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A SEROLOGIC SURVEY OF PRONGHORNS IN ALBERTA AND SASKATCHEWAN, 1970-1972

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Abstract: To determine the exposure of free-ranging pronghorns (*Antilocapra americana* Ord) to selected pathogens, serum samples were obtained from 33 live-trapped animals from southwestern Saskatchewan in 1970, and from 26 and 51 animals from southeastern Alberta, in 1971 and 1972, respectively. Antibodies were found to the agents of parainfluenza 3, bovine virus diarrhea, eastern and western encephalomyelitis, infectious bovine rhinotracheitis and the chlamydial group. No serologic reactors were found to the agents of bluetongue, epizootic hemorrhagic disease, brucellosis, or leptospirosis (4 serotypes).

INTRODUCTION

Serologic surveys have been utilized commonly to indicate the presence of pathogens in many wildlife species. Furthermore, Trainer[31] has suggested that certain species might be valuable sentinels in monitoring diseases of wildlife. In Alberta, such investigations have centered on arbovirus activity in the snowshoe hare (*Lepus americanus*)[15,16,35] and on serology in moose,[28,32] (*Alces alces*). Several researchers have reported on serologic surveys of pronghorns in parts of the United States[12,29,30,34] but no similar work has been conducted in their northern ranges.

In Canada, pronghorns occupy primarily the native short grass prairie biome in southeastern Alberta and southern Saskatchewan. They move considerable distances between summer and winter ranges and share their habitat with domestic sheep, cattle and horses as well as white-tailed deer (*Odocoileus virginianus*), mule deer (*O. hemionus*), elk (*Cervus canadensis*), and moose. Consequently, the opportunity for contact between pronghorns and other ruminants is great. Because pronghorns move freely and regularly between Alberta, Saskatchewan and Montana, (M. W. Barrett, unpublished data) their importance as potential vectors and sentinels of disease cannot be underrated. The purpose of this paper is to report on a 3-year serologic survey of pronghorns in Alberta and Saskatchewan, for the presence of antibodies to a number of viral and bacterial pathogens. Such studies are particularly timely in Alberta as we have documented unusually low kid production, during the years 1971 to 1973, inclusive.

MATERIALS AND METHODS

Pronghorns were live-trapped during the fall months from 1970 to 1972. Blood samples were drawn by jugular venipuncture from captured pronghorns and the resulting serum samples were frozen until used. The 1970 blood samples were obtained from one herd of pronghorns captured in southwestern

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Saskatchewan, while the 1971 and 1972 samples were obtained from animals captured in three different locations in southeastern Alberta. Pronghorns from different capture sites do not represent completely distinct populations as some mixing of tagged animals from the various sites has been documented. Tag returns from antelope captured in Saskatchewan indicate that they have only sporadic contact with the pronghorn population sampled in Alberta. Conversely, frequent exchange occurs between the Alberta herds sampled in 1971 and 1972 and therefore these animals should be considered essentially part of the same population (M. W. Barrett, unpublished data). Antelope were aged according to the tooth eruption and wear technique.[14]

Serologic examinations were conducted according to the following procedures: 1) modified direct complement fixation test for bluetongue (BT)[9] using an acetone-ether extracted mouse brain antigen of California type 10 BT virus, and eastern (EEE) and western (WEE) encephalomyelitis[7,8] using commercial antigens;[1] 2) hemagglutination-inhibition test for parainfluenza 3 (PI_3)[2] using SF_4 antigen; non-specific inhibitors to hemagglutination were removed by absorption in kaolin;[2] 3) virus neutralization test for epizootic hemorrhagic disease (EHD)[18] using the Alberta strain[10] of EHD virus in suckling mouse brains, infectious bovine rhinotracheitis (IBR)[11] using antigen strain 108[2] and bovine virus diarrhea (BVD)[24] using the NADL-MD strain of BVD antigen;[3] 4) agglutination tests for leptospirosis[5] using four serotypes, and brucellosis;[19] and 5) direct complement fixation tests[4,6] for the chlamydial group using antigen prepared from yolk sac membranes inoculated with the agent of enzootic abortion of ewes.

