Need and Value of Selenium in Animal Feeding

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INTRODUCTION

Until a few years ago, most nutritionists were not much concerned about selenium deficiencies. Difficulties in analyzing for selenium at the extremely low levels involved delayed understanding the function of selenium. Developing diets deficient in selenium was also a problem. But in the last 5 to 7 years a great deal of basic and applied data on selenium and its role in animal nutrition has been obtained.

DEFICIENT AREAS ARE WIDESPREAD

Selenium deficiencies have been shown to occur in the major livestock producing areas of the world. A survey by Wolf et al. (1963) showed that over half the states in the U.S.A. (32 states) have white muscle disease (WMD) in calves and lambs. This is verified in the National Research Council (NRC) report by Oldfield et al. (1971) wherein they report only 10 states where it is felt that selenium deficient areas do not exist. This indicates that a selenium deficiency is of concern throughout the United States since feeds produced in selenium deficient areas may be fed in areas where selenium is high enough in the locally produced feeds.

NEEDED BY ALL FARM ANIMALS

Selenium has been shown to be an essential mineral element for swine, chickens, turkeys, cattle, horses, sheep and many other animals. During recent years, and especially during the last 5 to 7 years, many field cases of selenium deficiency have occurred under practical feeding conditions with livestock and poultry. Therefore, selenium supplementation is needed by all classes of farm animals in the U.S. Thus, when FDA approves the use of selenium in livestock feeding, it will join the other trace minerals (copper, iron, iodine, manganese, cobalt and zinc) as the seventh one which the animal producer and feed industry need to be sure that all feeding programs supply the level needed. Oldfield et al. (1971) stated that "the losses in livestock and poultry production caused by a deficiency of selenium are difficult to estimate, but they are substantial."

INTERRELATIONSHIP WITH OTHER NUTRIENTS

There is an interrelationship between selenium and vitamin E. Both are needed by animals and both have metabolic roles in the body in addition to an antioxidant effect. In some cases, vitamin E will substitute in varying degree for selenium or vice versa. However, there are syndromes which will respond only to selenium or vitamin E. There is extensive evidence that, although selenium cannot replace vitamin E in nutrition, it reduces the amount of tocopherol required and reduces the onset of symptoms of a deficiency. Oldfield et al. (1971) state this may be due to selenium functioning in the absorption of dietary vitamin E.

The sulfur amino acids protect against several diseases associated with low intakes of selenium and vitamin E. Oldfield et al. (1971) state that the only suggestion that has been offered to explain the efficacy of these amino acids is that they contribute to the antioxidant activity of the tissue. This theory is based on their known antioxidant activity in vitro and the observation that synthetic antioxidants are capable of replacing them as protective agents.

This brief discussion indicates that there is still much to learn about selenium and its interrelationship to other nutrients. In many cases, scientists refer to a deficiency syndrome as a selenium and/or vitamin E deficiency. This is because the exact role of either selenium or vitamin E

or both is not yet entirely understood. The sulfur amino acids, and possibly other nutrients, can also further complicate the picture.

WHY DEFICIENCIES ARE OCCURRING

A selenium deficiency is related to the level of vitamin E in the diet. Therefore, a selenium deficiency may occur when levels of vitamin E are low or lacking whereas it might not occur if the level of vitamin E is adequate. Thus, it is interesting to review reasons as to why selenium and/or vitamin E deficiencies are showing up more frequently throughout the U.S.A. and other parts of the world.

Reasons for selenium deficiencies

Following are some possibilities as to why selenium deficiencies are being encountered more often:

1. Selenium deficient areas are being recognized throughout the world as analyses of feeds and diets are being conducted. Highly sensitive chemical methods are now available for analyzing selenium levels as low as 0.005 ppm in feeds and tissues.

2. Vitamin E deficient diets are occasionally occurring and this will increase the need for selenium under certain conditions.

3. It is possible that certain strains and/or breeds of animals require more selenium than others.

4. Selenium needs could also be higher because of the trend toward cross-breeding and the increased growth and productivity of these animals. Cross-breeding of dams, properly done, can increase pork produced 40 per cent and beef produced 35 per cent with Brahman-British breed crosses. Moreover, selection for faster growing animals and more productivity per animal (which are not cross-bred) also increases nutrient needs.

5. Analyses of feeds for selenium can be misleading. In many cases, the availability of selenium is low. Thus, many diets thought to be adequate in selenium, may be deficient, when the availability of selenium is taken into consideration.

