Vitamin E and Selenium in Cattle Production

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Introduction

Vitamin E and selenium (Se) have an important role in efficient, profitable cattle production and in maintaining optimum health during early growth, reproduction and lactation. They overlap in functioning as antioxidants and each has additional specific functions related to maintaining health. Vitamin E functions predominantly in cellular membranes, and selenium functions in the glutathione peroxidase system of intracellular components. Many factors are concerned with vitamin E-Se metabolism and these factors influence requirements as well as the clinical signs and lesions of deficiency.

Like classical nutritional diseases in the past such as rickets, osteoporosis, grass tetany and goiter, much of the current interest in the deficiency syndrome of vitamin E-Se is due to practical field problems causing economic losses in cattle production. A deficiency of these nutrients is a world-wide problem and thus there is much interest in current information. Controversies have existed for many years, with valid reasons, concerning the response and value of vitamin E-Se in cattle disease problems. The controversies stem not so much in errors of observations and results, but in interpretation of information as applied to a complex nutritional deficiency. As new information is obtained from experimental work in cattle on the specific role of each nutrient and the interrelationship between them and other factors, real progress will be achieved in improving cattle production.

History

In 1922, California workers reported the existence of a nutrient needed to prevent sterility and insure normal reproduction in the female rat (1). It was referred to as vitamin E and later identified as alpha-tocopherol and its homologs. Information is available from the early work that is as valid and applicable today as when it was first published, namely:

1. It required a high (8 to 15%), rancid (unsaturated) fat diet to readily produce the deficiency.
2. Wheat germ oil was a potent source of the nutrient (vitamin E).
3. Cod liver oil (as a source of vitamin A and D) made the deficiency worse and has been used for many years in experimental diets to produce a deficiency.
4. It took a long time to deplete reserves of vitamin E and produce a deficiency in some rats.
5. Results were highly variable, some rats were more resistant to a deficiency than others.

For three decades following the early work on the value of wheat germ oil in infertility in rats, it was promoted as a cure for infertility problems in cattle with no experimental evidence to prove its effectiveness. At times the claims were rather extravagant—i.e., that vitamin E would cure and prevent contagious abortion. These claims were questioned in the 1940's when Minnesota workers fed four generations of cattle on special diets very low in vitamin E and incapable of supporting reproduction in rats (2). There were no deleterious effects on growth, reproduction or lactation in cattle. Stress factors such as polyunsaturated oils were not included in the ration.

In 1938 the "suckling paralysis" of rat pups nursing mothers deficient in vitamin E was discovered to be due to skeletal muscle degeneration (3). From this, vitamin E was seen as having a role in prevention of nutritional muscular dystrophy in calves. Work soon followed demonstrating that vitamin E had a role in preventing myopathy in calves, whether produced experimentally or occurring under field conditions (4-5).

Role of protein and selenium: In the early 1940's, in an entirely different area of research, the role of dietary factors in prevention of liver necrosis produced by feeding low-protein diets or diets high in cod liver oil was pursued. In 1951 it was concluded that three factors were needed to prevent liver necrosis, namely cystine (from protein), vitamin E and an unknown nutrient referred to as "Factor 3" (6), determined in 1957 to be selenium (7). Subsequent work demonstrated that lambs with myopathy responded to selenium treatment (8).

Manifestations of Deficiency in Cattle

A number of disease manifestations in cattle that respond to vitamin E-Se treatment have been described (Table 1). They have a rather insidious appearance in herds. Except for the myopathy in calves, most of the manifestations have been established by the response of field problems to vitamin E-Se treatment and information extrapolated from sheep and other species. There is much overlapping in clinical signs, lesions and consistency of manifestation depending on the activity and stress on specific tissues at the time of the deficiency. Skeletal myopathy in growing calves has been long recognized as an important problem. Impaired growth, while described, has not been a characteristic manifestation

and may be more of a secondary effect. The manifestations are consistent with the deficiency in swine and other species (21). Many ill-defined locomotor, muscular weaknesses, and related disturbances have been described in the literature by veterinary practitioners as responding to vitamin E-Se. Biochemical and functional lesions might be expected to precede morphological changes.

