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EPIZOOTIC VESICULAR STOMATITIS IN COLORADO, 1982: SOME OBSERVATIONS ON THE POSSIBLE ROLE OF WILDLIFE POPULATIONS IN AN ENZOOTIC MAINTENANCE CYCLE

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ABSTRACT: Sera obtained from wild ungulates, carnivores, and rodents in Colorado were tested for neutralizing (N) antibody against vesicular stomatitis, New Jersey serotype (VSNJ), virus to determine their involvement in the 1982 Colorado VSNJ epizootic in domestic animals. Viremic and N antibody responses of two local rodent species to a 1982 Colorado isolate of VSNJ were determined in the laboratory. The rodents produced only weak viremias, but all developed N antibody. N antibody prevalences for VSNJ in sera from wild ungulates was sufficiently high to indicate their involvement during the epizootic. In addition, the demonstration of N antibody in elk (*Cervus elaphus*) and mule deer (*Odocoileus hemionus*) prior to the epizootic in cattle and horses suggests that an enzootic cycle may exist in Colorado.

Key words: Vesicular stomatitis, New Jersey serotype, neutralizing antibody, virus, rodents, elk, *Cervus elaphus*, mule deer, *Odocoileus hemionus*.

INTRODUCTION

In 1982, an epizootic of vesicular stomatitis (VS) virus, New Jersey serotype (VSNJ), affected large numbers of livestock in Colorado and neighboring western states. This was one of the largest VS epizootics on record in the Rocky Mountain States, and it caused considerable economic losses to dairy farmers (Alderink, 1985) and horse breeders. We studied the possibility of persistence of VS in an enzootic focus in the western United States and the involvement of wildlife in a natural cycle.

In 1957, Hanson visualized a cycle of natural persistence of sylvan VS in wild mammals and undetermined reservoirs with domestic animals involved by chance (Hanson and Brandly, 1957). Pronghorn (*Antilocapra americana*), deer, and elk (*Cervus elaphus*) are known to graze with cattle on range pasture, but their role as possible reservoir hosts or in active disease transmission to domestic animals during an outbreak remains speculative. Exper-

imental infections of deer result in short-lived disease followed by high titers of neutralizing (N) antibody (Hanson and Brandly, 1957). Thus, their importance as a true reservoir host seemed unlikely. Whether infection in wildlife occurs between periods of active transmission in livestock or at the same time is also unknown. Serologic surveys of wild animal populations have revealed the presence of N antibody in white-tailed deer (*Odocoileus virginianus*) (Karstad et al., 1956; Hanson and Brandly, 1957; Karstad and Hanson, 1957; Trainer and Hanson, 1969; Jenney et al., 1970; Fletcher et al., 1985; Stallknecht and Erickson, 1986), mule deer (*O. hemionus*) (Jenney et al., 1980), pronghorn, bighorn sheep (*Ovis canadensis*), wild pigs (*Sus scrofa*), raccoons (*Procyon lotor*), bobcats (*Lynx rufus*), opossums (*Didelphis virginianus*), squirrels, and wild turkeys (*Meleagris gallopavo*) (Karstad et al., 1956; Hanson and Brandly, 1957; Glazener et al., 1967; Trainer and Hanson,

