A SEROLOGIC SURVEY OF MULE DEER AND ELK IN UTAH[1]

CHARLES L. MERRELL and DONALD N. WRIGHT, Department of Microbiology, Brigham Young University, Provo, Utah 84602, USA.

Abstract: Sera from mule deer *(Odocoileus hemionus)* and elk *(Cervus canadensis)* in central and northern Utah were tested for the prevalence of antibodies to 11 diseases communicable to man or domestic livestock. Antibodies to *Francisella tularensis* (at 1:20) were found in 47 of 88 (53.4%) elk and 1 of 89 (1.1%) deer. A screening slide agglutination test for titers to Brucella (at 1:20) showed two reactors in elk but none in deer sera. No positive antibody titers were obtained in tests for anaplasmosis, Colorado tick fever, Rocky Mountain spotted fever, Q-fever, psittacosis, Powassan, western equine encephalitis, St. Louis encephalitis and California encephalitis.

INTRODUCTION

The prevalence of zoonotic diseases in Utah, with the potential for transmission among free-ranging wildlife, man and domestic animals is essentially unknown. There is a growing concern among outdoor enthusiasts, stockmen and wildlife biologists that with increasing intrusion by man and domestic animals into big game habitats these diseases may become more frequent or even initiate epidemics among more susceptible host populations.[3]

Prior serologic surveys of wildlife in Utah have been published, but they have dealt primarily with small mammals or birds.[2,5,18] A few studies have included variable numbers of mule deer *(Odocoileus hemionus),* [2,17,21,24] but to our knowledge none have reported on elk *(Cervus canadensis).* The purpose of this survey was to determine the prevalence of selected diseases of free-ranging wild ruminants in Utah. Sera from mule deer and elk selected from two separate, major herds in Utah was tested for antibody to 11 diseases considered to be of concern to stockmen, wildlife managers and public health officials.

Sera were tested for antibody to the etiologic agents of tularemia, brucellosis, Rocky Mountain spotted fever (RMSF), Q-fever, psittacosis (Ovine-abortion strain), western equine encephalitis (WEE), St. Louis encephalitis (SLE), California encephalitis (CEE), Powassan (POW), Colorado tick fiver (CTF) and anaplasmosis. These diseases have been reported either in Utah or in adjacent states.

MATERIALS AND METHODS

Deer were trapped between December, 1975 and March, 1976 in east central Utah. The animals were manually restrained and blood was collected in 15 ml vacutainer tubes.

Blood samples from elk were obtained between January and March, 1976 from approximately 20% of a northern Utah herd overwintering at the Hardware Ranch Elk Refuge (Cache River elk herd) in northeastern Utah.

Blood samples from both deer and elk were transported to a microbiology diagnostic laboratory at Brigham Young University (BYU). Sera was collected by centrifugation, divided into aliquots and

[1] Supported by the Department of Microbiology, Brigham Young University, in cooperation with the Utah Division of Wildlife Resources.

stored at -14 C until examined for antibodies.

Slide agglutination tests for brucellosis and tularemia were initially performed in the laboratory at BYU.[2] A second examination of sera for brucellosis antibodies was conducted at the Utah State Veterinary Laboratory, Salt Lake City, Utah, by means of the rapid brucellosis card test. Follow-up examinations for antibodies to *Francisella tularensis* were performed using a hemagglutination technique by the Environmental and Ecology Branch of Dugway Proving Grounds, Utah.

Rocky Mountain Laboratory, Hamilton, Montana, tested all sera for complement fixing antibodies to RMSF, Q-fever, psittacosis, WEE, SLE, CEE, Powassan and CTF. Sera were further tested for hemagglutination inhibiting antibodies to WEE and SLE. The anaplasmosis card test was used by the Utah State Veterinary Laboratory to test sera for antibody to anaplasmosis.

All positive sera were retested using the same procedure also by at least one alternative serological procedure.