These diseases respond to vitamin E-Se treatment and other factors such as the amount of polyunsaturated fat in the ration, low protein rations, spoiled (rancid fat) in feed and the physical and mental stress of cattle have important roles. Many of the manifestations are not pathognomonic for vitamin E-Se deficiency, but are also manifestations of other diseases—some due to infectious agents. The term "responsive" seems appropriate until there is more experimental evidence of the exact role of various contributing factors, and to avoid implications of specific dietary deficiencies. Referring to all of the manifestations as the vitamin E-selenium deficiency (VESD) syndrome, as the Scandinavian workers do for the analogous disease in swine (22) might be less confusing.

TABLE 1. Vitamin E-Selenium Deficiency Manifestations In Cattle.

White muscle disease (myopathy) in young calves (4,5)
Myodegeneration in adult cattle (9, 10)
Retained placenta (11-15)
Unthriftiness in growing and mature cattle (16)
Metritis, cystic ovaries (17)
Diarrhea in growing cattle (16)
Mastitis (15, 17)
Increased susceptibility to infection, toxic chemicals and decrease in immune response (17-20)

Feed and Tissue Values

Feed and tissue values associated with a vitamin E-Se deficiency are summarized (Table 2) (23-24). These values may be used as guidelines since additional factors influence requirements and are involved in producing a deficiency. Recent reports suggest that erythrocyte glutathione peroxidase (GSH-PX) values are a reliable indication of the selenium status of cattle. The GSH-PX values indicative of Se deficiency in cattle will depend on the assay technique used in the laboratory, tissue used and how it was handled. Polyunsaturated fats or oils, added to a ration or from deterioration (oxidative rancidity) of fats in cereals in the ration during harvesting or in storage, are of special importance. These oxidized products from fat not only destroy vitamin E in the ration, but on consumption deplete body reserves and enhance a deficiency. It has been stated that small amounts of polyunsaturated oil in a ration can increase the requirement for vitamin E as much as 100 fold (25-26). At the other extreme, in the absence of unsaturated oil in a ration and the presence of a small amount of selenium, the requirements for vitamin E are low and a deficiency is difficult to produce experimentally. There is considerable loss of vitamin E in the rumen associated with feeding rations high in concentrates and as the amount of concentrate feeding increases, vitamin E losses also increase (27). Disturbances of the rumen resulting in tympany and diarrhea have been associated with vitamin E-Se deficiency in cattle (10).

TABLE 2. Suggested Feed and Tissue Values in Vitamin E and Selenium Defficiency In Cattle.

Item	Deficient	Borderline	Adequate*
Blood			
Tocopherol (plasma) (ug/ml)	<0.20	0.20 to .80	0.80>
Selenium** (serum) (ng/ml)	<40.	40. to 70.	70.>
Liver			
Tocopherol (ug/g, wet wt)	<3.0	3 to 10	10.00>
Selenium (ppm, dr wt)	<0.4	0.4 to 1.0	1.20>
Feed			
Tocopherol IU/kg	<5.0	5—20	20.00>
Selenium (ppm)	<0.04	0.05 to 0.1	0.10>

* In absence of stress factors.
** Whole blood values are 2 to 2.5 times higher.