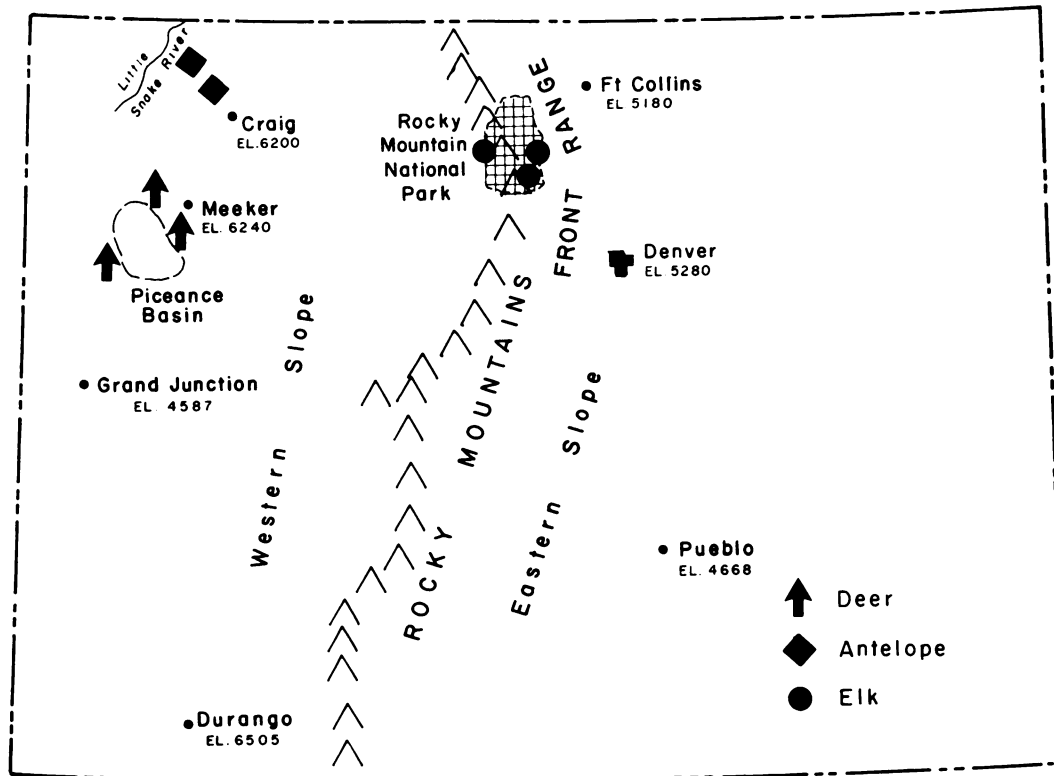


FIGURE 1. Sites in Colorado, showing wildlife trapping and capture areas for blood collection for serologic studies on vesicular stomatitis, New Jersey (VSNJ) virus.

1969; Jenney et al., 1975; Fletcher et al., 1985). Although some speculation concerning latent infection of vertebrates had been proposed (Hanson and Brandly, 1957; Mason, 1978), the first real evidence that reactivation of lesions following stress occurred during the 1982 outbreak. Shipment of cows that were either asymptomatic, clinically recovered, or with healing lesions of VS infection from Colorado to other states resulted in exacerbation of clinical disease and the introduction of VS to five states not known previously to have infected cattle (Buisch, 1983). The second impetus to examine the question of an existing enzootic focus came from isolations of VSNJ virus from wild-caught *Culicoides* spp. (Walton et al., 1987), Simuliidae, and various muscoid flies (Francy et al., unpubl. data) during the 1982 epizootic. In light of newer and more precise sero-

logic techniques and the isolation of VS from insects, it seemed timely to reevaluate the role of wildlife as reservoir hosts and reexamine the possibility that an enzootic focus existed in Colorado. Animal sera collected in years both before and after the epizootic were studied.

MATERIALS AND METHODS

Serosurveys were made on samples collected from pronghorn, mule deer, and elk with the cooperation of, and in conjunction with, personnel of the Colorado Division of Wildlife (CDW) and Colorado State University (CSU), Department of Pathology. Pronghorn sera were collected in 1983 from animals trapped in the Little Snake River Drainage north of Maybell, Moffat County, Colorado, 18–20 January (Fig. 1). A helicopter was used to drive the pronghorn into a large funnel-shaped fence where, at the narrow end, a modified drop net catch pen (Schmidt, 1976) was used to immobilize the animals. Deer sera were received from Dr. C. Hib-

TABLE 1. Neutralizing (N) antibodies to vesicular stomatitis, New Jersey (VSNJ) virus in sera collected from wildlife in Colorado, before and after a 1982 epizootic.

