Effect of prepartum parenteral supplementation of pregnant beef cows with selenium/vitamin E on cow and calf plasma selenium and productivity

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Interest in selenium (Se) as an essential micronutrient began with the discovery that Se was the so called "factor 3" in bread yeast, which was shown to be important in preventing hepatic necrosis in rats (1). Supplementation with Se has been associated with trends toward increased calf weight gains, decreased calf death losses (2), reduced incidence of retained placentas, and improved reproductive efficiency in herds (3). Selenium also plays a major role in maintaining cell membrane integrity (4). Vitamin E deficiency alters the stability of cell membranes and can lead to damage of these membranes (5); Se and vitamin E can replace part of the requirement for each other (4). It is possible that a marginal to deficient Se/vitamin E status in the cow may cause a greater movement of Se across the placenta due to decreased membrane integrity, possibly to supplement the fetus. If this occurs, reduced production in the cow (weight changes, re-breeding) may be more likely to occur than reduced calf vigor or weight gain. However, it has been reported that, although Se transfer in sheep was bi-directional across the placenta, the transfer rate was reduced in both directions when the dam was Se deficient (6). The effect of Se/vitamin E deficiency in beef cows on Se transfer across the placenta is not known.

We conducted the work reported herein to determine the effect of a single Se/vitamin E injection of the pregnant beef cow on the Se status of the cow and calf at parturition and on their subsequent performance traits.

On March 2, 1987 two-hundred-and-twenty-six cows (Hereford, Hereford × Angus, Hereford × Simmental, and some three-way crosses) from the University of Saskatchewan Termuende Research Station, Lanigan, were allocated to two groups: Se-E and control. The cows had previously been determined to have a low to marginal vitamin E status of 0.274 ± 0.084 mg/100 mL plasma. Normal levels are >0.4 mg/100 mL (5). The two groups were balanced for breed and age. Blood samples were taken from all cows via the jugular vein into heparinized vacuum tubes. One-hundred-and-ten cows (611 ± 70.9 kg live weight) in the Se-E group received a subcutaneous injection of 6 mL Dystosel DS containing 6 mg Se as sodium selenite, 136 IU vitamin E and 15 mg benzyl alcohol per mL (rogar/STB, Division of Pfizer, Kirkland, Quebec) according to the manufacturer's instructions and allowing for the weight of the fetus; 116 cows (607 ± 63.5 kg live weight) in the control group remained unsupplemented. Calving began March 7 and ended May 4, and blood samples were again collected from the cows and calves within three days of calving. The cows were fed 11.3 kg of alfalfa-brome hay daily with a free choice mineral supplement that contained Ca, P, NaCl, Cu, Mn, Zn, Co, and I but no Se or vitamins. From November 1986, to February 1987, all cows had been given a monthly injection of 5 mL vitamin supplement containing 500,000 IU vitamin A, 75,000 IU vitamin D3, and 50 IU vitamin E per mL.

There was little benefit from injecting cows having low vitamin E status with a selenium-vitamin E preparation

Plasma samples from the cows and calves were analyzed for Se concentration, using the method described in the applications manual for a Zeeman modulated atomic absorption spectrophotometer (Perkin-Elmer Corporation, Norwalk, Connecticut, USA), to determine Se status prior to injection and at parturition. The performance traits measured included the weights of cows at the time of injection, of cows and calves at parturition and every 28 d after the last cow had calved, calf vigor at birth, incidence of diarrhea in the calves, time between parturition and the expulsion of the placental membranes, and conception rate in the cows.

Plasma Se concentrations, live weight data, and time for expulsion of placental membranes were compared between groups by Student's *t*-test. Calf vigor, incidence of diarrhea, and conception rate data were compared between groups by chi-square analysis.

Plasma Se concentrations did not differ between Se-E and control cows either before injection or at calving (Table 1). Similarly, plasma Se concentrations did not differ significantly between calves from Se-E and control cows at birth (Table 1). Plasma Se concentrations increased from the initial sample to the sample taken at calving for both groups of cows ($p<0.05$). There were no significant differences between cow groups for length of time taken to expel the placental membranes after calving (1.8 ± 1.2 h for both groups), placental membranes in all cows were expelled within 8–12 hours of calving, so none were considered to be retained.

Observations were made on calf vigor at birth to determine the degree of unthriftiness of calves. There

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Table 1. Plasma selenium concentration (μg/mL) of cows immediately before injection and of cows and calves at birth (mean ± SD)

	Cows		Calves	
Sample time	Control	Se-E	Control	Se-E
Before injection	0.091[a] ± 0.041	0.095[a] ± 0.049	N/A	N/A
At birth	0.138[b] ± 0.208	0.158[b] ± 0.060	0.055 ± 0.025	0.077 ± 0.058

[a,b]Values with different letters are significantly different ($p < 0.05$)
N/A = Not available

were no significant differences between the two groups of calves. Three calves died in the injected group compared with nine in the control group but the difference was not significant ($\chi^2 = 2.84$; $p < 0.10$). In the injected group, one calf died following a dystocia, one as a result of atypical interstitial pneumonia, and one from unidentified causes. In the control group, two calves were born moribund (one was euthanized and one died naturally), two were premature and stillborn, one died of unidentified causes, one of pneumonia, and three of diarrhea. There was no significant difference between groups in the number of calves that were treated for diarrhea. In the Se-E group, 25 calves were treated for diarrhea, with two receiving treatment again 5 d later, whereas 29 calves from the control group were treated for diarrhea. The three deaths due to diarrhea in the control group occurred at this time, while there were no deaths in the treatment group attributed to diarrhea. No calves were treated once they were on pasture.

