PEER REVIEWED

Selenium Status in Cattle: Interpretation of Laboratory Results

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Abstract

Selenium may affect a wider range of metabolic functions than only those associated with nutritional myodegeneration, but there is controversy regarding the type of problems that may be associated with suboptimal selenium status in cattle. An animal's nutritional requirement for selenium and vitamin E depends on age, physiological stage and species. In cattle, low selenium status becomes critical near calving when cows are more susceptible to intramammary infections, retained placenta, or infections in the neonate. Analysis of serum samples submitted to the Iowa State University Veterinary Diagnostic Laboratory between January 2000 and July 2001, showed a mean (±SD) serum selenium value of 68.4±17 ppb for adult Holstein cows in the Iowa and Wisconsin areas. Because cattle receiving higher levels of selenium and vitamin E than typically recommended may show improved immunocompetence, the levels found in this survey can be considered adequate for reproductive performance, but marginal for optimal resistance to mastitis pathogens or for adequate transfer of selenium to the suckling calf. This report also contradicts previous claims that selenium deficiency contributes to stillborn/weakborn calves. Studies relating selenium concentrations in blood and serum to common clinical conditions of cattle attributed to poor selenium status are reviewed; methods of supplementation are also addressed.

Résumé

Le sélénium peut influencer un large éventail de fonctions métaboliques au delà de la myo-dégénérescence nutritionnelle. Toutefois, le genre de problèmes associés à un statut sous-optimal de sélénium chez le bétail est encore sujet à controverse. Les besoins alimentaires d'un animal en sélénium et en vitamine E dépendent de l'âge et de l'état physiologique et varient d'une espèce à l'autre. Pour le bétail, un faible statut en sélénium est particulièrement critique au moment du vêlage lorsque les vaches sont plus sujettes aux infections mammaires, à la rétention placentaire ou aux infections des nouveaux-nés. L'analyse d'échantillons de sérum soumis au Iowa State University Veterinary Diagnostic Laboratory entre janvier 2000 et juillet 2001 dévoilait une moyenne (± écart-type) de sélénium de 68.4 ±17 ppb pour les vaches Holstein adultes de certaines régions de l'Iowa et du Wisconsin. Parce que le bétail qui reçoit des doses de sélénium et de vitamine E plus élevées que la norme établie peut devenir plus immunocompétent, les valeurs de sélénium rapportées dans cette étude peuvent être considérées comme adéquates pour une bonne performance en reproduction mais sont moins conformes pour une résistance optimale contre les pathogènes impliqués dans la mammite ou pour le transfert adéquat de sélénium aux veaux allaitants. Notre rapport contredit aussi l'affirmation qu'un déficit en sélénium contribuerait à une hausse du nombre de veaux morts-nés ou faibles à la naissance. Les études qui établissent un lien entre la con-

centration du sélénium dans le sang ou le sérum et les conditions cliniques communes chez le bétail qui sont attribuées à un pauvre statut en sélénium sont revues et les méthodes disponibles pour l'apport supplémentaire sont aussi discutées.

Introduction

Severe selenium (Se) deficiency has long been associated with nutritional myodegeneration (NMD). More recently it has been recognized that marginal Se status can also increase susceptibility to neonatal diseases, mastitis and reproductive disorders in cattle.[3,15,16,65] Thus, assessment of Se status has become an integral part of production medicine and consequently, various Se assays are offered by veterinary diagnostic laboratories worldwide. At the Iowa State University Veterinary Diagnostic Laboratory, Se analysis is requested more than any other trace element, well over 500 per year. Across laboratories, a major limitation in determining adequate Se status in cattle has been the different methods of assessing and interpreting Se values. Serum, blood, or blood glutathione peroxidase (GSH-Px) are the three most common parameters used. Ideally, interpretation of these analytical results should be based on the association between a clinical or subclinical disease and the parameter studied. In the case of Se, the clinical effects of an extreme deficiency are nutritional myodegeneration and reduced growth rate (Figure 1), while marginal Se status is associated with subclinical production losses, such as reduced fertility, increased susceptibility to neonatal infections, mastitis and reproductive disorders (*i.e.,* retained placenta, cystic ovaries, metritis).[22,23,29,30,55,65]

