. At eight weeks the selenosis was more severe at concentrations greater than 5 ppm dietary selenium. The symptoms included alopecia and hoof separation (Kephart, 1999).

As selenium in the diet increased, the daily weight gains and total pig weights decreased. The symptoms for pigs with white hair appeared sooner than pigs with black hair (which retained more selenium). Early 1960's research in South Dakota indicated that hide color may be correlated with adverse growth and reproduction symptoms of swine on seleniferous range plants (Kephart, 1999). Case study producer reported that selenosis symptoms and blood selenium content differed according to the hair color of the swine: swine with white hair accumulated more selenium than swine with red hair and swine with black hair accumulated the smallest amount of selenium. This may reflect other genetic differences (i.e. amount of lean tissue formation) and thus precipitating the onset of selenium toxicity, rather than simply hair color.

The two swine operations discussed in this report had severe cases of selenosis due to feeds bought locally. The origin of the grains mixed into the feed is unknown. Other local swine producers are also analyzing their herds for blood selenium and mineral content, but the results are not yet known. Unknown are the total impacts and losses to each swine operation and the industry in general. The two swine operations currently have healthy herds after they diluted the diet with a low selenium grain (local barley and corn based feeds) and eliminated the selenium mineral supplements. Visual monitoring of the health of the swine herd is ongoing. The grain and protein sources must be evaluated for selenium content as the maximum amount of selenium must be carefully monitored.

Table 4. Grain & Feed Analysis Data for Case Studies

Grains	Sources/Minerals	Results ug Se/g (ppm)
9/25/07	Swine Premix	112
	Hog Grower	2.6
	Bentonite	0.158
	Alfalfa Pellets	0.104
9/25/07	TPI Selenium (0.2%)	1420
	TPI Selenium (0.06%)	602
	TPI Trace Mineral H	9.8
	Premix (10 ppm Se)	9.8
	Ameribond 2X	<0.02
10/17/07	Swine Feed CHS	1.06
10/18/07	Dry Swine Feed	0.17
		No Mycotoxins (Aflatoxins)
10/30/07	SOY ADM Meal	0.718

III. Recent Research—Selenium in Feeds & Swine

A study by Oregon Department of Agriculture in September, 2007 evaluated swine feed samples for selenium content from four NW states. The types of feed included complete hog grower, oat mill byproduct, grain mix, and all purpose livestock feed. The product sources guarantee their products to contain a minimal amount of selenium ranging from about 0.1 to 0.3 ppm selenium. The laboratory analysis from South Dakota State University indicated that all the feed samples (13) had selenium concentrations ranging from 0.20 to 1.22 ppm selenium or 150% to 544% higher than the guaranteed minimum level. In other words, all the feeds had higher selenium content than guaranteed, up to five times the guaranteed levels of selenium. Soybean meal tested high at 0.72 ppm or 718% above expected 0.1 ppm selenium. Actual selenium concentration in product should be within 25% of feed label amount (0.1 to 0.3 ppm selenium). The Oregon Department of Agriculture is considering proposing a maximum selenium guarantee be added to the feed label if the above observations persists (November 27, 2007).

The selenium content of corn and soybean meal were recently analyzed over a three year period in various areas of the Midwest. Figures 4 and 5 show the selenium levels in corn and soybean meal, respectively. Note that corn from Iowa, South Dakota, North Dakota, Missouri and Kansas were higher in selenium compared to corn from Illinois, Wisconsin, Indiana, Michigan, Kentucky, Indiana and Ohio (less than 0.05 ppm selenium). The range from the highest to lowest in both corn and soybean meal was up to a seven fold difference. Maximum selenium concentrations were found in soybean meal from Nebraska (~0.85 ppm selenium) The ranges between highest to lowest concentrations of other minerals such a calcium and phosphorus in corn and soybean meal were considerably less (Cromwell, 2008).