There was no significant difference in the mean birth weight of calves from injected cows (39.8 ± 7.1 kg) and calves from control cows (41.2 ± 6.1 kg). Calf average daily gains from birth to weaning were 1.12 ± 0.18 kg/d in the injected group and 1.09 ± 0.17 kg/d in the control group; this difference was not significant. Since the calves were weighed every 28 d, each period was examined from birth to each of the weigh dates and from one weigh date to the next. There were no significant differences for any of the periods. Weaning weights averaged 224 ± 37 kg for the Se-E group and 221 ± 35 kg for the control group.

Cows in both groups lost weight during the breeding period, probably because of every hot, dry weather. However, the Se-E injected cows lost less weight than the control cows during the first four weeks of the six week breeding season from June to July (−0.17 ± 0.08 vs −0.32 ± 0.08 kg/d, respectively; $p < 0.05$). The pregnancy rate was 85.5% in the Se-E injected compared with 78.4% in the control cows, but this difference was not significant ($\chi^2 = 1.87$; $p < 0.10$).

The initial plasma Se levels for cows (0.091 ± 0.041 and 0.095 ± 0.049 μg/mL) for the control and Se-E groups, respectively, indicated that their status was adequate at the start of the experiment, that is, >0.07 μg/mL (7), although the initial vitamin E status was low. Thus, this study allowed us to determine if Se supplementation could benefit cows with a vitamin E deficiency. Commercial vitamin preparations contain only small amounts of vitamin E, and massive doses of vitamins A and D would be injected if these preparations were used to correct a vitamin E deficiency. The difference in plasma Se between groups was not significant for both cows and calves at parturition. There was a significant ($p < 0.05$) increase in plasma Se concentration from the initial samples on March 2 to the samples taken at parturition. This suggests mobilization of Se from body stores for lactation in the cows. Similar though less marked results have been reported in dairy cows (8).

Humoral immune response to bacterial and viral antigens has been reported to be improved by vitamin E-Se supplementation of several species (9,10) and so has vigor in the young calf (2). A significant reduction in death loss due to Se-E treatment has also been reported (2). In that report, most of the deaths in the control calves were caused by diarrhea. The observational data in this study showed no difference in vigor at birth. There were no differences in the number of calves treated for clinical signs of diarrhea in either group, although 33% of the death loss in the control group was attributed to diarrhea while none of the deaths in the treated group was related to diarrhea.

Cow weights and gains were similar for both groups at all times except during the first four weeks of the breeding season, during which time the Se-E group lost less weight than the control group ($p < 0.05$). This difference appears to have been reflected in an increased pregnancy rate in the treated group (85.5%) compared with the control group (78.4%), but this difference was not significant ($p < 0.10$). It has been suggested that embryonic mortality at the time of attachment, that is, three to four weeks after conception, was the main cause of reproductive failure in Se deficient ewes (10). However, it is not possible to speculate whether the apparent difference between the two groups of cows in our experiment was due to a difference in embryonic mortality or the incidence of estrus, which may have been associated with the differences in weight change during the breeding period, or both.

Since the cows were low in vitamin E status and adequate in Se status at the start of the study, it is possible that greater responses may have been obtained if parenteral supplementation with vitamin E was possible without giving concomitantly high doses of vitamins A and D or Se with the use of presently available commercial products.

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References

1. Schwarz K, Foltz CM. Selenium as an integral part of Factor 3 against necrotic liver degeneration. Am Chem Soc 1957; 79: 3292-3293.
2. Spears JW, Harvey RW, Segerson EC. Effects of marginal selenium deficiency and winter protein supplementation on growth, reproduction and selenium status of beef catte. J Anim Sci 1986; 63: 586-594.
3. Harrison JH, Hancock DD, Conrad HR. Vitamin E and selenium for reproduction of the dairy cow. J Dairy Sci 1984; 67: 123-132.
4. Diplock AT. Metabolic aspects of selenium action and toxicity. CRC Crit Rev Toxicol 1976; 4: 271-329.
5. Newberne PM, Corner MW. The Vitamins. In: Kaneko JJ, ed. Clinical Biochemistry of Domestic Animals. 4th ed. San Diego:Academic Press, 1989: 796-833.
6. Shariff MA. Kinetics of selenium metabolism in the ewe and fetal lamb. (Dissertation). Vancouver, British Columbia: University of British Columbia, 1987.
7. Puls R. Veterinary Trace Mineral Deficiency and Toxicity Information. Ottawa: Information Services, Agriculture Canada, Publication 5139, 1981: 76-78.
8. Kappel LC, Ingraham RH, Morgan EB, *et al.* Selenium concentrations in feeds and effects of treating pregnant Holstein cows with selenium and vitamin E on blood selenium values and reproductive performance. Am J Vet Res 1984; 45: 691-694.
9. Levander OA. Selenium. In: Mertz W, ed. Trace Elements in Human and Animal Nutrition. 5th ed. Vol 2. Orlando: Academic Press, 1986: 209-279.
10. Hartley WJ. Selenium and ewe fertility. Proc NZ Soc Anim Prod 1963; 23: 20-27.