In the last few years, there have been great advances in understanding the function of Se in mammals. Thus, in addition to the antioxidant functions of glutathione peroxidases[6] which have been associated with NMD, Se is an essential component of three iodothyrodine deiodinases involved in thyroid hormone metabolism[33] and three thioredoxin reductases[58] which help regulate the redox state of cells. Selenoprotein W, which is found predominantly in muscle, is thought to have a role in preventing NMD.[68] There are at least another 8-20 selenoproteins which regulate oxidation reduction reactions in cells.[8] Because all selenoproteins are dependent on Se intake, Se status in cattle must be considered in light of this newer information. In particular, it is important to recognize that Se may affect a wider range of metabolic functions than just those associated with NMD. From the bovine practitioner's per-

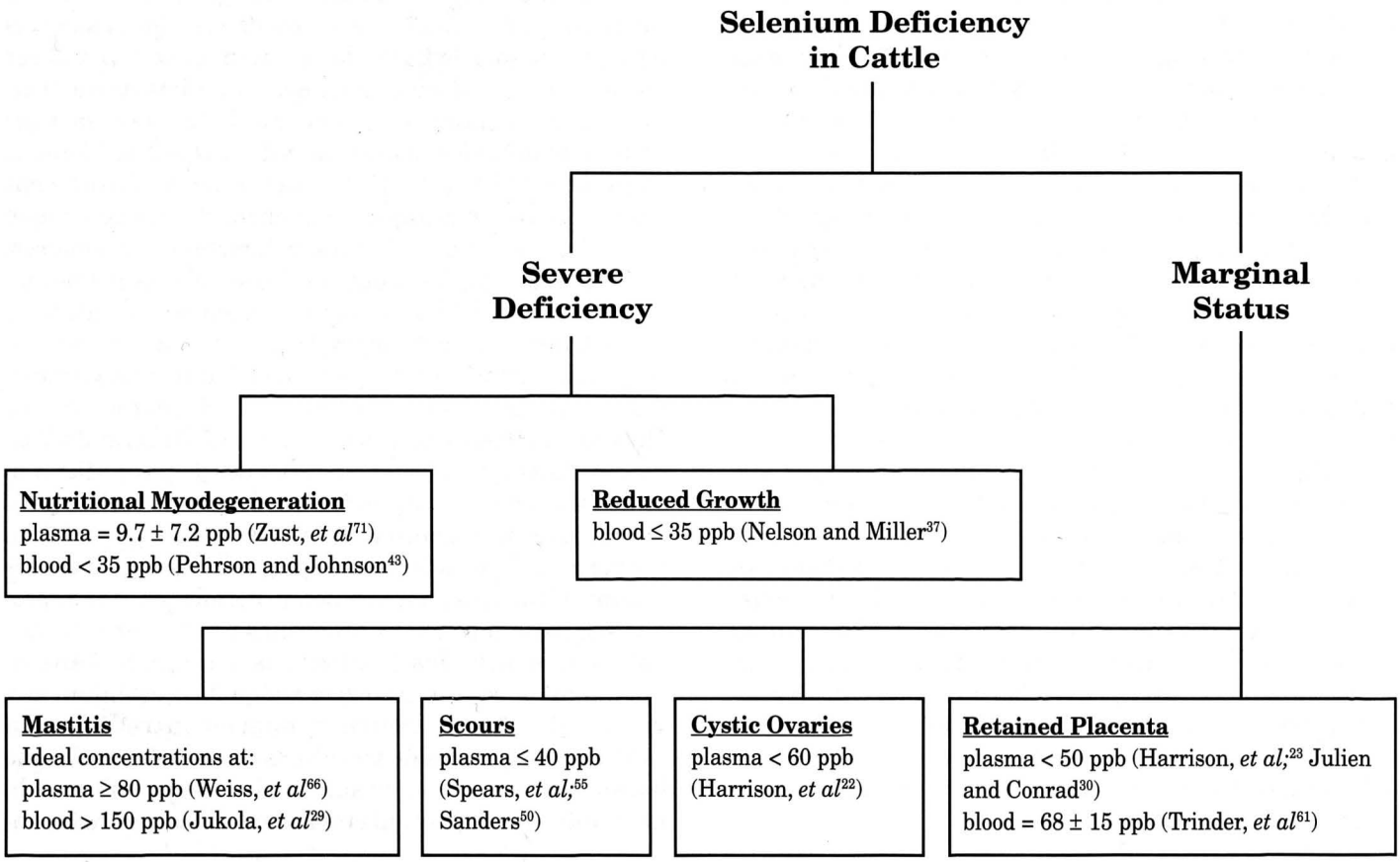

Figure 1. Selenium responsive conditions.

spective, it is unfortunate that most recent work on Se metabolism has been done in small rodents and human cells, rather than cattle.