A long duration study in England has evaluated the concentrations of selenium in the wheat grains from different plots over the past 160 years. The results indicated that selenium decreased in grains from fertilized or manured plots, influenced by sulphur inputs and atmospheric deposition. Improving grain yields through plant breeding has not resulted in a significant decrease in grain selenium from the fertilized plots. Maximum selenium value was 0.24 ug/gram grain from unfertilized plot (Fan, 2008). It should be noted that plants do not require selenium for their growth, while animals do. Therefore the amount of selenium in grains reflects soil and environmental conditions (Mahan, 2008).

An evaluation of concentrations of both selenium and arsenic in processed rice from the south central US versus rice from California was compared. The maximum concentrations were about 0.20 ug selenium/gram and about 0.30 ug arsenic/gram of rice grain. California rice concentrations were about half for both metals (Williams, 2007). No significant differences in grain levels of manganese, cobalt, copper, or zinc were observed between the samples. Maximum daily intakes of arsenic and selenium may be potentially exceeded if drinking water concentrations and intakes are considered.

A study in California evaluated the selenium levels in swine after a local feed company accidentally supplied about 150 grower pigs with high concentrations of feed selenium (average 122 ppm selenium). Biological samples of the swine blood selenium concentrations were taken initially and up to 72 days after the high selenium feed had been replaced with low selenium

(0.50 ppm) feed. About 46 to 72 days on the low selenium feed, the pigs blood selenium levels returned to normal (<0.10 ppm) and swine muscle selenium concentrations were considered to be at background levels (<0.40 ppm) which are similar for beef muscle consumption. The report concluded that after swine selenosis, the meat is safe to consume at about 60 days at minimal final feed concentrations of 0.50 ppm selenium (Davidson-York, 1999) as shown in Table 5.