Reasons for vitamin E deficiencies

Following are a number of possibilities as to why vitamin E deficiencies are being encountered more often:

1. There are indications that heating grain to lower the moisture level can destroy some of the natural vitamin E. Many nutritionists in Europe feel that the need to dry feed grains is one reason why supplemental E is helpful there. More grain drying is also being used in the U.S.A. and other areas of the world.

2. There are some who think that storing high moisture grains, and perhaps spoilage, can also be just as conducive to destroying natural vitamin E as heating the grain. Some of this thinking also exists in Europe since the incidence of vitamin E deficiencies is the greatest during damp cold seasons. This is an important possibility to explore since high moisture grain feeding is increasing in the U.S.A. The deficiency is probably here to stay because feed efficiency is increased when high moisture grains are fed as compared to dried grains to cattle.

3. It is also possible that the heat treatment during the pelleting of feeds may destroy natural vitamin E, although experimental evidence is lacking on this suggestion.

4. The requirements for vitamin E are increased by feeds or fats with higher levels of polyunsaturated fatty acids (PUFA) especially under oxidizing conditions. During oxidation of fatty acids vitamin E can be destroyed. In addition, PUFA also increases the physiological need for vitamin E.

5. More solvent extraction of the protein supplements is being practiced. This tends to lower the fat content and also the vitamin E level in these feeds.

6. Less use is being made of pasture and dehydrated alfalfa meal in swine and poultry rations. This lowers the vitamin E level in the ration since both are excellent sources of this vitamin. It may also lower the selenium level since certain samples of alfalfa are good sources of selenium.

7. Animals are being bred to grow faster with less feed and better carcass quality. Animals are also producing more during reproduction. All of this could be increasing vitamin E needs.

8. It is possible that certain strains and/or breeds of animals require more dietary vitamin E than others. There is considerable evidence accumulating that genetic differences can alter certain nutrient needs of animals.

9. There are factors in feeds which interfere with the utilization of vitamin E.

10. Vitamin E deficiencies may occur in areas (such as has been demonstrated in Sweden) where the grains are very low in selenium. These areas are important to know since grains form such a large part of the ration of swine.

11. The vitamin E level in feeds has been overestimated. Unfortunately, most of the vitamin E values reported for feeds consider total tocopherol content only. The most important form, however, is alpha-tocopherol because it has the greatest biological activity. The other six forms of vitamin E vary in biological activity from ⅓ to 1/100 that of alpha-tocopherol. With the exception of alfalfa meal, most feeds contain less than 50% of the total tocopherol in the alpha form. Thus total tocopherol content of a feed is not a reliable guide of its biological vitamin E value.

These are a number of possibilities for an increased need for selenium and vitamin E. There may be others. They are sufficient, however, to indicate why deficiencies of selenium and/or vitamin E are occurring more frequently.

EFFECTS OF A DEFICIENCY

The first evidence that selenium was important in nutrition was obtained with non-ruminants. Recently, the use of synthetic amino acid diets very low in selenium with chicks has shown that severe deficiency symptoms and death occur even in the presence of very high levels of vitamin E in the diet (Thomas and Scott, 1969). Supplementing with selenium prevented all deficiency symptoms. As special low selenium diets are studied more extensively with other animals more information will be obtained on what the exact role of selenium and/or vitamin E is in the deficiency syndromes obtained. Following is a brief summary of the selenium and/or vitamin E deficiency symptoms obtained in farm animals.

Swine

Swine develop liver necrosis (hepatosis diatetica), degeneration of cardiac muscle (mulberry heart) and skeletal muscle, deposits of ceroid pigments in the adipose tissue, subcutaneous edema, accumulation of straw colored fluid in the body cavity and death. Penhale et al. (1972) recently showed that selenium supplementation of the sows diet with 0.1 ppm of selenium prevented deaths and other selenium and/or vitamin E deficiency symptoms in the young pigs to 12 weeks of age. Information by Wastell et al. (1969) at Iowa State University showed that selenium and/or vitamin E deficient sows farrowed stillborn and other pigs which died shortly after birth which were small in size. The author observed the same thing in a trip to Australia in 1970.

One of the important selenium and/or vitamin E deficiency symptoms is that an apparently healthy looking pig will suddenly die. Sometimes this happens to recently weaned pigs which are penned with pigs from other litters. The stress of establishing the new boss and the peck order in the pen causes many to die. Many times the addition of selenium and/or vitamin E may not give a growth response in experimental trials but it will prevent death losses. Iowa State and Michigan State University scientists have shown this to be the case in recent experiments. Therefore, one needs to closely observe any death losses and one should also necropsy the pigs and observe the histopathology in both the control and treated animals. The histopathology may be the best clue when borderline deficiencies are present.