In this report, we review some of the studies relating to selenium assays, the effect of Se deficiency in cattle, and the use of data collected by our laboratory from dairy cows in the midwest to interpret serum values.

Se Analysis and Interpretation

Analytical results from 204 sera collected from adult Holstein Wisconsin and Iowa cows between January 2000 and July 2001 and submitted to the Iowa State University Diagnostic Laboratory are presented in Figure 2. These are not survey samples representing herd status, and because the history is usually unknown, the health status of the cows was also unknown.

Serum was analyzed for selenium using Zeeman atomic absorption spectroscopy. Briefly, serum was diluted with Triton X-100 and compared to a standard curve of 40 and 80 ng Se. A palladium/magnesium nitrate matrix modifier was used and the spectrometer is a Perkin-Elmer Z5100 with furnace controller HGA-600 and autosampler AS-60.[45,56]

The overall mean (±SD) serum Se concentration was 68.4±17 ppb from the 20 farms represented. If a serum Se concentration of 80 ppb is considered necessary for maximal immune response against mastitis pathogens,[66] 71.1% of the cows sampled were below the target level. When whole blood was analyzed instead of serum, the overall mean (±SD) whole blood Se concentration in 50 cows was 139.3±26.9 ppb, with a range from 90–200 ppb (data not shown). Of these, 60% were below the 150 ppb target value suggested for maximal immunity against mastitis pathogens.[29] Serum values of 70 ppb and whole blood levels of 138 ppb have been associated with Se intake of 3.0–3.5 mg Se/day as sodium (Na) selenite;[14,34] however, higher levels of 5–6 mg Se/cow per day of the inorganic Se salts, or alternatively 3 mg cow per day of an organic yeast form,[a,b] have been recommended for lactating and gestating dairy cows to optimize immunocompetence and the Se status of the newborn, but this level of Se is presently extralabel.[32,42,57] Lactating cows receiving 3 mg Se per day as Na selenite or Na selenate will produce milk containing approximately 15 ppb Se;[42] this is lower than the 30-50 ppb considered adequate in milk for the neonate.[38] A yeast product,[42] or dried brewers grain,[14] fed to cows to provide 3 mg of Se each day resulted in mean Se concentrations of approximately 30 ppb in the milk.

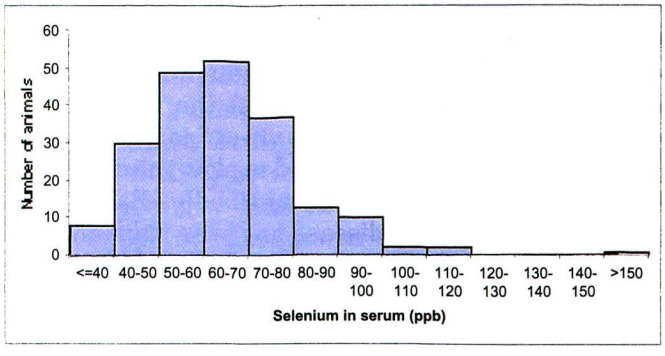

Figure 2. Summary serum selenium concentrations for 204 adult Holstein cows assayed by the Iowa State Univeristy Veterinary Diagnostic Laboratory from January 2000 through July 2001. Selenium was determined by electrothermoatomization-atomic absorption spectroscopy.[45,56]

For interpretative purposes, it is important to know how serum Se and whole blood Se values compare with one another in the same animals. However, the diagnostic accuracy for estimating the blood Se concentration from the serum Se value and vice versa is quite limited, and each should therefore be interpreted separately.[36]

In general, serum or plasma Se concentrations more accurately reflect recent changes in Se intake whereas whole blood Se more accurately represents long-term status because Se is incorporated into the red blood cells during their formation. The advantages and limitations of each have been discussed more extensively elsewhere.[20,36,60,64]

