Reproductive Response of Ewes Fed Alfalfa Pellets Containing Sodium Selenate or Astragalus Bisulcatus as a Selenium Source

KE Panter and LF James
United States Department of Agriculture, Agricultural Research Service,
Poisonous Plant Research Laboratory, 1150 E 1400 N, Logan, Utah 84321

and

HF Mayland
United States Department of Agriculture, Agricultural Research Service,
Snake River Conservation Research Center, Kimberly, Idaho 83341

ABSTRACT. Selenium fed to open cycling ewes in the form of sodium selenate or Astragalus bisulcatus (a selenium accumulator plant) at 24 or 29 ppm selenium, respectively, in alfalfa hay pellets did not alter the estrous cycle length, estrus behavior, progesterone or estrogen profiles, pregnancy rate or outcome of parturition ($P>0.05$). There was wool loss in some ewes fed seleniferous pellets and the mean whole blood selenium levels were 0.45, 1.3 and 2.4 ppm, respectively, for control, A bisulcatus and sodium selenate; however, ewe condition and appearance remained good. All lambs appeared normal and the number of lambs born and the individual and total lamb weight averages were not significantly ($P>0.05$) different between treatment groups and control group.

Selenium has been recognized as a natural toxicant since the early 1930s when seleniferous forage was implicated in disease processes widespread in North America. Toxic or potentially toxic forbs and grasses grow on seleniferous soils on many ranges in vast areas of the world.

In the late 1950s, trace amounts of selenium were shown to have important nutritional and metabolic functions in many animal species (1). In 1960, Drake et al (2) reported that oral administration of small quantities of selenium either as selenite or selenate prevented "white muscle disease" in sheep and improved the growth rate of weaned lambs. As selenium deficiencies have been an important concern for livestock producers, so have selenium excesses in some regions of the country.

Selenium excesses are involved in numerous disease processes, including reproductive disorders. Olson (3) suggested that the effect of excess dietary selenium on reproduction had a greater economic impact on animal agriculture than overt selenium toxicity. This influence is supported by reports in rats (4-6), chickens (7), mice (8), pigs (9) and cattle (10,11) where excessive but not clinically toxic levels of selenium impaired reproduction. It has been reported that reproductive failure in sheep and cattle occurs if selenium is ingested in excess of 10 ppm (5,11). Glenn et al (12) reported no apparent effect on reproductive performance in ewes fed high doses of sodium selenate. However, Dinkel et al (11) reported a dramatic decrease in reproductive efficiency in cattle grazing grasses high in selenium. It was determined that stage of plant growth and development was an important factor in the severity of reproductive impairment.

Studies describing the effects of high-level exposure of dams during pregnancy to selenium have been reviewed by Combs and Combs (13). They documented embryonic and fetal selenium-induced death in chickens, mice, pigs and rats, as well as reduced growth rates in chicks, mice and pigs. Malformations and poor hatchability have also been described in chickens (13), and eye extremity and reproductive organ defects were observed in sheep from field cases grazing seleniferous pastures in Wyoming (5).

We conducted this study to determine if selenium, in the form of sodium selenate or the plant form (Astragalus bisulcatus), fed free choice in a hay pellet would adversely affect reproductive performance in ewes. We observed estrous cycle length, estrus behavior, and progesterone and estrogen profiles throughout the estrous cycle and the subsequent effects of selenium on pregnancy rate, lambing rate and birth weight. We observed lambs for any abnormal behavior and developmental anomalies.

MATERIALS AND METHODS

Fifteen yearling Suffolk-Columbia-Targhee crossbred ewes which were non-pregnant and cycling, and weighing between 50 and 80 kg, were divided into 3 groups of 5 ewes/group. Ewes were weighed and assigned to groups to balance the groups by weight. Group 1 averaged 67±10 kg/ewe and received alfalfa pellets containing selenium as sodium selenate. The amount of sodium selenate added to the alfalfa for pelleting was calculated to equal 25 ppm selenium; the actual amount on analysis was 24 ppm selenium. Group 2 averaged 66±9 kg/ewe and received alfalfa pellets containing selenium in the form of Astragalus bisulcatus. The amount of A bisulcatus added to the alfalfa for pelleting was calculated to provide 25 ppm selenium based on 300 ppm selenium in the A bisulcatus; the actual amount in the pellet upon analysis was 29 ppm selenium. Group 3 averaged 66±10 kg/ewe and

received alfalfa pellets only (0.8 ppm selenium). Selenium levels were measured by a fluorometric method (14). Consumption of pellets was monitored and control pellets were adjusted to match the consumption level of the treatment group which consumed the least amount of feed, thus keeping energy intake equal among all groups.

