SELENIUM CONCENTRATIONS IN BLOOD OF FREE-RANGING MULE DEER IN CALIFORNIA

MICHAEL N. OLIVER, Veterinary Medicine Extension, University of California, Davis, CA 95616

- GLORIA ROS-MCGAURAN,¹ Department of Epidemiology and Preventive Medicine, School of Veterinary Medicine, University of California, Davis, CA 95616
- DAVID A. JESSUP, Wildlife Investigations Lab, California Department of Fish and Game, Rancho Cordova, CA 95670

BEN B. NORMAN, Veterinary Medicine Extension, University of California, Davis, CA 95616

CHARLES E. FRANTI, Department of Epidemiology and Preventive Medicine, School of Veterinary Medicine, University of California, Davis, CA 95616

1990 TRANSACTIONS OF THE WESTERN SECTION OF THE WILDLIFE SOCIETY 26:80-86

Abstract: Whole blood samples from 1,695 mule deer (Odocoileus hemionus) were collected from 15 geographical herd groups from 1980 to 1988, and analyzed for whole blood selenium (Se). Mean Se concentrations for each group were compared to accepted values for livestock species. Eleven groups had mean blood Se concentrations <0.10 mg/L (deficient by livestock standards for groups of animals). Two-thirds of the groups had first quartile Se concentrations (Q1) that were considered seriously deficient (<0.05 mg/L). Significant (P < 0.05) difference in blood Se concentrations were found for geographical location, season (reproductive phase), sex, and resident versus migratory behavior. No significant differences were detected for sub-species or age. The likelihood and manifestations of Se deficiency in deer, especially decreased fawn survivability, are discussed with possible explanations for predisposing influences.

Selenium (Se) deficiency may be as significant a state-wide problem in California as man-made Se toxicity at specific local sites. The likelihood of selenium-responsive diseases in livestock has been shown to increase as blood Se concentrations fall below certain limits (Puls 1981). Selenium deficiency may contribute to neonatal mortality in California mule deer herds (Flueck 1989).

Previous studies have documented the distribution of Se in soils and demonstrate a linear correlation between soil Se and the Se content of forage. Additionally, these studies demonstrated a positive correlation between Se content of forage and Se concentration in blood and other tissues of ruminants (Muth 1963, Kubota et al. 1967, Allaway 1973). Surveys of blood Se levels in beef cattle in California have demonstrated an overall Se herd deficiency (<0.10 mg/L in whole blood) incidence of about 64% (Williams 1980, Dunbar et al. 1988).

Severe Se deficiency in domestic ruminants is expressed as white muscle disease or nutritional muscular dystrophy wherein animals die of cardiac failure. Sub-clinical Se deficiency is manifested by reduced rate of growth, reduced feed efficiency, and lowered immune response (Hartley 1963, Sheppard 1984, Reffett et al. 1988).

Precise nutritional requirements and blood concentration guidelines for Se have not been established for wild ruminants. The pathology of capture myopathy, a condition of wild ruminants, is similar to that of white muscle disease (Ullrey et al. 1981), and most often occurs when blood Se concentrations in blood are below 0.05 mg/L. Capture myopathy has been observed in deer in California (D. Jessup, pers. commun.).

Nutritional myopathy, characterized by stiff, nonfunctional, and painful muscles, may increase the predation of Se deficient deer fawns and yearlings. In a study done in Shasta County, California (Flueck 1989) where livestock and deer typically have deficient blood Se concentrations (by livestock standards, Williams 1980) there was significantly higher survival in fawns born of does that were treated with intraruminal Se pellets as opposed to fawns born of untreated does. Over a three-year period, fawn survival was increased by an overall factor of 2.6. Does given intraruminal pellets had significantly higher blood Se concentrations than their untreated counterparts. For the three-year period, whole blood levels were increased by an overall factor of 3.6 (Flueck 1989).

The present study was undertaken to describe the Se status of various deer herds and groups of deer herds in California, and to examine some of the possible biological and environmental influences of Se status in deer. Ultimately, this information may be useful in explaining and predicting poor fawn survival in some deer herds in California and may be the impetus for innovative herd management techniques to enhance fawn survival.

¹Present address:2928 San Antonio Drive, Walnut Creek, CA 94596

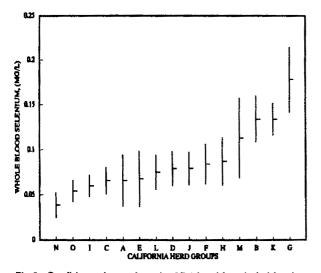


Fig.2. Confidence intervals at the 95% level for whole blood Se concentrations by deer herd groups.

concentration was 1.056 mg/L. All groups contained individual animals with blood Se concentrations at or below the detection limit (BDL) of 0.01 mg/L. Two-thirds of the groups had Q1 values that were < 0.05 mg/L. Seven of the groups had Q4 values < 0.10 mg/L. To compare the means found in Table 1, 99.95% confidence intervals (CI) were constructed. These data suggest that the 3 highest groups (B, K, and G) were significantly (P < 0.05) different than the lowest 10 groups (Fig. 2).

