Selenium Nutrition of Cattle: An Update[1]

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Research regarding the nutritional importance of selenium (Se) has changed markedly over the last 75 years. In the 1930's Se was identified as the toxic agent causing alkali disease in animals. In the 1940's and early 1950's research was conducted to identify the specific seleno-compounds causing toxicity and to develop prophylactic and treatment schemes for selenium toxicity. Selenium research changed completely after Schwarz and Folz (1957) reported that Se was an essential nutrient for laboratory animals. Research quickly focused on domestic animals and Se deficiency was identified as the cause of white muscle disease (Muth et al., 1958). In 1979, after years of research showing many beneficial effects of Se supplementation of domestic animals, the U.S. Food and Drug Administration published regulations that legalized Se supplementation of diets for beef and dairy cattle. The current U.S. FDA regulation was published 1987 and allows ruminant diets to be supplemented with 0.3 ppm Se from either sodium selenite or selenate (selenized yeast is also approved in Canada). This paper will 1) briefly review the effects of Se on cattle, 2) discuss factors affecting bioavailability of Se, and 3) present recommendations to maintain proper Se status of cattle.

Responses to Selenium Supplementation

Growth responses to Se supplementation have generally been nonsignificant or slightly positive for beef cattle. Likewise, milk production rarely increases when Se deficient animals are supplemented with Se. Although substantial production responses are rarely observed when Se is supplemented, Se supplementation can improve animal health.   Research with dairy and beef cattle have found the following clinical responses to Se supplementation of Se deficient diets: 1) reduced prevalence of retained fetal membranes (Harrison et al., 1984), 2) reduced severity and prevalence of clinical mastitis (Smith et al., 1997), 3) reduced milk somatic cell counts (SCC) (Weiss et al., 1990), and 4) reduced calf mortality (Spears et al., 1986). In addition to clinical responses several studies (mostly with dairy cattle) have reported that various measures of immune function were improved with Se supplementation (Hogan et al., 1990; Maddox et al., 1991). An important factor common to all the clinical and immunological experiments is that the control diets would be considered deficient in Se

and treatment diets generally contained either 0.1 or 0.3 ppm supplemental Se. Supplementation of Se to deficient diets often elicits a positive response; however, supplementation of Se-adequate diets should not be expected to produce additional clinical benefits.

 Retained fetal membranes (retained placenta). A survey of high producing herds in the U.S. found that about 9% of all calvings resulted in retained fetal membranes (RFM) (Kellogg et al., 2001). The estimated total cost associated with RFM range from about $100 to $280/case (Joosten et al., 1988; Kelton et al., 1998). The majority of studies in which the control diet contained less than 0.1 ppm total Se have found that the incidence of RFM is reduced when Se is supplemented via the diet (0.1 to 0.3 ppm) or via injections (about 50 mg of Se given 21 d prepartum), but Se supplementation had limited or no effect in studies in which the control diet contained more than 0.1 ppm Se. The effect of Se supplementation on RFM is influenced by the vitamin E status of cows. The incidence of RFM for cows that have low vitamin E status often is not influenced by Se supplementation (Harrison et al., 1984). A likely mode of action is via effects of Se (and other antioxidant nutrients) on arachadonic acid metabolism. Recently, Kimura et al. (2002) reported that neutrophils from cows with RFM had significantly less killing ability than neutrophils from cows without RFM. Neutrophils from Se-deficient cows have lower killing ability than neutrophils from Se-adequate cows (Hogan et al., 1990).

 Mastitis. Mastitis is still an extremely prevalent and expensive problem for dairy farmers. On well-managed farms, approximately 50 cases of clinical mastitis can be expected per 100 cow-years (assuming 305 d lactation). The total costs associated with clinical mastitis range from about $100 to $140 per case (Hoblet et al., 1991). Compared with cows receiving no supplemental Se, dietary supplementation or injections of Se has reduced the prevalence and severity of clinical mastitis from natural infections and experimental *E. coli* challenge (Erskine et al., 1989; Smith et al., 1984). Selenium supplementation did not affect the response to *Staphylococcus aureus* challenge (Erskine et al., 1990). The positive effect of Se supplementation on clinical mastitis is probably mediated via effects of Se on neutrophils and other immune cells. In the U.S., studies have found a negative correlation between SCC and Se status (Erskine et al., 1987; Weiss et al., 1990), but no relationship was observed in a study from New Zealand (Grace et al., 1997). One likely difference between the U.S. and New Zealand studies is the vitamin E status of the cows. Cows on the New Zealand study grazed high quality pasture (expected to have very high intakes of vitamin E); cows in the U.S. studies were in confinement and vitamin E intakes probably were much lower.

