

Physical, hematologic, biochemical, and immunologic effects of supranutritional supplementation with dietary selenium in Holstein cows

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Objective—To measure responses of cows to supplemental Se intake in excess of nutritional requirements, but lower than recognized toxic dosages.

Animals—24 healthy adult Holstein cows.

Procedure—Cows were allotted to 4 groups and fed sodium selenite to provide 0, 3, 20, or 50 mg of Se/cow/d for 90 days. Subsequently, the dosage for the group receiving 50 mg/cow/d was increased to 100 mg/cow/d for 28 d. Blood, liver specimens, feces, and urine were obtained at points during the trial.

Results—Serum and blood Se concentrations in groups receiving 20 or 50 mg/cow/d increased over time, compared with controls ($P < 0.01$). Increasing supplemental Se intake to 100 mg/cow/d further increased serum and blood Se concentrations ($P < 0.05$). Urine, fecal, and liver Se concentrations increased more markedly in response to treatment than did those of serum or blood. No effect of Se treatment was seen on blood cell counts or serum activities of hepatocellular enzymes. Likewise, neither titer response to rabies vaccination nor lymphocyte blastogenic response to nonspecific mitogens was affected by Se treatment. Objective or subjective physical signs of Se toxicosis were not observed at any Se dosage.

Conclusions—Inorganic Se intakes of up to 50 mg/d for 90 days or 100 mg/d for 28 days by adult Holstein cows do not affect the variables measured.

Clinical Relevance—Intakes of Se as sodium selenite in amounts 10 to 30 times the nutritional requirements are unlikely to cause health problems in adult cows. Urine and feces are good test samples for detection of Se supplementation greater than requirements. (*Am J Vet Res* 1997;58:760–764)

The minimal lethal dosage of inorganic Se required to induce acute toxicosis appears to be in the range of 3 to 5 mg/kg of body weight for most species,¹⁻³ but may be twice that for cattle.^{3,4} The dosage necessary to cause chronic Se intoxication appears variable, and is probably affected by the form of Se (valence state, organic vs inorganic), characteristics of the diet, route of exposure, and previous Se exposure of the animal, among other factors.⁵ The dosage of Se as sodium selenite necessary to cause chronic toxicosis in cattle ap-

pears to be in the range of 0.25 to 0.5 mg/kg over a period of several weeks.^{6,7}

The adverse effects of dietary Se in cattle, in dosages greater than those required but less than those associated with chronic toxicosis, have not been investigated by clinicopathologic or immunologic means. The primary major objectives of the study reported here were to evaluate clinicopathologic variables that might indicate low-level Se toxicosis in cattle, and to evaluate various methods of detecting Se intake in excess of nutritional requirements and legal limitations. A secondary objective was to evaluate the effects of supranutritional, but not overtly toxic, intake of Se on immune function. Selenium is known to have negative as well as positive effects on the immune system.⁸

Materials and Methods

Cows and diets—Twenty-four adult nonlactating Holstein cows of unknown age were housed in individual tie stalls. Cows were fed a diet of mixed grass hay ad libitum. The hay for the entire study was obtained from a single source in south central Michigan, where Se concentration is typically < 0.2 mg/kg. The hay was supplemented daily with 0.45 kg of a ground corn-based concentrate to which treatments were added to provide 0, 3, 20, or 50 mg of supplemental Se. Selenium was added as sodium selenite. The concentrate mix contained trace mineralized salt and vitamins A, D, and E in concentrations adequate to meet NRC recommendations for nonlactating cows.⁹ Prior to receiving the treatment diets, all cows were fed the control (no supplemental Se) diet for 2 weeks, and serum Se concentration was determined. These initial values were used to block cows into treatment groups so as to minimize variation among group means at initiation of the study. Each cow was assigned to 1 of 4 treatment groups (A through D) receiving 0, 3, 20, or 50 mg of supplemental Se/cow/d, respectively. Cows were fed the treatment-containing concentrate portion of their diet at 7 AM and were not given hay until the concentrate had been consumed. The experimental diets were fed to all groups for 90 days. Group-D cows continued on study, receiving 50 mg/d for an additional 10 days, at which time the Se supplement was increased to 100 mg/d for 28 days. Supplemental Se was then discontinued in these cows so that changes in response to withdrawal of Se could be monitored.

Sample collection—All blood samples were taken from the coccygeal vessels between 6 and 7 AM, before the daily concentrate supplement was fed. Blood samples for hematologic analysis, enzyme activity determinations, and lymphocyte response testing were analyzed on the day of collection. Antemortem liver biopsy specimens were taken as described.¹⁰ Hoof growth rate was determined by grooving the hoof near the coronet at the start of the study, then monitoring the rate of progression of the groove down the hoof. Body condition scoring was done as described by Edmonson et al.¹¹ To allow determination of humoral immune response, cows were vaccinated against rabies at 45 and again at 73 days into the study.^a Urine and fecal samples were taken as voided, with no attempt to quantify total daily excretion.

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