Figure 4. Selenium in USA Corn

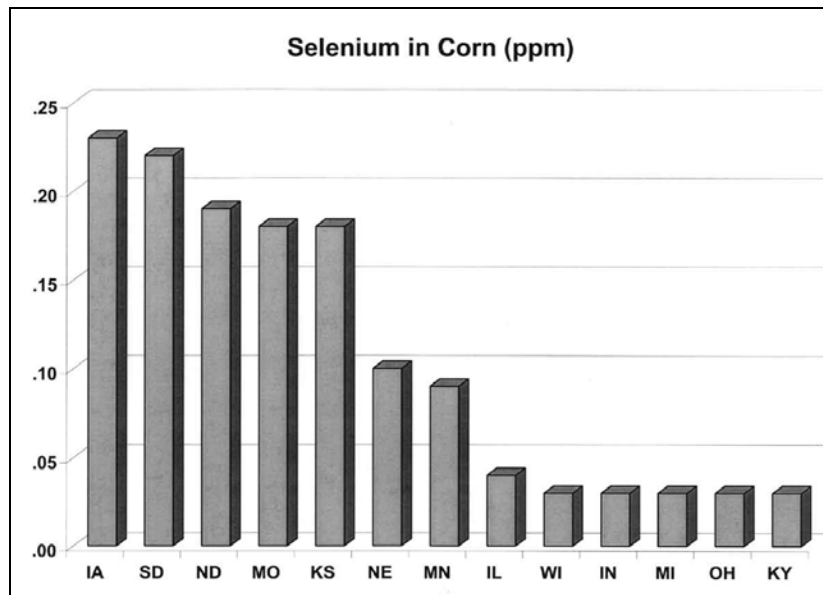


Figure 5. Selenium in USA Soybean Meal

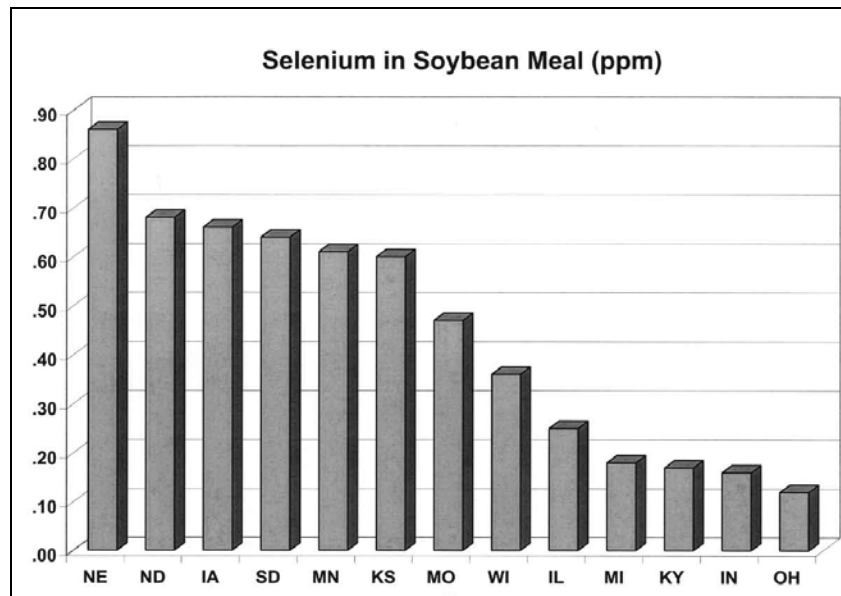


Table 5. After Swine Selenosis—Safe Consumption of the Pork?

1999 California Study—150 Grower Pigs

Accidentally Fed Feed ~122 ppm Se dry weight

Duration ~18 days

Selenosis Symptoms—

75% Pigs Affected; 15% Pigs Died

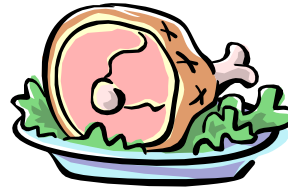
89% Pigs Had >1.0 ppm Blood Selenium

Post Selenosis Diet <0.5 ppm Selenium

Duration ~46 to 60 days

Muscle Selenium ~0.40 ppm wet weight

Safe to Consume



IV. China Experiences with Selenium—Micronutrient & Poison

One of the first maps identifying areas of selenium toxicity were passed along by Marco Polo in the 13th century depicting the silk road of western China. He noted where his horses could not eat the local range plants, which caused their hoofs to crack and drop off.

The areas of China which have been identified to be deficient in soil selenium include a wide belt from northeastern provinces of Heilongjiang and Jilin down to Yunnan province in the southwest (Li, 2006). The endemic cardiomyopathy and white muscle disease affected young people (especially males) and sheep. Biological sampling of blood indicated 0.02 to 0.04 ppm selenium which increased to 0.11 ppm selenium after supplementation with sodium selenite (Liu, et al., 1987). Figure 6 is a distribution map showing selenium concentrations in feeds and forages in China (Oldfields, 1995).

The prevalence of Keshan disease in Xunyi County of Shaanxi Province in 2005 was evaluated (Yang, 2006). Keshan disease is related to dietary selenium deficiency in juveniles and causes cardiomyopathy related symptoms. The recent survey reported no acute or subacute Keshan disease based on electrocardiography and clinical exams in the surveillance areas. Hair and grain selenium concentrations were low levels of 0.58 ug/g hair and 0.09 ug/g grain, respectively.

In contrast to deficient areas, selenium toxicity exists in Enshi county of Hubei Province (Liu et al., 1987). Human exposures include dietary sources but primarily inhalation exposures from using bone and coal, which are both rich in selenium and fluoride, as fuel for cooking.