Bioavailability of Selenium

 Selenium metabolism. The active form of Se in selenoenzymes is selenocysteine (Se-cys). Se-cys is identical to the amino acid, cysteine (cys) except that Se replaces sulfur (S). A specific tRNA exists for Se-cys, therefore cys cannot

replace Se-cys during synthesis of selenoproteins. In animals, selenate is reduced to selenite (this can occur in the rumen). Through a series of reactions, selenite is reduced to selenide which is then used in the synthesis of Se-cys. Selenomethionine (Se-met) is identical to the amino acid methionine except that Se replaces the S. A specific tRNA has not been identified for Se-met and it appears that no discrimination occurs between met and Se-met during protein synthesis. Much of the Se found in feeds is in the form of Se-met and to a lesser extent Se-cys. Based on rat studies, Se-cys that is consumed is not directly incorporated into selenoproteins but rather must be degraded to selenide and resynthesized into Se-cys (Sunde and Hoekstra, 1980). Se-met can be degraded to yield selenide which then can be incorporated into Se-cys.

Inorganic selenium sources. The only approved selenium supplements in the U.S. are sodium selenite and sodium selenate. In Canada, sodium selenite, sodium selenate, and selenized yeast are approved. This discussion will be limited to those products. Measuring the bioavailability of Se by ruminants is extremely difficult and reliable data are scarce. Relative bioavailability can be measured by comparing concentrations of Se or enzyme activity (usually glutathione peroxidase, GSH-px) in different tissues or fluids or by comparing apparent absorption (Se intake minus fecal and urinary excretion) when different Se compounds are fed. Any differences observed may or may not relate to different clinical responses (e.g., incidence of RFM). One study using sheep fed high amounts of Se found that selenium from sodium selenate increased tissues concentrations of Se about 30% higher than the same amount of Se from sodium selenite (Henry et al., 1988). However, studies with dairy cows and heifers using diets with typical Se supplementation rates (Ortman et al., 1999; Ortman and Pehrson, 1999) found little difference between selenite and selenate based on GSH-px activities and Se concentrations in blood and milk. Overall, differences between bioavailability of sodium selenite and sodium selenite for dairy cows appear to be small or insignificant. Based on Se metabolism (i.e., selenate is converted to selenite and then follows the same pathways), few differences in bioavailability should be expected.

Selenized yeast. The Se from selenized yeast (Se-yeast) differs substantially from Se from selenate or selenite. The vast majority of Se in Se-yeast is in the form of selenomethionine (Se-met). Absorption of met (and Se-met) from the gut is usually quite efficient. In a study with sheep using radioisotopes, absorption of Se from Se-yeast was higher than absorption of Se from selenite (47.5% vs. 43.8%) (Koenig et al., 1997). Based on these results, bioavailability of Se from Se-yeast was about 8% higher than that from selenite. When based on concentrations of Se in whole blood or plasma of cattle, the relative bioavailability of Se-yeast compared with selenite (or selenate) has ranged from about 100% (i.e., equal availability) to about 130% (Awadeh et al., 1998; Fisher et al., 1995; Knowles et al., 1999; Ortman et al., 1999; Ortman and Pehrson, 1997; 1999). One study (Malbe et al., 1995) reported an extremely high relative bioavailability for Se-yeast of 189% of selenite. The average relative bioavailability of Se-yeast across studies based on blood concentrations is 122% (SD = 31) of selenite based on plasma and whole blood Se (Figure 1). Without the extremely high value reported by Malbe et al., the mean relative bioavailability of Se-yeast is 112% (SD = 9)