RESULTS

Sera were obtained from 88 elk: 44 mature females, one mature male, two yearling females and 41 calves. Five mature females and 20 calves were trapped twice. One male calf was trapped four times. A total of 113 elk serum samples were obtained. All elk were considered to be in good to excellent health. Of the 113 elk sera tested by the hemagglutination technique for *F. tularensis* antibody, 56 (49.6%) gave positive reactions at dilutions of 1:20 or higher. This represented 53.4% of the elk sampled (Table 1).

Analysis of sera from retrapped elk showed that antibody levels in 7 of 9 animals either increased or remained constant during the trapping period (Table 2). When compared by age and sex (Table 3) antibody to *F. tularensis* appears to be more common in female than in males.

Sera were obtained from 89 deer: seven mature males, 45 mature females, six yearlings (five females and one male) and 31 fawns. Nineteen deer were trapped three times and 16 were trapped twice, providing a total of 111 serum samples. With the exception of two aged females, all 89 deer were considered to be in good to excellent condition.

None of the deer or elk had antibody titers to RMSF, Q-fever, psittacosis, WEE, SLE, CEE, Powassan, CTF and anaplasmosis (Table 1). Both deer and elk were negative for brucellosis when tested with the rapid brucellosis antibody detection card. However, two elk (three sera), both mature pregnant cows, had positive brucellosis agglutination tests of 1:20 or greater (Table 1).

DISCUSSION

The purpose of this study was to measure the extent to which 11 zoonotic diseases have developed among mule deer and elk in northern and central Utah. Such data would be useful in range, wildlife and public health management.

Both RMSF and Q-fever have been reported at very low prevalence in mule deer in Utah.[17,24] Reportedly mule deer are naturally resistant to RMSF,[2] and the *Coxiella burnetti* strain found in mule deer in Utah is of lower virulence than strains recovered from animals in other states.[2] Neither of these diseases have been reported in elk. Our data are in agreement with these observations and quite probably these diseases are negligible in the deer and elk herds examined.

A prevalence of less than 0.9% was reported for psittacosis among mule deer

[2] Control antisera and antigens were obtained from Difco Laboratories, Detroit, Michigan 48201, USA.

TABLE 1. Results of serologic analysis for antibodies to selected diseases in deer and elk in northern Utah.

		DEER SERA			ELK SERA		
Disease	Type of Test*	No. Pos.	No. Tested	% Pos.	No. Pos.	No. Tested	% Pos.
Tularemia	HA	1**	111	.9	56	113	49.6
	SA	1	111	.9	45***	102	44.1
Brucellosis	CT	0	111	—	0	113	—
	SA	0	111	—	3	113	2.6
RMSF	CF	0	111	—	0	113	—
Q-fever	CF	0	111	—	0	113	—
Psittacosis	CF	0	111	—	0	113	—
WEE	CF	0	111	—	0	113	—
	HAI	0	111	—	0	113	—
SLE	CF	0	111	—	0	113	—
	HAI	0	111	—	0	113	—
CEE	CF	0	111	—	0	113	—
Powassan	CF	0	111	—	0	113	—
CTF	CF	0	111	—	0	113	—
Anaplasmosis	CT	0	111	—	0	113	—

*HA—hemagglutination; SA—slide agglutination; CT—card test; CF—complement fixation; HAI—hemagglutination inhibition.

**Indicates a suspected positive. Sera tested positive on 7 June, but negative on 2 December 1976.

***Includes seven suspected positive sera.

RMSF—Rocky Mountain spotted fever; WEE—western equine encephalitis; SLE—St. Louis encephalitis; CEE—California encephalitis; CTF—Colorado tick fever.

TABLE 2. Antibody titers to *Francisella tularensis* in multiple serum samples from individual elk.