The A bisulcatus was collected in the Shirley Basin near Medicine Bow, Wyoming, in 1985. The plant material was air dried, chopped, bagged and stored at 20 C until used. Prior to including this plant preparation in pellets, a total selenium analysis by fluorometry (14) indicated 300 ppm selenium on a dry weight basis in the A bisulcatus.

All ewes in each group received their pelleted treatment feed for 34 to 45 d (mean 41 d) before planned exposure to a vasectomized ram. At this time observed estrus was recorded and subsequent estrous cycle length measured. Treatment feeds were continued through this estrous cycle of 18-20 d. At the subsequent estrus all ewes were bred to 2 fertile suffolk rams. The rams were rotated through each pen of ewes twice daily until all ewes were bred. Each ewe was hand-mated to both rams initially and on subsequent days until she was out of estrus (usually 1 d after initial mating). Treatment feeds were continued for 17 to 28 d (mean 22 d) after breeding to the fertile rams. The ewes were fed treatment feeds for a total of 88 d.

Blood samples were drawn daily via the jugular vein from each ewe beginning the first observed day of estrus (day 0), as detected by mating to the sterile ram. This was continued through 1 d after the second observed estrus (average of 18 d). Serum progesterone and 17β-estradiol levels were determined by radioimmunoassay using a no-extraction, solid phase I-125 radioimmunoassay kit (Diagnostic Products Corporation, 5700 West 96th Street, Los Angeles, CA 90045). Coat-A-Count progesterone and 17β-estradiol are highly specific with low cross reactivity to other steroids. Specific cross reactivities are listed in the procedure manual (Diagnostic Products Corporation). Inter- and intra-assay coefficients of variation were 7.85%, 4.7%, 7.53% and 6.7% for estradiol and progesterone, respectively. Kits were validated at 20, 50, 150, 500, 1800 and 3600 pg/ml for estradiol and 0.5, 2.5, 5, 10, 20 and 40 ng/ml for progesterone.

Progesterone and estrogen profiles, number of lambs born and lamb birth weights were analyzed by analysis of variance procedures. Whole blood and dietary selenium was determined fluorometrically (14).

RESULTS AND DISCUSSION

There was no significant difference in progesterone or 17β-estradiol profiles (P>0.05) between treatment groups and controls (Fig 1). There was no difference in estrous cycle length or estrus behavior. Some ewes fed seleniferous diets had minor wool loss on the neck and sides by the 10th w. Feed intake

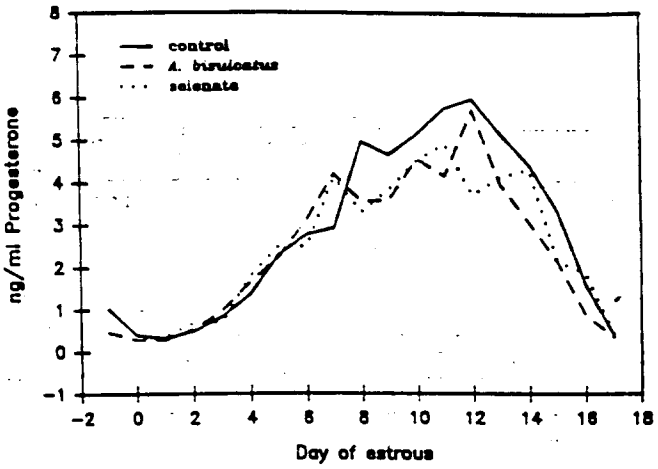

Figure 1. Serum progesterone profiles through the estrous cycle of ewes fed alfalfa pellets containing 0.8 ppm selenium (control), 24 ppm selenium (sodium selenate) or 29 ppm selenium (A bisulcatus).

remained relatively good in all ewes; however, rate of consumption between groups was not compared because all ewes were fed the same amount of pellets as limited by the group consuming the least. Obviously, the control ewes would have eaten more pellets if given free choice. All ewe weights remained constant throughout the selenium feeding period. Blood selenium levels were highest in the group fed sodium selenate pellets (2.4 ppm) followed by the group fed A bisulcatus pellets (1.3 ppm) and controls (0.45 ppm). At lambing there was no reduction in proportion of ewes lambing (Table 1). All lambs appeared normal at birth, and there were no differences in average individual or total lamb weights between groups (P>0.05). In Group 1, 1 lamb from a ewe with triplets died at birth and 1 twin lamb died 3 d after parturition. The cause of death in the first lamb was suffocation from placental membranes over the muzzle, but the cause of death of the second lamb was undetermined.

Excessive levels of selenium have been reported to adversely affect reproduction in many livestock species; however, in sheep the signs of excessive selenium ingestion in our report appear to be different (5,12). Rosenfeld and Beath (5) reported field observations in which 250 malformed lambs were born to a group of 2100 ewes grazing on seleniferous range. The malformations involved the eyes, extremities and reproductive organs.