of selenite. When whole blood GSH-px is used as the criteria, relative bioavailability of selenite and Se-yeast are about equal (Awadeh et al., 1998; Knowles et al., 1999; Ortman et al., 1999; Ortman and Pehrson, 1997; 1999). The Se concentration of milk is almost always significantly higher when Se-yeast is fed than when selenite is fed. Across all studies milk Se concentrations when Se-yeast was fed averaged 200% (SD = 57) of the concentrations when selenite was fed (Figure 2).
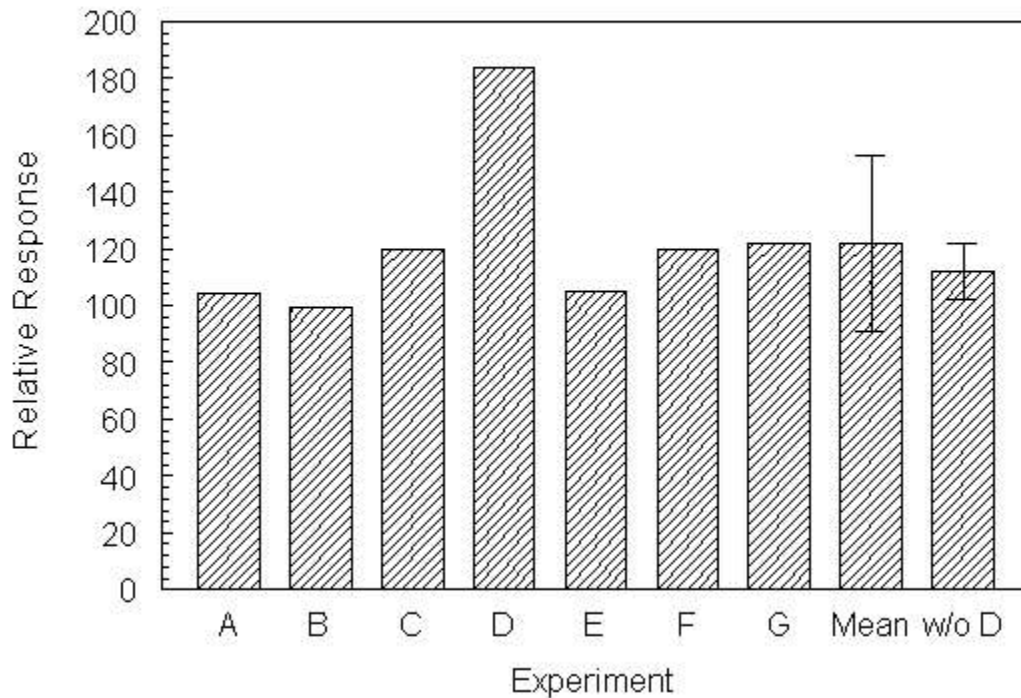

Figure 1. Relative effect of Se-yeast to selenite (equal dietary Se within an experiment) on concentrations of Se in whole blood or plasma (Expt. G). The values shown are the concentration of Se in whole blood (or plasma) of cows fed Se-yeast divided by concentration of cows fed selenite times 100. Source of data: A (Awadeh et al., 1998), B (Fisher et al., 1995), C (Knowles et al., 1999), D (Malbe et al., 1995), E (Ortman and Pehrson, 1997), F (Ortman and Pehrson, 1999), and G (Weiss, unpublished).

The vast differences among measures of relative bioavailability (absorption from the gut, plasma and whole blood Se, and milk Se) are easily explained. The Se-met in Se-yeast is probably more available (about 10 to 15%) for absorption than the Se in selenite. This would explain the differences between Se-yeast and selenite with respect to blood Se concentrations. The Se in yeast is probably absorbed predominately as Se-met. The mammary gland requires substantial amounts of met for protein synthesis. The Se-met is absorbed by the mammary gland and is simply used as a met source. Since milk is a terminal pool (once milk protein is made it is not available to the cow), milk will continue to accumulate Se as Se-met is absorbed and used for protein synthesis. Clinical data are not available comparing health effect of Se-Yeast to selenite. Based on my interpretation of the data, with respect to clinical responses, I would expect Se-yeast to be on average about 10to 15% more effective than an equal amount of Se from selenite or selenate. Because of the substantial increase in milk Se when Se-yeast is fed, the biggest benefit to Se-yeast supplementation may be to increase Se intake by humans consuming the milk.

Factors Affecting Selenium Utilization

Several minerals and vitamins have been shown to affect Se absorption and utilization, but most of the studies have used laboratory animals. In nonruminants, diets deficient or in excess in copper reduce Se status. Research conducted with cattle and sheep, however, has shown that neither marginal Cu deficiency nor supplementation of reasonable amounts of Cu (less than 20 ppm supplemental Cu) affected Se status when selenite was the Se source (Koenig et al., 1991; White et al., 1989a; White et al., 1989b). Selenium absorption by rats was reduced about 25% when dietary Zn was increased from 5 to 20 ppm (House and Welch, 1989), however data concerning possible interactions between Zn and Se in ruminants are not available. At very high dietary Se concentrations (approximately 1 ppm from selenate) supplementation of 500 IU of vitamin E/d reduced plasma Se and whole blood GSH-px in Jersey cows but no effects were observed when diets with 0.3 ppm Se were fed (Weiss et al., 1993).