Animal Number	Dates Sera Were Obtained					Change in Titer	Sex	Age
	3 Feb.	10 Feb.	24 Feb.	26 Feb.	2 Mar.			
5	20/40 (20)	20/40 (20)	40/80 (40)	40/80 (40)	—	Increase	M	Calf
8	40/160 (40)	40/80 (40)	—	—	—	No Change	F	Calf
11	20/80 (40)	20/80 (40)	—	—	—	No Change	F	Calf
12	40/80 (40)	20/80 (20)	—	—	20/40 (20)	Decrease	M	Calf
17	40/160 (40)	—	—	—	80/160 (80)	Increase	F	Calf
18	20/40 (—)	—	neg	—	—	Decrease	M	Calf
32	—	20/80 (20)	—	—	20/40	No Change	F	Cow
56	—	neg	20/40 (20)	—	—	Increase	F	Calf
63	—	—	40/160 (80)	—	160/320 (160)	Increase	M	Calf

1. The first two figures in each column represent the hemagglutination titers obtained when sera were tested on 7 June and 2 December 1976, respectively. The figure in parenthesis is the antibody titer obtained by slide agglutination.

TABLE 3. Elk antibody titers to *Francisella tularensis* in relation to sex and age.*

	Calf		Yearling		Adult		
Titer	F	M	F	M	F	M	Totals
1:20	—	3	1	—	2	—	6
1:40	6	4	1	—	14	—	25
1:80	1	2	—	—	9	—	12
1:160	2	—	—	—	1	—	3
1:320	—	1	—	—	—	—	1
Totals	9	10	2	0	26	0	47**
% of Total Animals by Group	69.2	35.7	100	0	59	0	53.4

*Titer indicated is the highest obtained during the study regardless of test procedure or multiple sera samples.

**Does not include multiple samples from the same animal.

in Utah between the years 1962 and 1967.[2] In 1963, Storz[19] found that one-third of the domestic sheep tested in Utah and Idaho had antibody to *Chlamydia psittaci*. Because of this relatively high prevalence of Chlamydia infections in domestic sheep and its possible transmission by an arthropod vector,[2] we suspected that psittacosis would be found in both deer and elk. However, sera examined at the Hamilton Laboratory failed to demonstrate antibodies to this bacteria in either animal.

The extent of arbovirus infection in the deer and elk of Utah is unknown. WEE is common in domestic livestock in Utah and CEE, SLE and POW have been suspected. Antibody titers to WEE, CEE, SLE and POW have been found in mule deer studied in other states,[6,12] but not in elk. Colorado tick fever has not been reported in either elk or mule deer. We were unable to detect evidence of these viruses in our study and conclude that they are of little immediate concern to the deer and elk herds tested.

Serum antibodies to *Anaplasma marginale* have not been reported in deer or elk of Utah, but anaplasmosis is quite common in beef and dairy cattle in Utah and surrounding states.[11,20] This disease has been reported in mule deer in Oregon,[14] and both deer and elk in Wyoming.[9] Anaplasmosis has been experimentally passed between black-tailed deer *(O.h. columbianus)* and cattle.[1,13] Attempts at such transmission between mule deer and cattle have not been reported. However, efforts to experimentally transmit anaplasmosis between elk and cattle have proven unsuccessful.[15]

Antibodies to *A. marginale* were not found in our sera samples. However, the modified card test for anaplasmosis is known to be somewhat unreliable for non-bovine sera.[8] A more sensitive test for the disease was not attempted. Therefore, even with negative data, there remains a possibility that this organism may be established in the deer and elk of Utah.

The combined state and federal test and slaughter programs have largely controlled brucellosis in Utah. Earlier surveys of mule deer in Utah for evidence of brucellosis have suggested a low prevalence of this disease. Fay[4] reported no positive sera in a test of 11 animals. Thorpe[22] found only three serologically positive mule deer in a 1963 study, and Vest[24] reported no serologic reactors in a group of 37 mule deer taken from a

western Utah desert. These studies, as well as a study of brucellosis in mule deer in Idaho,[16] have resulted in such low reactor rates that most authors have concluded that wild populations of mule deer are insignificant hosts for *Brucella*.