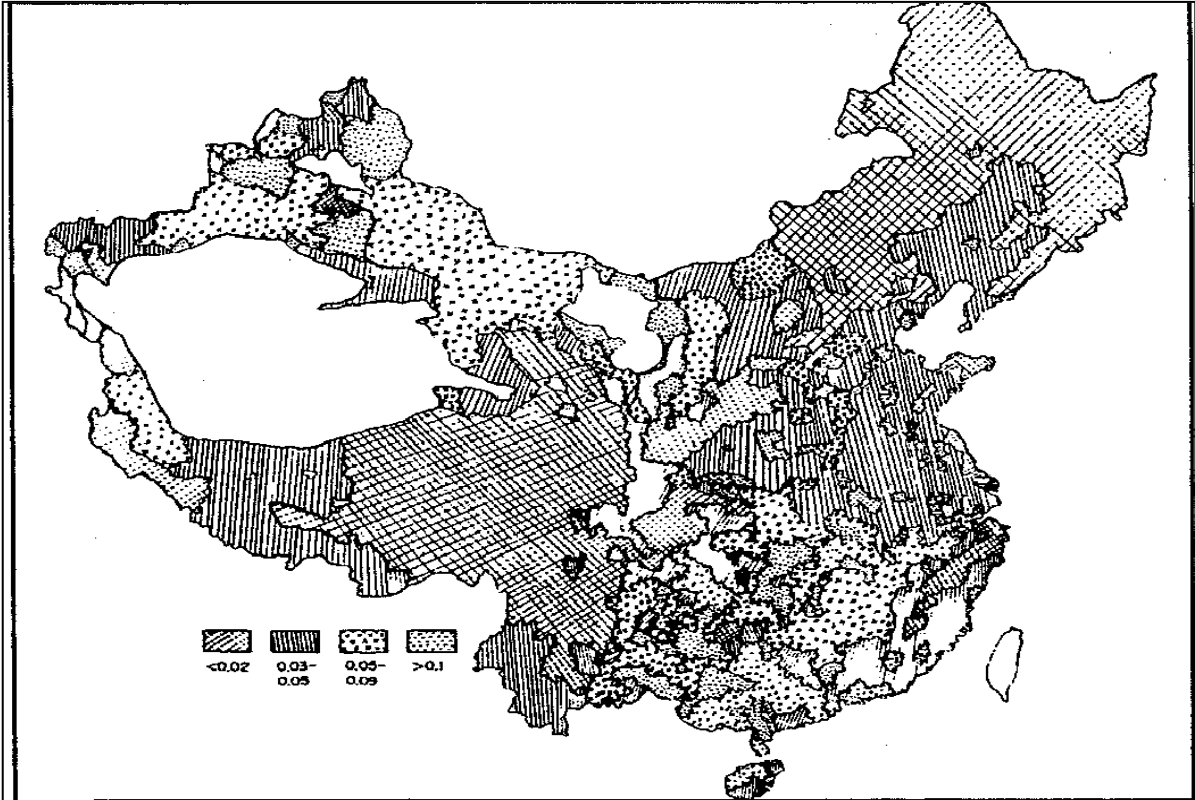
Shuang'an is a selenosis site in Ziyang County Shaanxi Province with soil concentrations as follows: Se = 17 ug/g, Mo = 99 ug/g, V = 1134 ug/g, F = 1041 ug/g, and As = 111 ug/g in the soil directly derived from bone coal and V-Mo ore dumps. Most plants which grow in the revegetated soil contain elevated Se, Mo, V and F concentrations. This ecosystem revegetation on the ore dumps may have been essentially responsible for selenosis incidence of Shuang'an in Ziyang County Shaanxi Province (Fang, 2004).

A recent study evaluated selenium in soil, grain, human hair and drinking water in relation to esophageal cancer in Cixian area, Hebei Province (Appleton, 2006). The study found that esophageal cancer areas had higher concentrations of selenium in the environment. Other factors need further evaluation such as mycotoxins, vitamins, nitrosamine in grains.

Recent reports indicate that the China pork industry is predominately family farms which raise 85% of the marketed pork, each family raising about two dozen pigs. Transportation of the rurally located swine to populated areas is a limiting factor in the growth of the industry. Unrelated to selenium, but impacting the Chinese pork industry is a contagious virus infection. The Blue Ear virus impacted about 290,000 pigs in 2007 with a respiratory and reproductive disease syndrome. The report noted that China does not need international help to test for the virus or disease. The disease is also known as PRRS (Porcine Respiratory & Reproductive Syndrome) which has a serological test to evaluate the serum virus levels or titers. A swine herd is "naïve" if the serum viral titer is zero and should not become serum titer positive under stress when the virus is shed.

The USA pork exports to China have recently been blocked due to trace concentrations of growth hormone (ractopamine) in USA swine. The European Union has recently approved a selenium enhanced yeast product for all animal feeds with a maximum total selenium content in final feeds of 0.5 ppm selenium (Murphy, 2006).