Mean blood Se concentration values

significantly decreased for the seasonality parameters chosen (P < .05): breeding phase (0.094 mg/L.), gestation phase (0.089 mg/L.), and lactation phase (0.076 mg/L). The lowest quartile means were at or below 0.040 mg/L., for all three phases (Table 2). Males (0.086 mg/L) had lower Se values than females (0.091 mg/L)(P < 0.05). Samples were combined for the purpose of seasonality analysis without regard to gender. Some samples were identified only by year of collection and were omitted from this analysis.

A significant difference (P < 0.05) was detected between resident (0.103 mg/L) and migratory (0.081 mg/L) groups (Table 3). There was a significant interaction (P < 0.001) between resident/migratory categories and reproductive phase categories with the greatest difference occurring between resident (0.119 mg/L) and migratory deer (0.065 mg/L) during the breeding phase. No significant (P < 0.05) difference was detected for age, but a significant (P < 0.001)interaction was detected between age and resident/ migratory behavior with resident adults (0.116 mg/)L) having the highest values and young migratory deer (0.078 mg/L) having the lowest values.

No significant (P < 0.05) differences were detected for blood Se concentrations in subspecies or age categories. There was a significant (P < 0.001) interaction between age and resident/ migratory behavior with adult resident deer (0.116 mg/L) having the highest and young migratory deer

Table 2. Results of one-way analysis of variance (ANOVA), comparing mean blood Se levels by reproductive phases of both sexes of deer in California.

Groups	mean	median	SD.	N	Max	Min	Range	Q1	Q4
B₽	.094	.066	.096	619	1.056	BDL	1.047	.031	.126
G⁵	.089	.075	.072	774	.660	BDL	.651	.040 Prob	.118 (Tail)
Lª	.076	.055	.063	285	.416	.010	.406		⁰²⁴ .100
Total			11 	1678		1	F ratio 3.7		

^aStandard Deviation

^bBreeding phase.

^cGestation phase.

Below detection limit (0.01 mg/L).

Missing cases 17.

dLactation phase.

Groups	mean	median	SDª	N	Max	Min	Range	Q1	Q4
R⁵	.103	.079	.1	563	1.056	BDL ⁴	1.047	.040	.128
M	.081	.062	.067	1132	.630	BDL	.621	.031	.113
Total				1695		F ratio 30.22		Prob (2-tail) 0.001	

Table 3. Results of one-way analysis of variance (ANOVA), comparing mean blood Se concentrations between resident and migratory groups of deer in California.

Standard Deviation

^bResident deer.

^cMigratory deer.

^dBelow detection limit (0.01 mg/L).

(0.078 mg/L) having the lowest Se concentrations.

DISCUSSION

No individual deer showed whole blood Se values that would elicit concern for toxicity in livestock (3 mg/L or greater)(Blood and Radostits 1989). Median herd Se values were equal to or less than 0.05 mg/L in one-third of the management groups.

Based on the work of Williams (1980) and others, the Veterinary Extension Unit, School of Veterinary Medicine, University of California, has established descriptive limits for livestock Se deficiency and adequacy for individual animals: whole blood Se greater than 0.08 mg/L (adequate); 0.04 to 0.08 mg/L (inadequate/marginal); less than 0.04 mg/L (deficient). For evaluating mean blood level for groups of animals, 0.10 mg/L is used as the cut-off value between adequate and deficient since most group means of 0.10 mg/L contain individual animals below the adequate value.

Selenium deficiency is widespread in California deer herds as evidenced by deficient first quartile values in two-thirds of the management groups, and individual animal deficiencies in all groups.

Since seasonality and reproductive phase are intimately confounded under natural conditions, we cannot determine the cause of the decrease in blood Se concentrations from breeding phase (August 15-December 14) through gestation phase (December 15-April 14), and lactation phase (April 15-August 14). Over a four-year period, we (Norman and Oliver) have observed blood levels of cattle to be consistently lowest in early summer which is consistent with these data from deer. Williams (1980) observed a tendency for cattle in higher elevations (> 600 m) to have lower blood Se concentrations. Since migratory deer tend to frequent higher elevations more than resident deer, this could explain the lower blood Se concentrations observed for migratory deer. These data suggest that migratory deer were able to move to areas with better sources of Se during their gestation phase, whereas, resident deer encountered their best feed Se sources during their breeding phase.

The lower blood Se concentrations observed in male deer is consistent with data collected from cattle (Norman, unpublished data). The observation that young migratory deer have the lowest blood Se concentrations of the migratory/ resident behaviors versus age may be explained by the stress of two migrations per year during the time of rapid growth when nutrient requirements are typically the highest.

Worth considering are possible explanations for why Se may currently be more of a limiting factor in deer nutrition and recruitment than it was several years ago. In acid to neutral soils that are well-drained, the natural cycling of Se suffers a very gradual depletion from the processes of leaching and volatilization (Allaway 1973). Added to natural losses of Se are those resulting from the removal of biomass from the environment. Removal of Se-containing biomass can take the form of timber harvest, livestock grazing, or fire.