It is interesting that Money (1970) of New Zealand stated "It is suggested that the sudden-death-in-infants syndrome might be due to selenium and/or vitamin E deficiency."

Horses

Observations in New Zealand by Dodd et al. (1960) and Hartley and Grant (1961) suggest that a selenium deficiency may lead to myopathy in the horse. White muscle disease has been endemic in foals in New Zealand and deaths usually occur with severely affected animals. They show many hemorrhages and thick, firm layers of yellow-brown fat. The degenerated skeletal muscle has a watery appearance and is chalky white. The horses also lose hair. There are many field reports indicating that selenium may be concerned with the "tying-up" syndrome in horses (Smithcors, 1962). Most veterinarians treating this condition use a combination of selenium and vitamin E.

Chickens

They develop exudative diathesis, poor growth, poor feathering, fibrotic degeneration of the pancreas, edema, hemorrhage, anemia, myopathy and death. More is known about selenium and vitamin E with chickens and turkeys than with the other farm animals. Scott et al. (1969) summarizes this subject very thoroughly in their book.

Turkeys

The symptoms differ somewhat from the chick. Turkeys show exudative diathesis, edema and hemorrhages but not to the same extent as the chick. The most characteristic symptom is a degeneration of the gizzard musculature. Turkeys grow poorly and have a high mortality rate.

Ruminants

White muscle disease (WMD) is observed in young calves or lambs born to dams fed an extremely low ration in selenium—about 0.02 ppm in dry matter (Oldfield et al., 1963). Both skeletal and heart muscle are affected. Their work, and that of other scientists, has shown that supplementing the dam with selenium will prevent WMD in the calves or lambs.

Hartley and Grant (1961) showed that selenium supplementation increased the lamb crop with sheep.

A number of workers have shown that selenium is effective with scours in lambs and cattle (Wolf et al., 1963; Kendall, 1960; Smithcors, 1962 and Hartley and Grant, 1961). In California trials, Kendall (1960) successfully treated cattle with selenium for scours that had not responded to antibiotics or anthelmintics. Smithcors (1962) reported that dosage of selenium similar to that used by Kendall (1960) of 10 mg of selenium and 500 mg of vitamin E was effective in clearing up a persistent diarrhea in cattle and improved growth rate. Hartley and Grant (1961) reported a beneficial response to selenium by beef and dairy calves in New Zealand that had exhibited severe and rapidly progressive unthriftiness associated with profuse diarrhea. These observations would indicate that selenium supplementation should be considered when one encounters scours which do not respond to other treat-

ments. Scours are occurring with cattle producers throughout the U.S.A. and are very serious in many areas. The possibility of selenium being involved should be considered.

LEVELS NEEDED

Oldfield et al. (1971) state that the critical level for dietary selenium, below which deficiency symptoms are observed, is apparently about 0.02 ppm for ruminants and 0.03 to 0.05 ppm for poultry. They state that when supplementary selenium is fed, higher levels than the minimal requirements have been proposed. This would permit satisfactory distribution of the selenium throughout the large feed mass and would overcome variations in feed intake by individual animals. Experimental evidence to date indicates that 0.1 ppm of selenium in the ration of all classes of livestock and chickens and 0.2 ppm in the diet of turkeys is a safe means of eliminating a deficiency of selenium.

TOXICITY OF SELENIUM

Selenium in excess is toxic. Consumption of plant materials containing 400 to 800 ppm of selenium have been fatal to sheep, hogs and calves (Oldfield et al., 1971). A chronic selenium toxicity in livestock occurs when animals consume seleniferous plants containing 3 to 20 ppm over a prolonged period of time (Oldfield et al., 1971).

The toxic levels of selenium in ppm in the ration according to the latest NRC nutrient requirement publications are as follows: Swine 5–8; Chickens 10; Dairy cattle 5; Beef cattle 8.5; Horses 5–40; and Sheep 10–20 ppm. A recent Wisconsin study showed that 2 ppm could be toxic to lambs (Rotruck et al., 1969). All of these animals require about 0.1 ppm of selenium in the total ration. The turkey requires 0.2 ppm. A look at the ratio between the level required and the level which is toxic shows a big safety factor between them. It would seem that a level of at least 2–5 ppm of selenium is needed before toxic effects may occur. This is a ratio of 20–50 to 1 between the level at which a toxic level occurs and the level needed in the diet. This ratio means that selenium is no more toxic than some other trace minerals which have been used for years in animal feeding. In the pig, for example, the ratio between the level which causes a toxic effect and the required level is as follows: copper 25 to 1; iron 50 to 1; manganese 12.5 to 1; and zinc 40 to 1 (Beeson et al., 1964). More recent studies indicate the ratio for copper is about 42 to 1 since copper requirements of the pig have been lowered. If extra zinc and iron are supplied, it prevents the toxic effects which occur occasionally from using 200 ppm of copper in the ration.