Species	Area collected ^a	Year	VSNJ N antibody	Prevalence (%)
Pre-epizootic				
Elk (<i>Cervus elaphus</i>)	Rocky Mountain	1980	3/81 ^b	3.7
	National Park	1981	6/58	10.3
Mule deer (<i>Odocoileus hemionus</i>)	Piceance Basin	1975	2/52	3.8
Post-epizootic				
Elk	Moffat County	1985	2/15	13.3
Mule deer	Piceance Basin	1982 ^c	11/59	18.6
Mule deer	Piceance Basin	1983 ^c	14/69 (adults)	20.2
			5/81 (fawns)	6.2
Mule deer	Piceance Basin	1984 ^c	13/65 (adults)	20.0
			2/105 (fawns)	1.9
Pronghorn (<i>Antilocapra americana</i>)	Craig ^d	1982	67/139	48.2
Pronghorn	Moffat County	1983	67/153	43.7

^a See Figure 1.^b Number positive/number tested (only sera with $\geq 1:40$ titer were considered positive).^c Sera were collected by Scribner et al. during a study by the Savannah River Ecology Group.^d Killed by hunting.

ler, CSU, and from Mr. Kim Scribner of the Savannah River Ecology Project. All sera were from mule deer live-trapped at 23 pre-baited permanent trap sites in the Piceance River basin of Colorado (Fig. 1). Animals were bled by jugular venipuncture, and the samples were stored on wet ice in the field. Upon completion of electrophoretic analysis testing of the samples, an aliquot of serum was sent to the Centers for Disease Control (CDC) for VS serology.

Elk were trapped with clover traps (Clover, 1954) at multiple sites on the eastern slope of the Rocky Mountain National Park (RMNP) (McLean et al., 1981) by personnel of CDW and the National Park Service. Blood samples were obtained also by jugular venipuncture, returned immediately to the laboratory, centrifuged, and sera removed and stored at -20°C until tested. Rodents were captured live in Sherman or National traps on several premises in Colorado where livestock were affected by VS during the 1982 epizootic; they were bled from the orbital sinus and released. Additional rodent and carnivore sera collected from Colorado were provided by the Plague Branch, Division of Vector-Borne Viral Diseases (DVBVD), CDC, Fort Collins.

Sera were heated at 56°C for 30 min before testing by plaque reduction N test in Vero cell monolayers (Lindsey et al., 1976). Sera were screened at a 1:20 dilution, and positive reactors

were then retested at serial dilutions to determine endpoint titers. The N titers are expressed as the reciprocal antibody dilution producing 80% or greater plaque reduction. For the wild ungulate sera, only N titers of 40 were considered positive. All sera were tested against both VSNJ Hazelhurst and VS Indiana (VSI) Lab strain viruses as a further check on specificity.

A breeding colony of deer mouse (*Peromyscus maniculatus*) was established in early 1983 from field caught animals in Larimer County, Colorado. Wood rats (*Neotoma mexicana*) were live-trapped in June 1983. The offspring of pregnant females were reared and used for experimental inoculation. The virus strain used to inoculate these animals was a field isolate of VSNJ obtained from a mixed pool of 24 male and deplete female Simuliidae collected in a CDC light trap during the epidemic. The original suspension was passed twice in cloned *Aedes albopictus* (C6/36) cell culture to prepare seed suspension for experimental studies.

RESULTS

Serologic surveys: N antibody to VSNJ was found in 4% of elk trapped in the RMNP (Fig. 1) in 1980 before the epizootic and 10% of elk in 1981 (Table 1). A similar increase in the prevalences of VSNJ

TABLE 2. Neutralizing (N) antibodies to vesicular stomatitis, New Jersey (VSNJ) virus in sera collected from wild rodents in Colorado, 1982.