Relationship of Se Levels to Clinical Conditions

Clinical mastitis

It is well established that Se and vitamin E (vit E) status of dairy cows is related to mammary gland health and prevalence of intramammary infections.[29,66] Different measures of mastitis have shown improvement with Se/vit E supplementation: somatic cell counts, duration of infection, number of clinical cases in the herd, number of quarters infected and bacterial concentrations.[16,54] Common mastitis pathogens reported include *Staphylococcus aureus*, *Actinomyces pyogenes*, *Corynebacterium* spp, *Streptococcus* spp, and *E. coli*.[2,16,29] When somatic cell counts (SCC) were used as an indicator of udder health and milk quality, an almost linear correlation was observed between increasing plasma Se concentrations and lower SSC[66] (Figure 3). If a SCC measure less than 250 x 10^3 cell/ml is considered acceptable, levels of 80 ppb Se in plasma are desirable.[66] However, because SCC do not necessarily indicate the presence of clinical mastitis at levels less than 500 x 10^3 cells/ml, and because low SCC may be observed with low Se status,[13] some researchers prefer to compare the relationship between Se status and the percentage of quarters harboring mastitis pathogens. Using this approach, Jukola *et al*[29] reported that prevention of major mastitis pathogens is attained at blood Se levels exceeding 150 ppb, and suggested values of 180 ppb or higher for maximal protection against *Staphylococcus* spp.

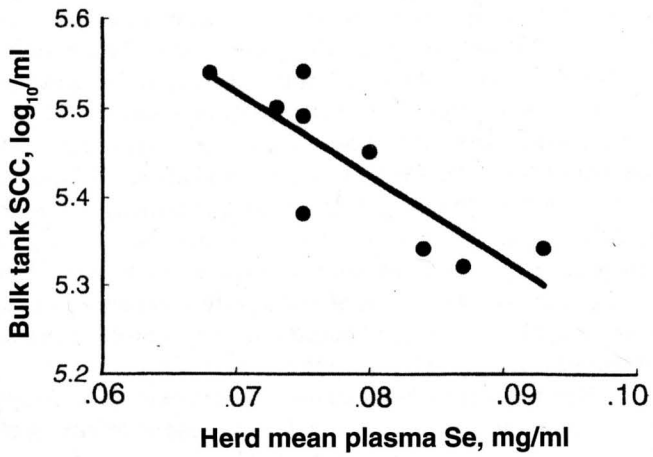

Figure 3. Relationship between herd mean selenium concentration in plasma and bulk tank milk somatic cell count (SCC). Reprinted from Weiss, *et al.*[66]

Nutritional myodegeneration (NMD)

NMD has been described in cattle from several months to 2-years of age.[11] Attempts to reproduce the disease experimentally by feeding diets deficient in Se/vit E have frequently failed in the absence of concurrent stress.[5] Thus, factors such as the composition of milk, the portion of polyunsaturated fatty acids in lush forages, exercise, sulfur content of pastures, etc., are important variables that may act to precipitate the disease.[5,24] Fortunately, the disease responds to therapy and calves showing signs of NMD may recover if promptly treated.[25] When the heart is affected, however, the myopathy is frequently fatal. Because the fetus can concentrate Se in the liver at levels three times higher than in the dam, it is usually born with sufficient reserves to compensate for the low Se intake through colostrum and milk during the first week of life.[63] Obviously, the age at which extra Se will be required depends on the amount of reserve Se stored during late pregnancy. When born with adequate Se levels, risk of NMD exists beyond two months of age,[43] therefore an injection of Se/vit E sometime during the first two months of age may be necessary. If the dam is consuming more than 3 mg of Se per day during the dry period, placental transfer of Se to the calf should be adequate and provide target levels of greater than 500 ppb (wet-weight) of Se in the liver of the newborn calf.[1]