Experimental Manifestation of Deficiency During Late Gestation and at Parturition

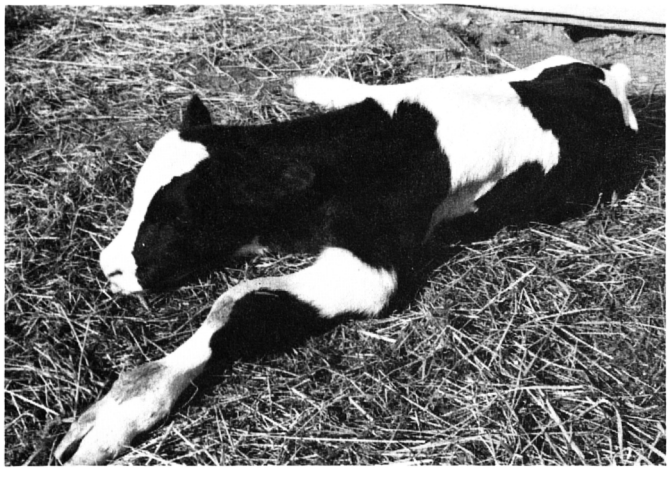
Most of the information on the role of vitamin E-Se during late gestation and parturition in cattle has been from field problems (4), information extrapolated from sheep and other species and the response to prepartum treatment with vitamin E-Se (11-15, 17). With the exception of a Minnesota study (2), which gave negative results, information on experimental production of a deficiency in pregnant cows was not found. Much of the interest in the role of vitamin E-Se in cattle is at parturition and the production of a viable, healthy calf. A pilot-type of experiment was conducted to determine the influence of feeding oxidized cod liver oil to pregnant cows during the last 3 months of pregnancy (28).

Seven, 4 to 5 year-old Holstein cows were used that were obtained from the Michigan State University Dairy Herd as culled cows due to low-milk production. They all had produced normal calves previously and there was no history of reproductive disturbances. Breeding dates and pregnancy examinations indicated the cows would calve at 1 to 2 week intervals during an 8 week period. At 3 weeks before the first cow was due to calve they were all fed grass hay free-choice and a concentrate mixture of ground corn and soybean meal to meet energy and protein requirements. An iodized salt-mineral mixture was offered free-choice. Each cow was fed 120 grams oxidized cod liver oil daily mixed in the con-

centrate. The cod liver oil was prepared as previously used to produce placental lesions of vitamin E deficiency in the rat (29).

The first 5 cows to calve (fed the ration for 3 to 8 weeks) had normal calves and no reproductive problems were evident. The last 2 cows to calve (fed the oxidized cod liver oil for 10 and 11 weeks) had weak calves that were unable to stand and nurse (Fig. 1). Both calves had an enlarged, hard tongue. The cows and calves had low blood tocopherol and selenium values. At parturition both cows had an excessive amount of hemorrhage. The placentas were expelled with the calves and were edematous. No other deleterious effects on the cows were noted. One calf was treated with 1200 IU alpha tocopherol and 5 mg selenium by injection and 1000 IU alpha tocopherol daily in milk from its mother given by stomach tube. It gradually recovered and was normal in 12 days. The other calf was given its mother's milk by stomach tube, but no vitamin E-Se supplementation. It died in 5 days. Lesions were widespread myopathy as described previously (4-5).

FIGURE 1. Prostrate, weak, 1-day-old calf born to cow fed oxidized cod liver oil during last 10 weeks of gestation. Blood plasma total tocopherol value was 0.42 (ug/ml) and selenium 24 (ng/ml). Plasma values for calf's mother were total tocopherol 0.83 (ug/ml) and selenium 12 (ng/ml). Note protruding tongue. This calf recovered after treatment with selenium and large amounts of alpha tocopherol.



The results suggested that polyunsaturated oils fed during late gestation would produce clinical signs of vitamin E-Se deficiency in cows and calves at parturition. The technique could be used for additional studies on the pathogenesis of vitamin E-Se deficiency during reproduction in cattle.