Species	VSNJ N antibody	
	Number positive/ number tested	%
<i>Neotoma mexicana</i> (Mexican wood rat)	6/48	12.5
<i>Peromyscus maniculatus</i> (deer mouse)	8/66	12.1
<i>P. difficilis</i> (rock mouse)	3/47	6.4
<i>Mus musculus</i> (house mouse)	1/41	2.4
<i>Spermophilus variegatus</i> (rock squirrel)	0/27	—
<i>S. tridecemlineatus</i> (thirteen-lined ground squirrel)	0/19	—
<i>Tamias</i> sp. (chipmunk)	0/7	—

antibody in deer was recorded in 1975 and 1982–1984 in the Piceance River basin (Fig. 1). The annual prevalences in deer during 1982 and 1983–1984 were similar (Table 1). The deer fawns were about 6 mo old, and the antibody in five of 81 (6%) sampled may represent maternal antibody. Antibody prevalences in pronghorns from northwestern Colorado (Fig. 1) were higher in 1982 than the prevalences in adult mule deer (48% and 44% versus 19% and 20%, respectively) (Table 1).

N antibody was found in one of 17 coyotes (*Canis latrans*). This seropositive animal had been trapped in Larimer County of northern Colorado in 1981 as part of a plague investigation study. Prevalences in domestic dogs (*Canis familiaris*), sampled during a plague survey in various western states, ranged from 0 to 29% (Table 3). The sera from dogs in Colorado all came from pets that were bled on premises with affected livestock during the VS epizootic. Six of 21 dogs (29%) had N antibody titers of ≥ 80 . Among eight dogs from one dairy farm where active disease in livestock was in progress, four of the dogs had titers of ≥ 640 . The other two antibody-positive dogs resided on a horse range and were bled 3 mo after the active disease occurred

TABLE 3. Neutralizing (N) antibodies to vesicular stomatitis, New Jersey (VSNJ) in sera collected from canids in the western United States, 1982.

State	Species*	VSNJ N antibody	%
California	Dog	15/79 ^b	19.0
New Mexico	Dog	16/225	6.3
Montana	Dog	5/19	26.3
Utah	Dog	0/9	
Wyoming	Dog	1/10	10.0
Colorado	Dog	6/21	28.5
Colorado	Coyote	1/17	5.8

* Dog (*Canis familiaris*); coyote (*Canis latrans*).

^b Number positive/number tested.

in horses. Two of three dogs sampled on this premise had titers of ≥ 80 and ≥ 320 .

Experimental studies: Wood rats and two species of *Peromyscus* collected during the epizootic had high N antibody prevalences (Table 2). Results of attempts to infect colonized *P. maniculatus* and field-trapped wood rats held in the laboratory for some months were not highly elucidating. During laboratory studies, viremia was detected for 1 day each in two of 18 deer mice following intramuscular inoculation. However, all 13 survivors developed N antibody when tested 21 days after inoculation. None of the animals became ill or developed any lesions. Most deaths were attributed to trauma of handling. Viremia or illness was not detected in wood rats, but all survivors developed N antibody.

DISCUSSION

The finding of N antibody in elk and deer before onset of the epizootic indicates that VSNJ virus was present in Colorado for at least 2 yr before the outbreak in domestic animals and that virus amplification may have occurred during the year preceding the epizootic (1981). It was also apparent that VSNJ transmission may have occurred at high elevations since seropositive elk were found in the Rocky Mountain National Park at altitudes above 2,400 m. Elk are known to undertake long

seasonal migrations between winter and summer ranges, although some elk herds are essentially nonmigratory (Peck, 1982). Movement onto winter range is initiated with the first snowstorm, when foraging areas are decreased by accumulation of snow. The timing of spring movement to summer range is often related to the presence of tabanid flies and development of vegetation (Peck, 1982).