Growth

Selenium is critical for optimal growth during the early stages of life, but has little or no influence on finishing cattle weighing 748 lb (340 kg).[12] Studies conducted in areas where pasture forage contained less than 0.05 ppm Se (dry matter [DM] basis) showed remarkable improvement in weight gain of yearling or weanling calves when supplemented with Se.[34,35,69] Koller *et al*[34] reported that heifers and steers offered a salt-mineral mix containing 90 ppm Se as Na selenite were 88 lb (40 kg) heavier at weaning at 10-months of age; the Se deficient calves were considered at risk of NMD (GSH-Px less than 15 mU/mg Hb). In another study, cows with blood Se of 20 ppb or less were supplemented with Se using intraruminal boluses, or their calves were injected at birth with Se. The calves had blood Se levels of 35 ppb or less. Weaning weights were increased by an average of 44 lb (20 kg) when cows were supplemented, and by 70.4 lb (32 kg) when calves were injected with Se.[37] When Se was supplemented using an intraruminal bolus in 1-year-old heifers, liveweight gain was improved by 15% over an 11-month period.[69] In this study, non-supplemented calves had a mean blood Se level of 10 ppb, whereas supplemented animals had a mean level of 79 ppb.[69] Blood concentrations represent the overall means from a random sample of five animals per group chosen on each bleeding day every two months for the eleven-month period after administration. Other studies have not observed this dramatic effect on weight gain as a result of Se supplementation, reporting 15.4-28.6 lb (7-13 kg) increased gain during the first year of life,[21,55] or no effect.[25] From a practical standpoint, reduced calf weight gain may be expected when plasma Se levels are less than 30 ppb, but levels of 40 ppb or higher during the first months of life have not resulted in better growth rates.[55] When GSH-Px values of 35 mU/mg Hb or higher are used as the standard, no difference in weight gain was observed during the first year of life.[27] It appears that growth rates may be affected when Se levels are comparable to those of calves at risk of NMD. In the studies cited, calves had adequate vit E levels.

Neonatal infections

Improved control of infectious disease is achieved by decreasing exposure to pathogens and by increasing the natural resistance of the host. There is an association between low serum immunoglobulin levels in young calves and increased incidence of disease, particularly neonatal diarrhea.[10] Supplementation with Se, vit E, or both, has been shown to improve humoral and cellular responses to vaccines,[15,48] to inhibit the replication of infectious bovine rhinotracheitis virus,[47] to raise IgG concentrations in the neonate,[59] and to slow the disappearance of maternally acquired antibodies.[39] Improved resistance to infectious disease has been substantiated by field observations showing a reduced incidence of diarrhea after Se and/or vit E supplementation.[50,70]

The Se levels necessary to enhance immunity, however, are still open to debate. In one study the incidence of enteric disease was similar in two groups of calves with average plasma Se levels of 20 ppb and up to 40 ppb;[65] however in three other studies, there was a decrease in pre-weaning mortality when plasma levels

were greater than 40 ppb.[9,50,55] Some authors suggest that the late-term fetal liver Se concentration should exceed 2200 ppb on a dry-weight basis (>500 ppb wet-weight basis), and that they may be deficient if levels fall below 1000 ppb on a dry-weight basis (<250 ppb wet-weight basis).[63]

According to Abdelrahman and Kincaid,[1] cows would need to be supplemented with more than 3 mg of Se/day for 60-d prepartum in order for suckling calves to have plasma Se concentrations of 50 ppb; this is about three times higher than the NRC minimum nutritional requirement of 0.1 mg/kg.[38]

Stillbirth and weak calf syndrome

Weak calf syndrome is a condition where newborn calves appear weak, are unable to stand and nurse and often die shortly after birth. There is no single causal agent that has been identified at this time. The rationale for assuming that selenium deficiency may contribute to weak or stillborn calves is partly based on its involvement in other reproductive disorders, such as retained placenta, cystic ovaries, as well as the development of NMD. Data from 60 stillborn or weakborn beef calves submitted to the Iowa State University Veterinary Diagnostic Laboratory during the winter of 2001 were examined. Of those cases submitted for identification of pathogenic organisms, one of 38 was positive for BVDV by immunohistochemistry, however no IBR (0/16), leptospira or campylobacter (0/19), or neospora (0/9) were diagnosed. Bacterial septicemia was diagnosed in four of the 60 cases. Of the 25 cases analyzed for liver selenium, 12% (3/25) were considered Se deficient (<250 ppb). In an earlier survey,[28] 24 stillborns examined had liver selenium levels within or above the adequate range of 250-500 ppb (wet-weight). These two studies suggest that selenium deficiency, as determined by Se liver concentration, is unlikely to be a cause of weak calf syndrome or stillbirths.