Figure 6. Distribution Map of Selenium Concentrations in Feeds & Forages in China



V. Summary

The minimum recommended dietary requirements reported in the Nutrient Requirements for Swine, 10th Revised Edition, 1998 are 0.30 to 0.15 ppm selenium for weanling pigs and finishing sows, respectively (National Research Council, 1998). In most animals, 3.0 ppm of dietary selenium approximates the minimum toxic dose. One constant factor in swine production is the production cost attributed to feed costs (55-65%). High quality feeds come at a high cost.

Organic selenium found in plants or yeast can be a source of selenium and are found in the protein component of the feed product. Therefore, the protein sources must be evaluated for selenium content as the maximum final selenium concentration must be minimized and carefully monitored.

A final rulemaking on “Selenium in Animal Feeds”, from the Food and Drug Administration and other government agencies was published April 27, 1973 (38 Federal Register 10458). Several provisions to be monitored are cited. 1) An upper limit on the concentration of selenium in premixes and will require the use of highly diluted premixes. 2) Each production batch of selenium premix is to be analyzed for selenium content. This analysis will assure that selenium premixes contain the labeled amount of selenium and in no case will this amount exceed 90.8 mg of selenium per pound. 3) Caution statements must be applied to ensure selenium premixes are prepared according to manufacturer’s label directions.

Mineral content of grains and protein sources must be analyzed to formulate the proper mineral intake for adequate swine nutrition. Research has indicated that 5.0 ppm total selenium content in the final feed formulation or grazing forage can be acutely toxic. Researchers have indicated chronic selenium toxicity at less than 1.0 ppm total selenium in the swine diet for a prolonged time. The initial or background blood levels of selenium in the swine will affect the positive or negative reactions to dietary exposures. The difference between required and toxic dietary levels of selenium has yet to be established for swine, but may reflect the narrow range of safe selenium intake in humans.

The acutely toxic symptoms of swine selenosis are well documented and individual symptoms are variable. One piglet in a litter may have crooked snot, due to cartilage deformity. The symptoms include staggering, tremors, anorexia, hair loss, degenerative liver, edema, separation of hoof/skin at the coronary band, and weight loss. Unfortunately, experts contend that once an animal is in the advanced stages of selenosis, it is difficult if not impossible return to health as the physiological damage has been done. The central nervous system damage is permanent, according to the case study producer. Chronic selenium dietary intake and the toxic threshold for human or swine selenosis have not been adequately evaluated.

The case histories of swine selenosis are located in USA regions with reportedly low soil selenium. However, the sources of the swine feeds are usually not reported by the grain producers or local marketing representatives. The swine producer's solution to the swine selenosis was to analyze the feeds, identify the sources of the feeds if possible, and perform ongoing visual monitoring of the swine herds for symptoms of selenosis. If symptoms of selenosis are identified, then biological monitoring and additional analysis can be performed. The selenium concentrations in the blood and liver samples were greater than twice the normal levels for swine.

The USA livestock and agricultural industry has evaluated chicken, cattle, sheep, swine, and horse toxicity of selenium from consuming seleniferous range forage for over 80 years. Research publications have indicated a maximum total selenium content of feeds, grains and natural forage at 5.0 ppm selenium. Setting a maximum value of selenium in grains and feeds marketed in the USA would be a significant contribution by the National Research Council and FDA to preventing livestock selenosis.