Serum titers recorded for EEE, WEE, PI_3, IBR, BVD, and the chlamydial group are presented in the results. Serum titers for the remaining microbial agents tested are not presented and were considered negative based on the following minimum criteria: 1) BT—1/5; 2) leptospirosis— 1/10; and 3) brucellosis—1/50. Sera for EHD were required to neutralize 1.5 $\log_{10}$ or more virus to be considered positive. Because little experience is available concerning the serologic responses of pronghorns to specific microbial agents, titers which would be considered low in domestic animals are presented here to facilitate future interpretation.

RESULTS

Clinical signs of disease were not evident in any animal handled and no attempts were made to collect material for isolation procedures. Serologic reactors were found to PI_3, IBR, BVD, EEE, WEE, and the chlamydial group (Table 1) but not to BT, EHD, brucellosis or the *Leptospira* group (4 serotypes) (Table 1).

Titers of the 17 reactors to EEE varied from 1:5 (n=7), 1:10 (n=6), 1:20 (n=1), 1:40 (n=2), to 1:160 (n=1). Similarly, the titers of 9 reactors to WEE varied from 1:5 (n=3), 1:10 (n=2), 1:20 (n=1), 1:40 (n=2) to 1:160 (n=1). The three reactors to BVD had titers of 1:20. Three of the five reactors to the chlamydial group had titers of 1:10 and the remaining two had titers of 1:5.

Antibodies to PI_3 were found in all 33 samples examined in 1970 but in none of the 51 samples examined in 1972. The 1970 titers were regarded as significant and are presented in Table 2. The highest titers were found in mature animals but conversely, not all mature animals had high titers.

Similarly, 29 IBR reactors were found in the 1970 samples (Table 2) while the 1971 and 1972 sera were negative. A larger percentage of samples with IBR antibodies and the highest individual

[1] Beckman Instruments, Inc., Fullerton, Calif., U.S.A.
[2] Veterinary Services Branch, Edmonton, Alberta.
[3] National Animal Dis. Lab., Ames, Iowa.

titers were found in mature pronghorns. The 33 animals bled in 1970 were part of a single herd moving towards the winter range.

There were more reactors to BVD, EEE, WEE, and chlamydial group antigens in adult pronghorns than in juveniles (Table 3). The reverse appears true for PI_3 but the figures are influenced by the fact that all 1970 samples were positive whereas proportionately fewer kids have been tested for PI_3 since 1970. There were no differential sex specific reactor rates for any antigen tested.

TABLE 1. A summary of serologic results for 110 pronghorn antelope in Saskatchewan (1970) and Alberta (1971 and 1972).

Microbial Agent	Serologic Results			
	1970	1971	1972	Total
PI_3	33/33(100)[a]	—	0/51 (0)	33/84 (39)
IBR	29/33 (88)	0/26 (0)	0/51 (0)	29/110(26)
BT	—[b]	0/26 (0)	0/51 (0)	0/77 (0)
EHD	0/33 (0)	—	—	0/33 (0)
BVD	1/33 (3)	—	2/51 (4)	3/84 (4)
EEE	6/33 (18)	5/26(19)	6/51(12)	17/110(16)
WEE	4/33 (12)	5/26(19)	0/51 (0)	9/110 (8)
Brucella abortus	0/33 (0)	0/26 (0)	0/51 (0)	0/110 (0)
Leptospira pomona	0/33 (0)	0/26 (0)	0/51 (0)	0/110 (0)
L. sejroe	0/33 (0)	0/26 (0)	0/51 (0)	0/110 (0)
L. autumnalis	0/33 (0)	—	—	0/33 (0)
L. icterohemorrhagiae	0/33 (0)	—	—	0/33 (0)
Chlamydial Group	—	0/26 (0)	5/51(10)	5/77 (7)

[a] Number positive/number examined (percent positive).
[b] No data.

TABLE 2. A summary of antibody prevalence and titers to PI_3 and IBR in live-trapped pronghorns in southwestern Saskatchewan, 1970.