Reproductive disorders and fertility

The primary reproductive diseases of dairy cattle that respond to Se/vit E supplementation are retained placenta (RP), cystic ovaries and metritis. These diseases can affect fertility by increasing days open. The administration of 50 mg of injectable Se three weeks prior to calving to cows on a Se deficient diet (plasma levels 30 ppb at calving) was effective in reducing the incidence of cystic ovaries and metritis; combined Se and vit E supplementation also reduced the incidence of RP.[23] In other studies, injection of Se/vit E three weeks before expected parturition reduced the incidence of RP, even when adequate levels of Se were provided in the diet.[3] Trinder *et al*[61] and Julien and Conrad[30] reported that herds fed a diet containing less than 0.05 ppm Se (DM basis) experienced a high incidence (~40-50%) of RP. Blood Se levels of affected animals were 68±15 ppb, whereas control herds without history of RP had blood Se levels of 93 ± 15 ppb.[61] Their studies also suggested that RP, when not induced mechanically (i.e., difficult delivery, twin births) or pathologically, could be reduced to nearly zero by administering 50 mg Se and 680 IU of vit E intramuscularly (IM) at 21-days prepartum. However, factors such as age of the cow, twin births and overconditioning during the dry period should always be considered when the RP rate is high, and in many herds a 10% RP rate is commonly reported.[52]

Of the 204 serum Se values reviewed by our laboratory in this survey (Figure 2), only 5% had values less than 50 ppb, which is the level suggested to increase the risk of RP, and also considered inadequate for optimal ovarian function and uterine health.[23]

The effect of Se administration on fertility has been inconsistent across studies. Many factors, including age, variation in Se/vit E content of the basal diet, timing of supplementation, dosage, and method of administration may account for some of the discrepancies. *In vitro* studies have shown that Se stimulates the proliferation of granulosa cells, potentiates the effect of FSH, and enhances estradiol output.[7] This effect on ovarian cells is supported by studies where Se/vit E injections increased the total percent of fertilized ova in cattle on an adequate plane of nutrition.[51] When 50 mg of injectable Se was given to cows 30 days postpartum, fewer services per conception, and higher pregnancy rates at the second service were attained.[4] Similar doses administered three weeks before parturition improved the first service pregnancy rate,[3] although this effect was not found in another study.[31] In the latter study, both treated and non-treated cows had blood Se levels over 100 ppb. In cattle with poor conception rates and nearly undetectable GSH-Px, conception rates were improved by a single injection of barium selenate.[40]

A major portion of the mid-piece of sperm is a polymerized form of the membrane associated phospholipid hydroperoxide glutathione peroxidase.[62] If this structure is altered because of Se deficiency, it could theoretically lead to decreased male fertility.

Methods of Supplementation

A popular and practical practice in recent years has been to fortify concentrate rations to provide Se salts at a rate of 0.3 ppm Se (DM basis), which is the maximum legal daily level allowed by the FDA since 1987.[19] When an additional 2 to 3 mg of Se/cow/day were added to a basal diet containing 0.1 ppm Se (DM), there was no difference between selenite (SeO_3) or selenate (SeO_4) salts based on blood, plasma and GSH-Px activities.[41,42] Feeding both Se forms resulted in adequate serum and blood Se levels of 70-80 and 140 ppb, respectively. How-

ever, the organic form of Se given as a yeast product increased both plasma and blood Se levels more effectively than the inorganic compounds. Regardless of Se source, plasma levels reached a plateau after four weeks of supplementation. Interestingly, the concentration of Se in milk was nearly 3-fold higher when the diet was supplemented with the Se yeast product, whereas equal supplementation with inorganic Se failed to elevate milk levels.[42] This is believed to be due to the selenoamino acids present in the yeast, which replace some of the sulfur-containing amino acids used for the synthesis of milk proteins. Organic sources of Se have been available in other countries for several years, but have only recently been approved by the FDA for use in the US by poultry. They are currently under review for use in swine and ruminants.[a] For lactating dairy cows, Weiss *et al*[66,67] recommended that cows be fed 5 mg/day as Na selenate instead of 3 mg as the higher dosage resulted in lower somatic cell counts. These levels may exceed the maximum level approved by the FDA.