Although some elk are known to live to 20 yr of age, the mean life expectancies are much lower (bulls 2.7–3.0 yr, cows 2.1–4.9 yr) (Peck, 1982). Therefore, it is unlikely that antibody persisted at detectable levels in elk from the last epizootic in Colorado in 1966, when both VSNJ and VSI serotypes were present. In 1972, there were 19 cases of VS confirmed in horses and cattle in Colorado, New Mexico, and Louisiana. Only five cases were confirmed in Colorado; four from LaPlata County and one from Mesa County (Jenney et al., 1980).

The N antibody titers of seropositive ungulates sampled prior to the 1982 epizootic were all 40, except for one elk with a titer of >320. High N titers would not be expected during an enzootic period of low transmission, as was found with VSNJ antibody in white-tailed deer from Ossabaw Island, Georgia in 1981 (Stallknecht and Erickson, 1986). High N antibody titers usually represent recent infections and are of short duration, except in previously infected animals receiving virus boosters. Succeeding natural exposures or boosters to vector-borne viral diseases should be rare during an enzootic period of low virus activity, even in a long-lived ungulate, but common during periods of high virus activity in local areas.

Higher N antibody prevalences and titers were found in wild ungulates during and after the 1982 VSNJ epizootic in Colorado, indicating significant exposure of these wildlife populations to the virus. The finding of antibody in a wildlife species in a particular area does not confirm its role as a natural reservoir host, but merely in-

dicates its exposure to and infection with the virus. Such species can at least be used as sentinels, as with the white-tailed deer in Georgia (Stallknecht and Erickson, 1985), and also are candidates for further investigation to determine if enzootic wildlife reservoirs of VSNJ exist in Colorado.

A few attempts to artificially infect wildlife have been made. Karstad and Hanson (1957) reported the occurrence of fever, vesicles, and mouth lesions in three of four deer infected by introducing virus onto tongue abrasion. They were unable to detect viremia at 50 and 72 hr postinoculation. If viremia does occur, it must be transient. However, virus can be reisolated from vesicular fluid and tongue tissue. High levels of N antibody develop quickly after infection. Experimental inoculation of two pronghorns (Thorne et al., 1983) with VSNJ virus by the intra-dermolingual route produced results in an adult doe similar to those reported in deer, but clinical signs of VS were not noted in a yearling pronghorn doe. VS virus was isolated from tongue tissue, but not from blood 3 days postinoculation. Likewise, it is extremely difficult to produce severe disease, such as occurs in naturally acquired VS infection, in horses or cows following experimental virus inoculation. Whether wild ruminants suffer severe clinical illness in a natural setting remains unproven. However, if salivation occurs and oral vesicles develop in an animal that depends on grazing for its food, weight loss and weakness quickly follow. We believe ungulates are epizootic hosts at least and may serve as a source of infection for vectors.

It has been suggested that a latent carrier state might exist in cattle, and evidence of animals developing VS disease following shipment was reported by Hanson 30 years ago (Hanson and Brandly, 1957). Suggestions that fluctuating N antibody titers could be due to persistent infection with periodic reshedding of virus, followed by return to a latent state (So-

rensen et al., 1958), were largely speculative. In the 1982 epizootic, a shipment of cattle that had recovered from clinical VS disease from a dairy dispersal sale in Colorado resulted in the appearance of disease in several states not previously involved (Buisch, 1983), and cattle with previously healed lesions were observed to develop new lesions upon arrival. Wild ungulates are subjected to many stresses during harsh winters and by man's encroachment onto their grazing land and frequently intermingle with domestic livestock. Mule deer, in particular, browse freely with cattle in Colorado. Pronghorn feed from hay stacks kept for cattle. Elk are less often seen intermingling with cattle, because cattle are not kept in large numbers at higher altitudes. However, they may share salt licks and drinking sources.

Dogs have been reported to be resistant to artificial inoculation (Brandly et al., 1951; Kowalczyk and Brandly, 1954). Suspected, but unverified cases of disease were reported in two dogs residing on infected premises during earlier VS outbreaks (Brandly et al., 1951). Although only a few dogs were bled on each premise during the 1982 epizootic, we did find N antibody titers suggestive of recent infection on two of the affected farms. Of the owners questioned, only one described a recent illness in his dog, but no vesicular lesions were observed. It is interesting that a coyote was found with N antibody. Since coyotes are carnivores and rodent predators, it is possible they might acquire their infection by feeding on dead ungulates or infected rodents.