Age (Years)	PI_3 Titer			IBR Titer		
	No. Positive / Sample Size	Mean	(Range) [a]	No. Positive / Sample Size	Mean	(Range) [a]
½	11/11	2.1	(1.7-2.9)[a]	8/11	0.8	(0.7-1.0)[a]
1½	8/8	2.3	(1.7-3.2)	8/8	0.7	(0.7-1.0)
2½	4/4	2.7	(1.7-3.2)	4/4	1.0	(0.7-1.3)
3½ +	10/10	2.6	(1.7-3.2)	9/10	0.9	(0.7-1.3)

[a] Mean and range expressed using $-\text{Log}_{10}$ conversion for titers. Only positive reactors included.

TABLE 3. Adult and juvenile antibody prevalence rates in Alberta and Saskatchewan pronghorns, 1970 to 1972.

Age [a]	PI_3	IBR	BVD	EEE	WEE	Chlamydial Group
Adults	22/60(37)[b]	21/78(27)	3/60(5)	16/78(21)	8/78(10)	5/56(9)
Kids	11/24(46)	8/32(25)	0/24(0)	1/32 (3)	1/32 (3)	0/22(0)

[a] Adults were 1½ to 7½ years old and kids were 5 to 7 months old when bled.

[b] Number positive/number examined (percent positive).

DISCUSSION

The presence of antibodies to PI_3, BVD and WEE in pronghorns from Alberta and Saskatchewan was consistent with findings for moose collected in essentially the same geographic area.[25,32] This, to our knowledge, is the first serologic evidence of PI_3, EEE, and chlamydial infections in pronghorns. EEE reactors were not found in either Cypress Hills moose[32] or in pronghorns in Wyoming.[29] Nine of the 17 samples with antibody to EEE were also positive to WEE antigen, suggesting the possibility of cross reaction. However, in the absence of experimental data one cannot dismiss the possibility that the nine pronghorns were actually exposed to both agents. Antibodies have been reported to the agents of WEE, California and St. Louis encephalitis, and vesicular stomatitis in pronghorns in Wyoming[29] and to BT in pronghorns in Colorado and Wyoming.[20]

PI_3 virus has been associated with active respiratory disease in cattle,[22,23] domestic sheep[17,18] and captive bighorn sheep (*Ovis canadensis*).[21] However, there was no evidence of respiratory or other clinical disease in pronghorns at the time of handling in 1970 and therefore no attempt was made to obtain nasal washings for PI_3 isolation. The significance of the high titers is unknown, but these findings warrant further investigation. Some of the high titers detected in this study exceeded those reported for domestic sheep in which there was depression, moderate ocular discharge or a mild to moderate nasal discharge.[18] Colostral immunity to PI_3 in domestic calves decreases steadily with age and disappears by weaning at six months.[1,27] In the absence of data to the contrary, it was assumed that an analogous situation may exist in pronghorn kids. Consequently, titers in both sexes and in all age classes suggested that the 1970 results reflected recent PI_3 infection. In interpreting the PI_3 results for 1970 it should be remembered that only one herd was sampled. The low prevalence of PI_3 antibodies in deer sampled in the United States[20] imparts further significance to the number and magnitude of titers reported in this study.

IBR titers have been reported consistently in approximately half of the cattle sampled in Alberta[11,20] and Saskatchewan[25] and the virus has been implicated in an increase in clinical disease in recent years;[3] no similar parallel has been observed in wildlife in these provinces. One serologically positive animal in Texas[13] and the 29 titers recorded in this study are the only IBR reactors reported for pronghorns. Except to indicate exposure and subsequent response to the virus the significance of IBR antibodies in pronghorns is unknown. Darcel[11] regarded titers in excess of 1:4 for cattle as positive and some authorities[1] consider any measurable titer for IBR as protective. No explanation is known for the high prevalence of IBR reactors in the 1970 pronghorn samples and the contrasting absence of such antibodies in 1972 but again we caution that the 1970 IBR results were obtained by sampling only one herd.