Sulfur can compete with selenium. Sulfurfertilized forage has been shown to contain less Se than non-fertilized forage, and sheep consuming sulfur-fertilized forage have lower blood Se values (Jones et al. 1987). The use of sulfur-fertilizers is a common and extensive agronomic practice to improve forage production in California.

Acid precipitation may contribute to Se depletion in two ways. Selenium in acid and neutral soils tends to be converted to stable, less biologically available forms (Allaway 1973). Sulfate, often a significant component of acid precipitation, may be slowly displacing Se uptake by forage. A study of sulfate deposition from acid rain was measured at eight locations in central and northern California. During a seven-month period from November to May, average sulfate deposition was 3.17 kg/ha (McColl et al. 1982). Although this amount might not contribute significantly to the sulfur requirement of agricultural crops, it might represent a cumulative impact on lower yielding natural forages.

These data suggest that wildlife managers should consider the potential for Se deficiency when working to improve or maintain healthy productive deer herds in California.

ACKNOWLEDGMENTS

This study was made possible by the efforts of a large number of biologists who collected blood samples and other pertinent data. In particular, we appreciate the assistance of Dave Smith, Ron Thomas, Martha and Henry Coletto, Tim Burton, Henry Coletto, and Karen Jones. We also thank Bob Nelson for the many years of quality analytical work that produced the blood Se values.

LITERATURE CITED

- ALLAWAY, W. H. 1973. Selenium in the food chain. Cornell Vet. 63:151.
- BLOOD, D. C. and O. M. Radostits ed. 1989. Veterinary Medicine, seventh edition. Bailliere Tindall. London, England. 1502pp.
- DANIEL, W. W. Biostatistics: A Foundation for the Health Sciences, 4th Ed. J. Wiley and Sons. New York. 1987.
- DIXON, W. J. and M. B. BROWN ed. 1985. BMDP Biomedical Programs. P-Series. Berkeley: Univ. of Calif. Press. 358pp.
- DUNBAR, J. R., B. B. NORMAN, M. N. OLIVER. 1988. Preliminary report on the survey of selenium whole blood values of beef, herds in twelve central and coastal California counties. Pages 81-83. Selenium contents in animal and human food crops grown in California. Cooperative Extension, University of California, Division of

Agriculture and Natural Resources, Publication 3330, Oakland, California.

- FLUECK, W. 1989. The effect of selenium on reproduction of black-tailed deer (Odocoileus hemionus columbianus) in Shasta County, California. Ph.D. Thesis, Univ. of California, Davis. 284pp.
- HARTLEY, W. J. 1963. Selenium and ewe fertility. Proc. New Zealand Soc. Animal Production 23:20.
- JONES, M. B., D. M. CENTER, V. V. RENDIG, M. R. DALLY, B. B. NORMAN and W. A. WILLIAMS. 1987. Selenium enhances lamb gains on sulfur-fertilized pastures. Calif. Agric. 41(5-6):14.
- KUBOTA, J., W. H. ALLAWAY, D. L. CARTER, E. E. CARY and V. A. LAZAR. 1967. Selenium in crops in the United States in relation to selenium-responsive diseases of livestock. J. Agr. Food Chem. 15:448.
- MCCOLL, J. G., L. K. MONETTE and D. S. BUSH. 1982. Chemical characteristics of wet and dry atmospheric fallout in Northern California, USA. J. Environ. Qual. 11:585.
- MUTH, O. H. 1963. White muscle disease, a selenium-responsive myopathy. J. Amer. Vet. Med. Assoc. 142:272.
- PULS, R. 1981. Veterinary trace mineral deficiency and toxicity information. Publication No. 5139, Information Services, Agriculture Canada, Ottawa, 101pp.
- REFFETT, J. K., J. W. SPEARS and T. T. BROWN JR. 1988. Effect of dietary selenium on the primary and secondary immune response in calves challenged with infectious bovine rhinotracheitis virus. J. Nutr. 118:229.
- ROS-MCGAURAN, G. V. 1989. Blood selenium in free-ranging deer in California. Master of Preventive Veterinary Medicine Thesis. University of California, Davis. 56pp
- SHEPPARD, A. D., L. BLOM and A. B. GRANT. 1984. Levels of selenium in blood and tissues associated with some selenium deficiency diseases in New Zealand sheep. New Zealand Vet. J. 32:91.
- ULLREY, D. E., W. G. YOUATT and P. A. WHETTER. 1981. Muscle selenium concentrations in Michigan deer. J. Wildl. Manage. 45:534.
- WHETTER, P. A. and D. E. ULLREY. 1978. Improved fluorometric method for determining selenium. J. Assoc. Off. Anal. Chem. 61:927.

WILLIAMS, J. D. 1980. A survey into selenium deficiency in cattle in Northern California. Master of Preventive Veterinary Medicine Thesis. Univ. of California, Davis. 55pp