Two macrominerals, calcium and sulfur, have been shown to influence Se utilization in cattle. In calves, dietary Ca (ranged from 0.17 to 2.35%) had a minor affect on Se absorption and Se concentration in tissues (Alfaro et al., 1987). Maximum Se absorption occurred when diets contained 1.5% Ca. In dry dairy cows, maximum Se absorption occurred when diets contained 0.9% Ca (ranged from 0.4 to 1.3%) (Harrison and Conrad, 1984). Selenium absorption was about 40% higher when diets contained 0.9% Ca than when they contained either 0.5 or 1.3% Ca. In sheep, increasing dietary sulfur from 0.05% to 0.24% (from sodium sulfate) linearly reduced Se absorption (Pope et al., 1979). Ivancic and Weiss (2001) also found a linear reduction in Se digestibility by dairy cows as supplemental S (from a mixture of magnesium and calcium sulfate) increased from 0 to 0.4% (Figure 3). In that study, only cows fed no supplemental sulfate and 0.3 ppm Se (from selenate) were in positive Se balance and after 112 d, S supplementation reduced plasma Se concentrations. Conversely in an 85 d trial with growing beef cattle (initial weight 210 kg or 460 lbs) no differences in whole blood Se

were observed between calves fed no supplemental S or those fed approximately 0.3% added S from calcium sulfate (Khan et al., 1987). Van Ryssen et al. (1998) reported that feeding 0.2% added S from sodium sulfate reduced liver Se but did not affect plasma Se. A possible Khan et al. did not observe an effect of S on blood Se was that the experiment did not last long enough. Gant et al. (1998) reported no adverse effects on Se when dairy cows were fed a diet with 0.4% added S from sulfate the last 21 d of gestation. Elevated intake of S from sulfate (this may also be true for high sulfate water) for long periods of time (several months) appears to generally reduce Se status of ruminants fed selenite or selenate.
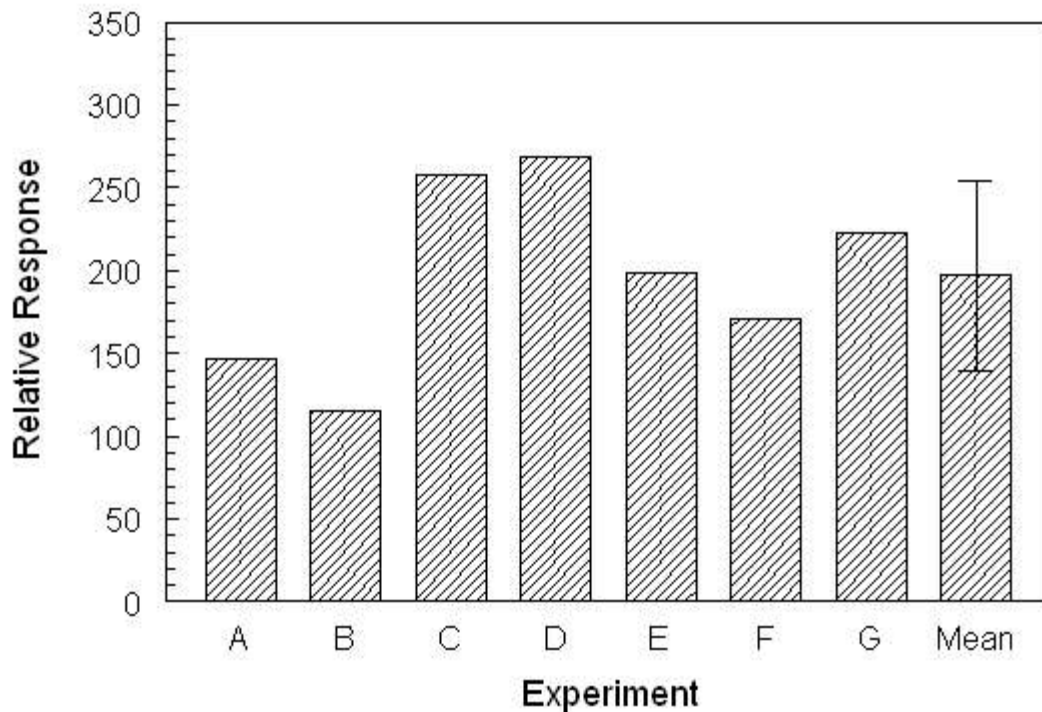