Therefore, it seems that if the addition of selenium to animal feeds is carefully controlled, that it can be used safely. However, care will need to be exercised in making sure this is done properly as is also the case with other trace minerals.

SUPPLYING SELENIUM TO ANIMALS

Probably one of the safest ways to supply selenium would be to add it to salt. All animals need salt and the approximate level needed is known. Moreover, when cattle, horses, sheep and other animals are on pasture with little or no concentrate supplementation they will consume salt from the mineral box and get their selenium on a continuous basis since they will eat salt frequently. Salt with selenium can also be mixed with calcium and/or phosphorus and the trace minerals to give a complete mineral mixture. The approximate intake of salt and/or a complete mineral mixture for animals is known. Therefore, the level of added selenium can be calculated to meet the requirements for selenium. Oldfield et al., (1971) stated "the level of selenium in the concentrates should be such that toxicity would not result even if, by error, 10 times the intended concentration were used." If selenium were added to salt or a complete mineral mixture to supply 0.1 ppm in the total ration, this means there could be a difference of 10 times the estimated mineral consumption by animals before Dr. Oldfield's NRC committee (Oldfield et al., 1971) recommended safety factor was reached. It is hard to visualize animals consuming as much as 10 times the estimated salt and/or complete mineral mixture they need. But even if they did, this would still be safe since they would need to consume 20 to 50 times the requirement before toxicity might occur. Therefore, it appears that adding selenium to salt would be a good method to supply it.

The Wisconsin Station (Rotruck et al., 1969) has already shown that the addition of sodium selenate to trace mineralized salt to ewes was effective in preventing white muscle disease in their lambs. They used levels of 26 and 132 ppm of selenium in the salt, which was about 0.15 and 0.90 ppm as added selenium to the total diet of the ewes. These levels of supplementation for the ewes provided maximal protection against occurrence of white muscle disease and promoted growth of their lambs. Supplementation of selenium at these levels on a continuous basis for 4 years did not result in abnormal appearance, deaths or excessive accumulation in the tissue of the ewes or their lambs. However, supplementation of a higher level (264 ppm of selenium) in the salt, which supplied about 2 ppm of selenium to the diet of the ewes, did depress gains of lambs in 2 of 4 years.

Selenium can also be supplied to animals by injection. In fact, injections of selenium, and usually in combination with vitamin E, are frequently used in the treatment of animals in selenium deficient areas.

TABLE I

Results from feeding Selenium to Pigs

Ration Fed for First 14 Weeks	All pigs switched to same ration after 14 weeks with no Selenium added		
	Slaughtered at Start	Slaughtered After 30 Days	Slaughtered After 65 Days
Basal (contained 0.041 ppm Se)	0.052[1] (0.206)	0.043(0.167)	0.041(0.155)
Basal + 0.1 ppm Se	0.080 (0.315)	0.082(0.304)	0.056(0.217)
Basal + 0.5 ppm Se	0.098 (0.390)	0.095(0.363)	0.083(0.311)

[1] Level of Selenium on a wet tissue basis and on a dry basis (in parentheses) of the longissimus dorsi muscle.

TISSUE LEVELS OF SELENIUM

Oldfield et al. (1971) state that "concentrations of selenium that might be toxic to humans have not been found in the skeletal muscle of cattle and sheep; they have not been found even when these animals have been exposed to toxic concentrations of dietary selenium. Liver and kidney from healthy animals, however, may contain more than 5 ppm of selenium." However, this should pose no problem since only 1.5 pounds of liver from cattle is consumed per person yearly in the U.S.A. A much lower level of sheep liver is available.

A Michigan State study (Groce et al., 1970) involved adding a level of 0.1 or 0.5 ppm of selenium to the ration of the pig. The data in Table I show the results obtained. A level no higher than 0.1 ppm in pork muscle on a wet basis or 0.4 ppm on a dry basis was obtained when 0.5 ppm of selenium was added to the diet. These selenium levels would be further diluted in the human diet since pork makes up only a small part of the total food consumed. Supplementation of swine rations probably would need to be no higher than 0.1 ppm of selenium. Therefore, the level of selenium in pork would be low and would pose no hazard as far as the human is concerned. Therefore, the use of selenium in livestock supplementation should pose no problem as far as the safety of the meat is concerned.

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