The absence of EHD reactors suggested that the disease was not enzootic in

pronghorns in the area, despite its being responsible for significant mortality among white-tailed deer, mule deer and probably antelope in the same area of Alberta in 1962.[10] Moreover, these findings suggest that potentially EHD-susceptible populations of pronghorns exist once again. The absence of antibodies to brucellosis and leptospirosis in pronghorns was consistent with the findings of Yoakum[34] in Oregon, but was in some contrast to those of DeArment[13] who reported reactors to leptospirosis, BVD, and IBR, but none to brucellosis, in the Texas panhandle.

Serologic reactions to IBR, BVD, EEE, WEE, PI_3 and the chlamydial group do not necessarily constitute evidence of the respective clinical diseases in pronghorns. In the absence of immunochemical and microbial support these serologic findings can only suggest that the animals had been exposed to the various agents tested for, and had developed measurable responses to them. The effect of these agents on free-ranging populations is not known and it would seem logical that the next investigative steps lie in attempting to isolate causative agents and to examine their impact on pronghorns.

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LITERATURE CITED

1. ANONYMOUS. 1967. Iowa State University, Veterinary Newsletter No. 91, December, 1967.
2. ————. 1971. Recommended standard laboratory techniques for diagnosing infectious bovine rhinotracheitis, bovine virus diarrhea and parainfluenza 3. Proc. 75th Ann. Mtg. U.S. Animal Health Assn. and 14th Ann. Conf. of Amer. Assoc. Vet. Lab. Diag. Oklahoma City, Okla. pp. 629-648.
3. ————. 1972. Annual Report. Vet. Services Div., Alberta Agriculture. pp. 95-105.
4. BANNISTER, G. L., P. BOULANGER, D. P. GRAY, C. H. CHAPMAN, R. J. AVERY and A. H. CORNER. 1962. Sporadic bovine encephalomyelitis in Canada. Can. J. comp. Med. 26: 25-32.
5. BOULANGER, P. and A. N. SMITH. 1957. Serological investigations of leptospirosis in Canada, II. Preliminary agglutination - lysis studies of cattle sera with *Leptospira pomona* and *Leptospira canicola* antigens. Can. J. comp. Med. 21: 4-11.
6. ———— and G. L. BANNISTER. 1959. Abortion produced experimentally in cattle with an agent of the psittacosis lymphogranuloma-venereum group of viruses. Can. J. comp. Med. 23: 251-265.
7. ———— and ————. 1960. A modified direct complement fixation test for the detection of antibodies in the serum of cattle previously infected with vesicular stomatitis virus. J. Immun. 85: 368-374.
8. ————. 1960. Technique of a modified direct complement fixation test for viral antibodies in heat inactivated cattle serum. Can. J. comp. Med. 24: 262-269.

9. ————, G. M. RUCKERBAUER, G. L. BANNISTER, D. P. GRAY and A. GIRARD. 1967. Studies on bluetongue III. Comparison of two complement-fixation methods. Can. J. comp. Med. 31: 166-170.

10. CHALMERS, G. A., H. N. VANCE and G. J. MITCHELL. 1964. An outbreak of epizootic hemorrhagic disease in wild ungulates in Alberta. Wildlife Dis. 42: 1-6.

11. DARCEL, C. le Q. 1973. The prevalence of neutralizing antibodies to Infectious bovine rhinotracheitis (IBR) in cattle in Alberta. Can. vet. J. 14: 167-169.

12. DeARMENT, R. 1965. Antelope in the Texas panhandle. Proc. Antelope States Workshop. New Mexico Dept. of Fish and Game, Santa Fe. 1: 54-60.

13. ————. 1968. Antelope herd population dynamics versus disease in the Texas panhandle. Proc. Antelope States Workshop. Wyoming Game and Fish Commission, Cheyenne. 3: 48-52.

14. DOW, S. A. Jr. and P. L. WRIGHT. 1962. Changes in mandibular dentition associated with age in pronghorn antelope. J. Wildl. Mgmt 26: 1-8.

15. HOFF, G. L., T. M. YUILL, J. O. IVERSEN and R. P. HANSON. 1969. Snowshoe hares and the California encephalitis virus group in Alberta, 1961-1968. Bull. Wildl. Dis. Ass. 5: 254-259.