A practical way for beef cow operations to supplement Se is to provide free-choice salt-mineral blocks and licks. Levels of 13.6 mg of Se/lb (30 mg/kg) have been described as adequate, assuming the cows consume at least 100 g/day.[44] However, this level was considered inadequate by Koller *et al*.[34] They concluded that Se should be added to the salt-mineral mix at a rate of 41 mg/lb (90 mg/kg). Obviously, the amount considered adequate in a mineral block is dependent on total consumption of salt per day, and intakes vary between cattle. Typically, mineral consumption ranges from 25 to 50 g/head/day, therefore the proposed level of 90-120 ppm Se seems adequate. The use of the organic Se form in a salt-mineral mix for nursing beef cows nearly eliminates the risk of NMD.[42]

Another effective method for providing Se under range conditions is administration of intraruminal pellets, glass boluses or osmotic pumps. In such formulations, Se should be dispensed slowly and evenly at a rate of 3 mg per day. Studies have shown they provide a consistent supply of Se by elevating plasma Se from less than 20 to more than 70 ppb for five months, and GSH-Px levels remain increased for over a year.[26,27,49] Furthermore, no calves born to supplemented cows suffered from NMD.[26] In calves, this method of supplementation should not be used until they are over three months of age to allow for adequate reticulum development. Unfortunately, intraruminal devices are not available for use in the US, however they are used in Europe and Australia.

Injectable Na selenite products generally offer short-term benefit, and are typically used in younger, fast growing calves. In severely deficient 440 lb (200 kg) heifers (mean ±SD blood Se concentrations = 24±12 ppb), labeled doses of 0.025 mg Se/lb (0.055 mg/kg) were found to significantly elevate blood Se for one month, but concentrations never reached blood target levels of 100 ppb or higher.[35] These studies suggest that currently approved injectable Se products, although beneficial for about one month for treatment/prevention of NMD, are not effective for long-term supplementation of cattle consuming a Se-deficient diet. Higher dosages of injectable Se of 0.091 mg/lb (0.2 mg/kg) at monthly intervals are recommended by some studies,[17] but are extra-label in the US. Erroneous injections of Se at 8-10 times the recommended dosage, that is 0.227 mg/lb (0.5 mg/kg) of body weight, were shown to be lethal to cattle six months of age.[53] Thus, because the safe range between sufficiency and toxicity is quite narrow, an estimation of the herd status is warranted to avoid excess or inadequate supplementation with injectables.

Implications and Conclusions

Severe Se deficiency is still encountered in cattle and can result in myopathy or other conditions in spite of adequate vit E status. Veterinary diagnostic laboratories can measure Se concentrations in serum or blood of cattle, and can relate these to the likelihood of association with disease. Additionally, many convenient methods are available to supplement cattle with Se to reduce the likelihood of deficiency. Recent advances in understanding Se biochemistry suggest we need to reconsider other problems that may be associated with suboptimal Se status in cattle. We may then be able to better understand potential effects on growth, fertility and control of infection. A better understanding of the association between these conditions and Se status may persuade the livestock industry to supplement cattle with Se when levels are inadequate in the diet.

Acknowledgements

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Footnotes

[a]Sel-Plex 50, Alltech Inc., Nicholasville, KY
[b]ALKOSEL 1000, Primalco Ltd Biotec, Rajamaäki, Finland

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Abstract

Aetiology of Clinical Mastitis in Six Somerset Dairy Herds

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Clinical mastitis was monitored in six Somerset dairy herds for one year. The herds all had three-month geometric mean bulk milk somatic cell counts of less than 250,000 cells/ml. *Escherichia coli* was the predominant pathogen isolated on all the farms and in all months of the year. Environmental pathogens accounted for 61.4 percent of all cases of clinical mastitis and for 79.3 percent of the mastitis cases in which an aetiological agent was identified. The mean annual incidence was 41.6 cases per 100 cows (range 14 to 75). Affected cows suffered a mean of 1.5 cases and 16.4 percent of quarters suffered at least one repeat case. Mastitis due to *E coli* was more severe than mastitis due to other causes and it tended to be more severe in early lactation and during the housing period. Mastitis was significantly more severe (grades 2 and 3) in the herd with the lowest bulk milk somatic cell count and in the herd which was kept indoors throughout the year than in the other four herds. Mastitis was fatal in 2.2 percent of cases and resulted in the death of 0.6 percent of the lactating cows.