Figure 2. Relative effect of Se-yeast to selenite (equal dietary Se within an experiment) on concentrations of Se in milk or colostrum (Expt A). The values shown are the concentration of Se in milk from cows fed Se-yeast divided by concentration for cows fed selenite times 100. Source of data: A (Awadeh et al., 1998), B (Fisher et al., 1995), C (Knowles et al., 1999), D (Malbe et al., 1995), E (Ortman and Pehrson, 1997), F (Ortman and Pehrson, 1999), and G (Weiss, unpublished).
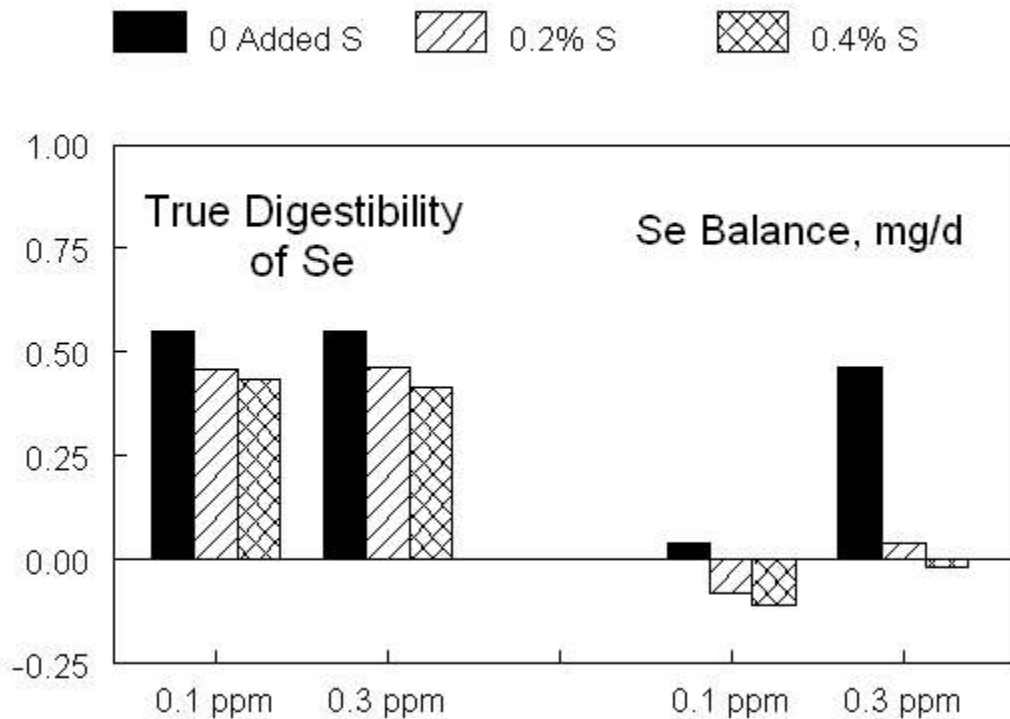
Figure 3. Effect of increasing dietary sulfur from a mixture of calcium and magnesium sulfate) on true digestibility (as a proportion) of Se and Se balance in lactating dairy cows when cows were fed either 0.1 or 0.3 ppm supplemental Se from selenate (Ivancic and Weiss, 2001).

Recommendations and Conclusions

1. Large production responses (either growth rate or milk yield) to supplemental Se are unlikely, however, reduced health disorders (retained fetal membranes, metritis, mastitis) are likely.

2. In most situations, diets containing between 0.2 and 0.4 ppm total Se (supplemental Se and the Se contained in the feeds) appear adequate to maintain health of cattle. In

most dairy areas of North America, diets for cattle should be supplemented with between 0.1 and 0.3 ppm Se from an approved source.

3. Sodium selenite and sodium selanate have similar bioavailabilities and probably have similar health effects.

4. Based on gut absorption and blood Se concentrations, selenized yeast is probably about 10 to 15% more available than selenite. Clinical effects (i.e., health) of selenized yeast compared with selenite are not known.

5. Selenized yeast greatly increases the concentration of Se in milk and this could improve Se status of dairy consumers.

6. Bioavailability of Se from selenite and selenate are reduced by long term consumption of large amounts of sulfur from sulfate.

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