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SEROLOGIC EVIDENCE OF ARBOVIRUS ACTIVITY IN A MOOSE POPULATION IN ALBERTA

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Abstract: Twenty-three sera from a moose (Alces alces) population in southeastern Alberta were serologically tested for virus activity. No reactors were detected in a metabolic inhibition test in tissue culture to eastern encephalitis, vesicular stomatitis or encephalomyocarditis; there was one reactor to St. Louis encephalitis, two reactors to western encephalitis, and 16 reactors to California encephalitis. No positive reactors were obtained against bluetongue or epizootic hemorrhagic disease in plaque-reduction neutralization tests. This is the first report of serologic reactors to these diseases in moose and the epizootiological significance of these findings is discussed.

INTRODUCTION

Information on infectious diseases in free-living moose populations is limited.[2,3] Available data concerning this subject suggests that moose are often exposed to many of the same maladies as domestic animals and human populations.[7,8]

As a result, when sera became available from the Cypress Hills Park moose population in Alberta, Canada, it was considered worthwhile to examine these specimens serologically so that the results might add to our knowledge concerning diseases of moose.

MATERIALS AND METHODS

As part of a population study of moose in Cypress Hills Provincial Park of southeastern Alberta, 25 moose were collected by shooting in February 1970. From the specimens collected, 23 sera were obtained and heat inactivated at 56° for 30 minutes prior to serologic testing.

Moose sera were tested for their ability to neutralize $10^{1.5}$ to $10^{2.5}$ ID_{50} of selected viruses in a metabolic inhibition test (MIT) using HeLa cells.[6] The viruses used included: eastern (EE), western (WE), California-snowshoe hare strain (CE), and St. Louis (SLE) encephalitis, encephalomyocarditis (EMC), and both the New Jersey and Indiana serotypes of vesicular stomatitis (VS).

A plaque reduction neutralization test described by Thomas and Trainer[4] was used to measure antibodies against bluetongue (BT) virus, strain BT_8, and epizootic hemorrhagic disease (EHD) virus, the original Shope isolate NJ-55.[5] All sera were diluted 10^{-2} and tested; virus was quanitated to produce 30-80 plaque forming units per plate.

RESULTS

There were few or no serologic reactors detected in the MIT to EE, WE, SLE, EMC, or VS (Table 1). Sixteen (70%) of the sera tested against CE

Table 1. A summary of metabolic inhibition test serologic results for moose from Alberta.

No.	Age*	Serologic results (percent positive)					
		EE	WE	CE	SLE	EMC	VS
18	adults	0	12	78	6	0	0
5	calf	0	0	40	0	0	0
Total 23	—	0	8	70	4	0	0

*Calves were approximately 8 months of age; adults ranged from 1.5 to 9.5 years of age.

were positive. California encephalitis reactors were detected in adult as well as young of the year, and in both sexes.

No positive reactions were obtained against BT virus or EHD virus in plaque reduction neutralization tests

DISCUSSION

Based on the serologic results of the MIT there appeared to have been some arbovirus activity in the moose population in the Cypress Hills Provincial Park. Of particular interest was the large percentage of reactors to CE. The serologic results for moose in this study are not unlike those reported for other wild ruminants in North America,[7] and adds the moose to the list of wild species which has antibody against viruses of the California encephalitis group, western encephalitis, and St. Louis encephalitis. The presence of reactors among young of the year suggests recent CE activity in the area. Based on studies in other wild ruminants it would appear that moose might serve as an indicator of CE activity, and that they are not adversely affected by the California group of viruses.

Cypress Hills Provincial Park, which also has white-tailed deer *(Odocoileus virginianus)*, mule deer *(O. hemionus)*, elk *(Cervus canadensis)*, and cattle is near the area in Alberta which experienced the greatest deer losses to EHD in 1962.[1] Although the sample tested was limited, it was of interest that no serologic evidence of BT or EHD viruses was detected.

The limitations of serologic testing on small numbers of wild sera are appreciated, but such studies do provide useful information, such as documenting the occurrence of serologic reactors to CE, WE, and SLE in moose for the first time, and providing a basis for defining areas for future study.

Acknowledgement

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