16. ————, R. O. ANSLOW, J. SPALATIN and R. P. HANSON. 1971. Isolation of Montana snowshoe hare serotype of California encephalitis virus group from a snowshoe hare and Aedes mosquitoes. J. Wildl. Dis. 7: 28-34.

17. HORE, D. E. 1966. Isolation of ovine strain of parainfluenza virus serologically related to type 3. Vet. Rec. 79: 466.

18. ————, R. G. STEVENSON, N. J. L. GILMOUR, J. T. VANTSIS and D. A. THOMPSON. 1968. Isolation of PI_3 viruses from the lungs and nasal passages of sheep showing respiratory disease. J. comp. Path. 78: 259-265.

19. MITCHELL, C. A. and F. A. HUMPHRYES. 1933. Serological diagnosis of Brucellosis. Can. Publ. Health J. 24: 337-342.

20. NIILO, L., R. J. AVERY, C. le Q DARCEL and A. S. GREIG. 1962. Serum antibolies to padlock and IBR viruses in Alberta. Can. J. comp. Med. 26: 39-41.

21 PARKS, J. B., G. POST, J. THORNE and P. NASH. 1972. Parainfluenza 3 virus infection in Rocky Mountain bighorn sheep. J. Am. vet. med. Ass. 161: 660-672.

22. REISINGER, R. C., K. R. HEDDLESTON and C. A. MANTHEI. 1959. A myxovirus (SF-4) associated with shipping fever in cattle. J. Am. vet. med. Ass. 135: 147-152.

23. ROSENQUIST, B. D., J. E. ENGLISH, D. W. JOHNSON and R. W. LOAN. 1970. Mixed viral etiology of a shipping fever epizootic in cattle. Am. J. Vet. Res. 31: 980-994.

24. RUCKERBAUER, GERDA M., A. GIRARD, G. L. BANNISTER and P. BOULANGER. 1971. Studies on bovine virus diarrhea: Serum neutralization, complement-fixation and immunofluorescence. Can. J. comp. Med. 35: 230-238.

25. SAUNDERS, J. R., M. A. WETZSTEIN and M. G. PRIOR. 1972. Infectious bovine rhinotracheitis (IBR): serum antibodies in cattle in Saskatchewan. Can. vet. J. 13: 240-241.

26. SHAH, K. V. and G. B. SCHALLER. 1965. Antibodies to myxovirus parainfluenza 3 in sera of wild deer. Bull. Wildl. Dis. Ass. 2: 31-32.

27. SWEAT, R. L. 1967. Epizootiologic studies of bovine myxovirus parainfluenza 3. J. Am. vet. med. Ass. 150: 178-183.

28. THORSEN, J. and J. P. HENDERSON. 1971. Survey for antibody to infectious bovine rhinotracheitis (IBR), bovine virus diarrhea (BVD), and parainfluenza 3 (PI_3) in moose sera. J. Wildl. Dis. 7: 93-95.

29. TRAINER, D. O. and R. P. HANSON. 1969. Serologic evidence of arbovirus infections in wild ruminants. Am. J. Epidem. 90: 354-358.

30. ————, and M. M. JOCHIM. 1969. Serologic evidence of bluetongue in wild ruminants of North America. Am. J. vet. Res. 30: 2007-2011.

31. ————. 1970. The use of wildlife to monitor zoonoses. J. Wildl. Dis. 6: 397-401.

32. ———— and G. L. HOFF. 1971. Serologic evidence of arbovirus activity in a moose population in Alberta. J. Wildl. Dis. 7: 118-119.

33. WILHELM, A. R. and D. O. TRAINER. 1966. A serological study of epizootic hemorrhagic disease of deer. J. Wildl. Mgmt 30: 777-780.

34. YOAKUM, J. D. 1957. Factors affecting the mortality of pronghorn antelope in Oregon. M.S. Thesis, Oregon State Univer., Corvallis. 112 pp.

35. YUILL, T. M., J. O. IVERSEN and R. P. HANSON. 1969. Evidence of arbovirus infections in a population of snowshoe hares: a possible mortality factor. Bull. Wildl. Dis. Ass. 5: 248-253.

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