Brucellosis has been reported in the elk of Yellowstone National Park,[7] but studies in Montana[10] and Idaho[16,23] did not detect antibodies to *B. abortus* in elk sera. Our data (Table 1) are consistent with reported findings in deer and elk. Of the 111 deer sera tested, none had detectable antibody levels, while only 2.6% of elk sera were positive. An antibody titer of 1:40 in one elk suggests the possibility of active or recent infection.

Following the work of Francis,[5] a number of studies have shown *F. tularensis* to be endemic in fauna of western Utah. Using the slide agglutination test, Vest[24] found that 9 of 37 (24.3%) mule deer had antibody titers and Thorpe[21] found that 21 of 126 (16.6%) mule deer had titers greater than 1:20. The etiologic agent was not isolated from tissues in either study. Twenty of 453 (4.4%) mule deer from west central Utah examined between 1962 and 1973 had antibody titers to *F. tularenis;* however, the number of positive sera declined from 16.1% positive in 1963 to no reactive sera during the years 1970-1973. (H. Stark, Environmental and Ecology Branch, Dugway Proving Grounds, pers. comm.)

We found only one (0.9%) suspected reactor to *F. tularensis* in mule deer sera which correlates well with previously published reports on mule deer. Why the sera tested positive on the first occasion and negative on the second (Table 1) is not understood. This is especially true in that the second test antibody titers were characteristically more sensitive for elk sera.

Our findings of antibodies to *F. tularensis* in elk sera is the first reported indication that tularemia may exist in this species. The fact that 49.6% of the sera were positive, six with antibody titers of 1:160 and 21 sera greater than or equal to 1:80, would indicate that *F. tularensis* may be quite common in the elk of Utah.

Because our survey sampled approximately 20% of the entire herd at the Hardware Ranch Elk Refuge, it is reasonable to assume that *F. tularensis* is highly enzootic in this area.

There is one recorded case in which tularemia was passed from mule deer to man,[3] suggesting that this species may act as a reservoir host for *F. tularensis*. To what extent this may be true of elk is yet to be determined. Considering the large annual elk harvest from the Cache River herd, and the results of this study, there seems to be sufficient justification for additional investigation.

With the exception of *F. tularensis* in elk, the deer and elk herds examined appear to be remarkably free of zoonotic disease. Brucellosis and anaplasmosis possibly may be significant, but serologic survey suggests that there is little cause for concern.

Acknowledgements

We wish to thank Dr. Leo Thomas of Rocky Mountain Laboratories, Dr. Harold Stark of Dugway Proving Grounds and Dr. Eldon Childs of the Utah State Veterinary Laboratory for their assistance with laboratory analysis; and the personnel of the Utah State Division of Wildlife Resources for their assistance in obtaining animals for the study.

LITERATURE CITED

1. CHRISTENSEN, J.F., J.W. OSEBOLD and M.N. ROSEN. 1958. Infection and antibody response in deer experimentally infected with *Anaplasma marginale* from bovine carriers. J. Am. vet. med. Ass. 132: 289-292.