Prevention and Treatment of Deficiency

Vitamin E and selenium have an important role in a complete cattle health program. Since these nutrients have a role in immune response and resistance to infection, they are important even though clinical signs or a history of a deficiency are not evident (18-19). Some veterinarians are of the opinion, that we are beyond the practicality of using injections for prevention and should emphasize proper supplementation of rations. Selenium should be in the total ration at the recommended and allowable amounts of 0.1 ppm. Selenium premixes are available that, when added to concentrate mixtures in specified amounts will provide 0.1 ppm in the total ration. Some investigators of field problems are of the opinion that the amount of selenium in the total ration should be higher than 0.1 ppm—as much as 0.3 ppm for a preventive program. If cattle are on pasture, selenized salt (20 ppm) is recommended.

The suggested amount of vitamin E activity in a total ration is 20 IU vitamin E/Kg if an adequate amount of selenium is in the ration and there are minimal stress factors present. Cattle fed mainly concentrates and high producing milk cows during late gestation should be fed 40 IU/Kg ration. If stress factors are present, such as high concentrate feed, unsaturated fats either added or from rancid cereals, or unaccustomed physical activity, the vitamin E content should be increased.

In therapy of clinical cases of deficiency, injectable vitamin E-Se preparations are available. Clinical cases respond rather slowly—or a "lag" effect. Apparently high amounts of vitamin E are required to correct metabolic lesions. The permissible amount of selenium is injected or supplemented followed by increasing amounts of vitamin E. While selenium can be toxic in excessive amounts, no toxicosis due to large amounts of vitamin E have been reported. Some report better results with higher amounts of vitamin E than requirements would suggest. Amounts of 20 to 30 IU/Kg body weight can be injected initially and the diet supplemented with 100 IU/Kg. Injectable sources of only vitamin E should be considered in administering higher amounts. To achieve higher amounts using preparations containing also vitamins A and D might cause toxicosis especially due to vitamin D.

Discussion

The importance of vitamin E and Se in cattle production has developed slowly over the years. A number of developments in recent years have influenced requirements. Harvesting, storing and processing of feeds have changed from slow, natural drying in the field or in cribs to artificial drying when storing or to feeding grains high in moisture after fermentation. More confinement systems are in operation with less access to pasture or fresh green forage. Milk production goals have about doubled from 30 years ago. High concentrate feeding and less roughage and pasture have provided less natural sources of vitamin E and conditions for the destruction of vitamin E in the rumen.

Dependable, simple techniques and information are needed for the practitioner to more accurately determine the vitamin E-Se status of cattle. The GSH-PX activity values

are suggested as a reliable indicator of selenium status and general health (30). The erythrocyte lipid peroxidation assay has been suggested as an indicator of the vitamin E status (31).

Some workers are of the opinion that vitamin E-Se function more like a hormone and, during a deficiency, active and proliferating tissues such as the skeletal musculature, fetal placenta, uterus and mammary gland are more susceptible to injury and infection.

The more recent information on the role of vitamin E-Se in resistance to infection, resistance to toxic substances such as the halogenated hydrocarbons and in improving the immune response have attracted much interest and attention.

Toxicity: Selenium can be toxic to cattle when injected or fed in excessive amounts. The clinical signs of toxicity are somewhat similar to vitamin E-Se deficiency. Cattle that are vitamin E-Se deficient appear to be more susceptible to a toxicity than those previously fed adequate amounts. Feeds containing amounts in excess of 3 to 5 ppm Se can be toxic to cattle. Premixes containing concentrated amounts of Se should be carefully labelled and stored so they are not accessible to cattle and not accidently used as such for feed.

Caution should be exercised in regard to any overdosage in the use of injectable selenium preparations. Muscular tissue is sensitive to excessive amounts of selenium causing necrosis at the site of injection and clinical signs of lameness (32). Multiple injection sites or subcutaneous administration should be considered.

Summary

In recent years vitamin E and selenium have been recognized as having an important role in the proper functioning of a wide variety of cells, tissues and organs that are essential in maintaining health of cattle. Since vitamin E and selenium are nutrients, and with judicious use, there should be no concern about harmful residues in tissues. Additional, much needed research, will do much to maintain healthy, profitable cattle production and clarify areas of uncertainty.

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