TABLE 1. LAMBING RESULTS FROM EWES FED SELENIFEROUS FEED

Number of ewes and treatment	Ewes lambed / ewes bred	Lambs born	Lamb wt lbs
5 *A bisulcatus*	5/5	9	9.2
5 selenate	3/5	5	9.4
5 control	3/5	5	11.7

No significant difference between groups for ewes lambing/ewes bred, lambs born or average lamb weights (P> 0.05).

Selenium was implicated as the cause, but our study suggests another factor might be involved.

Selenium excess in animals adversely affects reproductive performance. However, sheep appear to be somewhat resistant to this effect. Sheep did not show the typical clinical signs seen in other animals and appear more adapted to ranges with seleniferous soils.

In sows fed sodium selenite for several months, a reduction in conception rate, birth weights, percentage of live pigs at birth and weaning weights were observed (9). Selenium toxicity not only affected reproduction in swine, but also caused a multitude of pathological conditions that affect growth performance. Severe weight loss, hoof lesions and poliomalacia of the spinal cord and brain stem occur, resulting in paralysis and death (15). In sheep, however, clinical signs did not appear until the terminal stages and in some cases death occurred without any other signs of toxicity. In those cases where clinical signs were observed, the disease was manifest by anorexia, weakness, apathy, respiratory distress, cyanosis and nasal exudation a few hours to 4-5 d before death (12).

In cattle, the literature suggests that levels of 5-25 ppm selenium in forage dramatically reduced reproductive performance (11). In the past, selenium intake of range cattle in many of the South Dakota management systems was at highest level during the breeding season (early June to mid-July), which corresponds to the preheading to maturity stages of range grasses (16). By breeding earlier (May), the number of calves born and weaned over a 5-y period increased by 22.3% and 19.6%, respectively (11). Many ranchers in some of these areas have converted from a cow-calf operation to a steer operation because of the low reproductive performance of cows on such range.

There is a need to further investigate selenium toxicity in grazing livestock and to determine the mechanism of action of selenium on reproductive performance. Sheep and cattle are different in the way excess selenium affects their reproduction. Understanding this difference is important in elucidating the cause-and-effect relationship between selenium and livestock reproductive performance.

REFERENCES

1. Schwarz K, Foltz CM: Selenium as an integral part of factor 3 against dietary liver degeneration. J Amer Chem Soc 79: 3292-3293, 1957.
2. Drake C, Grant AB, Hartley WJ: Selenium and animal health. Part I. The effect of alpha-tocopherol and selenium in the control of field outbreaks of white muscle disease. NZ Vet J 8: 4-8, 1960.
3. Olson OE: Selenium as a toxic factor in animal nutrition. Proc Georgia Nutr Conf: 68, 1969.
4. Munsell HE, Devaney GM, Kennedy MH: Toxicity of food containing selenium as shown by its effect on the rat. Tech Bull No 534, USDA, Washington DC: 25, 1936.
5. Rosenfeld I, Beath OA: Effect of selenium on reproduction in rats. Proc Soc Exptl Biol Med 87: 295-297, 1954.
6. Franke KW, Potter VR: The effect of selenium containing foodstuffs on growth and reproduction of rats at various ages. J Nutr 12: 205-214, 1936.
7. Poley WE, Moxon AL: Tolerance levels of seleniferous grains in laying rations. Poult Sci 17: 72-76, 1938.
8. Schroeder HA, Mitchener M: Selenium and tellurium in mice. Effects on growth, survival and tumors. Arch Environ Health 24: 66-71, 1972.
9. Wahlstrom RC, Olson OE: The effect of selenium on reproduction in swine. J An Sci 18: 141-145, 1959.
10. Minyard JA: Selenium poisoning in beef cattle. S Dak Farm Home Res 12: 1-2, 1961.
11. Dinkel CA, Minyard JA, Ray DE: Effect of season of breeding on reproductive and weaning performance of beef cattle grazing seleniferous range. J An Sci 22: 1043-1045, 1963.
12. Glenn MW, Jensen R, Griner LA: Sodium selenate toxicosis: The effects of extended oral administration of sodium selenate on mortality, clinical signs, fertility and early embryonic development in sheep. Am J Vet Res 25: 1479-1499, 1964.
13. Combs GF, Combs SB: The Role of Selenium in Nutrition. Academic Press Inc, New York: 483-484, 1986.
14. Olson OE: Fluorometric analysis of selenium in plants. J Assoc Off Anal Chem 52: 627-634, 1969.
15. Baker DC, James LF, Hartley WJ et al: Toxicosis in pigs fed selenium-accumulating Astragalus plant species or sodium selenate. Am J Vet Res 50: 1396-1399, 1989.
16. Olson OE, Jornlin DF, Moxon AL: The selenium content of vegetation and the mapping of seleniferous soils. J Am Soc Agron 34: 607, 1942.