2. Ecology and Epizoology Research Group, University of Utah, Dugway and Salt Lake City, Utah. Ecology and Epizoology Series and Annual Summary Progress Reports from 1962 to 1969.
3. EMMONS, R.W., J. RUSKIN, M.L. BISSETT, D.A. UYEDA, R.M. WOOD and C.L. LEAR. 1976. Tularemia in a mule deer. J. Wildl. Dis. 12: 459-463.
4. FAY, L.D. 1961. The current status of brucellosis in white-tailed and mule deer in the U.S. Trans. 26th N. Am. Wildl. and Natural Resources Conf. March, 1961. Wildl. Manage. Inst., Washington, D.C.
5. FRANCIS, E. 1921. Tularemia. The occurrence of tularemia in nature as a disease of man. Publ. Hlth. Rpts., 36: 1731.
6. HOFF, G.L. and C.J. ISSEL, D.O. TRAINER and S.H. RICHARDS. 1973. Arbovirus serology in North Dakota mule and white-tailed deer. J. Wildl. Dis., 9: 291-295.
7. HONESS, R.F. 1956. *Diseases of Wildlife in Wyoming.* Wyoming Game Fish Comm. Bull. 9.
8. HOWARTH, J.A., Y. HOKAMA and T.E. AMERAULT. 1976. The modified card agglutination test: an accurate tool for detecting anaplasmosis in Columbian black-tailed deer. J. Wildl. Dis., 12: 427-433.
9. HOWE, D.L., W.G. HEPWORTH. 1965. Anaplasmosis in big game animals: tests on wild populations in Wyoming. Am. J. vet. Res., 26: 1114-1120.
10. KNIGHT, R.R. 1969. Some chemical characteristics of elk blood. J. Wildl. Dis. 5: 8-10.
11. LONG, J.D., E.H. STAUBER and F.W. FRANK. 1974. Prevalence and distribution of bovine anaplasmosis in Northern Idaho and Southeastern Washington. J. Am. vet. med. Ass. 165: 918-920.
12. McLEAN, D.M., A. DeVOS and E.J. QUANTZ. 1964. Powassan Virus: Field investigations during the summer of 1963. Am. J. Trop. Med. Hyg. 13: 747-753.
13. OSEBOLD, J.W., J.R. DOUGLAS and J.F. CHRISTENSEN. 1962. Transmission of anaplasmosis to cattle by ticks obtained from deer. Am. J. vet. Res. 23: 21-23.
14. PETERSON, K.J., T.P. KISTNER and H.E. DAVIS. 1973. Epizootiologic studies of anaplasmosis in Oregon mule deer. J. Wildl. Dis. 9: 314-319.
15. POST, G. and G.M. THOMAS. 1961. Anaplasmosis in elk. J. Am. vet. med. Ass. 139: 357-358.
16. SHAW, W.M. 1964. Idaho big game harvest, census and range study. Job. No. 15, Disease and Parasitism tests and reports, July, 1962 to June 30, 1963. Idaho Fish and Game Dept. Boise, pp. 17.
17. SIDWELL, R.W., D.L. LUNDGREN, J.B. BUSHMAN and B.D. THORPE. 1964. The occurrence of a possible epizootic of Q-fever in fauna of the great Salt Lake Desert of Utah. Am. J. Trop. Med. Hyg. 13: 754-762.
18. STOENNER, H.G., R. HOLDENRIED, D. LACKMAN and J.S. ORSBORN. 1959. The occurrence of *Coxiella burnetii,* Brucella, and other pathogens among fauna of the Great Salt Lake Desert in Utah. Am. J. Trop. Med. Hyg. 8: 590-596.
19. STORZ, J. 1963. Superinfection of pregnant ewes laterally infected with a psittacosis-lymphogranuloma agent. Cornell Vet. 53: 469-480.

20. THOMAS, G.M. 1958. The incidence of anaplasmosis in Wyoming. J. Am. vet. med. Ass. 132: 61-62.

21. THORPE, B.D., R.W. SIDWELL, D.E. JOHNSON, K.L. SMART and R. MOYES. 1965. Tularemia in the wildlife and livestock of the Great Salt Lake Desert region, 1951 through 1964. Am. J. Trop. Med. Hyg. 14: 622-637.

22. ——, ——, J.B. BUSHMAN, K.L. SMART and R. MOYES. 1965. Brucellosis in wildlife and livestock of West Central Utah. J. Am. vet. med. Ass. 146: 225-232.

23. VAUGHN, H.W., R.R. KNIGHT and F.W. FRANK. 1973. A study of reproduction, disease and physiological blood and serum values in Idaho elk. J. Wildl. Dis. 9: 296-301.

24. VEST, E.D., D.L. LUNDGREN, D.D. PARKER, D.E. JOHNSON, E.L. MORSE, J.B. BUSHMAN, R.W. SIDWELL and B.D. THORPE. 1965. Results of a five year survey for certain enzootic diseases in the fauna of Western Utah. Am. J. Trop. Med. Hyg. 14: 124-135.

Received for publication 28 October 1977