VI. References

- Appleton, J.D., et al., 2006. Selenium in soil, grain, human hair and drinking water in relation to esophageal cancer in Cixian area, Hebei Province, People's Republic of China", *Applied Geochemistry*, Vol 21(4), April 2006.
- Center for Disease Control, 2005. Human Exposures to Environmental Chemicals, Third Report, Atlanta, GA, July 2005.
- Combs, G., 2007. "Selenium Rich Wheat", Supplement to the *Dakota Farmer*, February 2007.
- Cromwell, G.L., 2008 "Selenium-A Unique Trace Element", Professor Swine Nutrition, University of Kentucky website.
- Davidson-York, D., et al., 1999. Selenium elimination in pigs after an outbreak of selenium toxicosis, *J. Vet. Diagn. Invest.* 11:352-356.
- Eisler, R., 1985. Selenium Hazards to Fish Wildlife, and Invertebrates: A Synoptic Review. Biological Report 85(1.5), Contaminant Hazard Reviews Report No. 5, Patuxent Wildlife Research Center, Laurel, MD, October, 1985.
- Emsley, J., 2001. *Nature's Building Blocks, An A-Z Guide to the Elements*. Oxford University Press Inc., New York, USA, 2001.
- Fan, M.S., et al., 2008. "Historical changes in the concentrations of selenium in soil and wheat grain from the Broadbalk experiment over the last 160 years", *Science of the Total Environment*, Volume 389(2-3), January 2008.
- Fang, W. and P. Wu., 2004. "Elevated selenium and other mineral element concentrations in soil and plant tissue in bone coal sites in Haoping area, Ziyang County, China." *Plant and Soil*, Volume 261(1-2), April, 2004. Springer Netherlands Publisher.
- Franke, K.W., 1934. A new toxicant occurring naturally in certain samples of plant foodstuffs. 1. Results obtained in preliminary feeding trials. *J. Nutr.* 8:597-608.
- Hagelstein, K. and J. Heinze, 2007. Environmental management of airborne metal particulate emissions in the recycling industry, *TMS Light Metals*, February 2007.
- Hamilton, S.J. and K.H. Buhl, 2003. Selenium and other trace elements in water, sediment, aquatic plants, aquatic invertebrates, and fish from streams in southeastern Idaho near phosphate mining operations: May 2001, U.S. Geological Survey Final Report. Columbia Environmental Research Center, Field Research Station, Yankton, SD, May 23, 2003.
- Kephart, K., editor, 1999. Selenium: Poison? Miracle Nutrient". In *Farm & Home Research*, South Dakota State University, College of Agriculture and Biological Sciences, Agricultural Experiment State, Volume 50(4):4-23, Fall 1999.

- Kim, Y.Y. and D.C. Mahan, 2001. Comparative effects of high dietary levels of organic and inorganic selenium in selenium toxicity of grower and finishing pigs. *J. Animal Science* 79:942-948.
- Kim, Y.Y. and D.C. Mahan, 2001. Prolonged feeding of high dietary levels of organic and inorganic selenium to gilts from 25 kg body weight through one parity. *J. Animal Science* 79:956-966.
- Li, Q. and Zhao, Z.H., 2006. "Prevalence state of Kashin-Beck disease in Qinghai Province: A retrospective analysis", *Chinese Journal of Endemiology*, Volume 25(6), November 2006.
- Liu, B.S. and S.S. Li., 1987. Endemic selenosis and fluorosis. In: *Selenium in Biology and Medicine*. Van Nostrand Reinhold, New York, pp. 708-716.
- Liu, C.H., et al., 1987. Regional selenium deficiency in feedstuffs in China. In: *Selenium in Biology and Medicine*. Van Nostrand Reinhold, New York, pp. 47-52.
- Mahan, D., 2008. Professor Animal Nutrition, Ohio State University, Columbus, OH. Personal Communication (mahan.3@osu.edu), April, 2008.
- Murphy, R., 2006. "Focus on Selenium: Part 3: Winds of Change", *Pig Progress*, Volume 22(8), 2006. (www.pigprogress.net)
- National Research Council, 1998. *Nutrient Requirements of Swine: 10th Revised Edition*, 1998. The National Academy of Sciences. (www.nap.edu/openbook/0309059933/html/57.html)
- Oldfield, J.E., 1995. *Selenium in Maps*. The Bulletin of Selenium Tellurium Development Association, Belgium, April, 1995.
- Smolin, L. and M.B. Grosvenor, 1999. *Nutrition Science & Applications*, Third Edition, Saunders College Publishing, Harcourt College Publishers, 1999.
- Williams, P.N., et al., "Market basket survey shows elevated levels of As in South Central U.S. processed rice compared to California: Consequences for human dietary exposures", *Environmental Science and Technology*, Volume 41(7), April 2007.
- Yang, J., et al., "Keshan disease surveillance in Xunyi County of Shaanxi Province in 2005", *Chinese Journal of Endemiology*, Volume 25(6), 2006.