In summary, we note that antibody prevalences in wild ungulates are sufficiently high to indicate significant involvement. These animals often graze with domestic herds and could serve as a source of infection or become infected themselves from the domestic animals. In severe winters, such as occurred in 1982, increased exposure may have resulted from sharing range pasture. In addition, stress induced

by low temperatures could have increased susceptibility or caused shedding of latent virus.

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We are particularly fortunate to have discovered that a study on heterozygosity of mule deer herds in the Piceance River Basin was being conducted by the Savannah River Ecology Project in conjunction with Messrs. R. Bartman and K. Scribner who readily agreed to send us aliquots of sera and field notes on all animals. Radio collars had been placed on the animals, and further studies continued in 1984 and 1985.

LITERATURE CITED

- ALDERINK, F. J. 1984-1985. Vesicular stomatitis epidemic in Colorado: Clinical observations and financial losses reported by dairymen. *Preventive Veterinary Medicine* 3: 29-44.
- BRANDLY, C. A., R. P. HANSON, AND T. L. CHOW. 1951. Vesicular stomatitis with particular reference to the 1949 Wisconsin epizootic. *Proceedings Book of the American Veterinary Medical Association*, pp. 61-66.
- BUSCH, W. W. 1983. Fiscal year 1982-83 vesicular stomatitis outbreak. *Proceedings of the United States of America Health Association* 87: 78-84.
- CLOVER, M. R. 1954. A portable deer trap and catch-net. *California Fish and Game* 40: 367-373.
- FLETCHER, W. O., D. E. STALLKNECHT, AND E. W. JENNEY. 1985. Serologic surveillance for vesicular stomatitis virus on Ossabaw Island, Georgia. *Journal of Wildlife Diseases* 21: 100-104.
- GLAZENER, W. C., R. S. COOK, AND D. O. TRAINER. 1967. A serological study of diseases in the Rio Grande turkey. *Journal of Wildlife Management* 31: 34-39.
- HANSON, R. P., AND C. A. BRANDLY. 1957. Epizootiology of vesicular stomatitis. *American Journal of Public Health* 47: 205-209.
- JENNEY, E. W., G. A. ERICKSON, W. W. BUSCH, W. C. STEWART, AND M. A. MIXSON. 1980. Surveillance for vesicular stomatitis in the United States in 1972 and 1979. *American Association of Veterinary Laboratory Diagnosticians, 23rd Annual Proceedings*, pp. 83-90.

- , F. A. HAYES, AND C. L. BROWN. 1970. Survey for vesicular stomatitis virus neutralizing antibodies in serums of white-tailed deer (*Odocoileus virginianus*) of the southeastern United States. *Journal of Wildlife Diseases* 6: 488–493.
- , ———, AND ———. 1975. Survey for vesicular stomatitis infection in Georgia wild mammals. *In* Developmental studies and laboratory investigations conducted by Veterinary Service Diagnostic Laboratories. FY 73. APHIS-USDA, Animal and Plant Health Inspection Service, pp. 47–50.
- KARSTAD, L. H., E. V. ADAMS, R. P. HANSON, AND D. H. FERRIS. 1956. Evidence for the role of wildlife in epizootics of vesicular stomatitis. *Journal of the American Veterinary Medical Association* 129: 95–96.
- , AND R. P. HANSON. 1957. Vesicular stomatitis in deer. *American Journal of Veterinary Research* 68: 162–166.
- KOWALCZYK, T., AND C. A. BRANDLY. 1954. Experimental infection of dogs, ferrets, chinchillas, and hamsters with vesicular stomatitis virus. *American Journal of Veterinary Research* 15: 98–101.
- LINDSEY, H. S., C. H. CALISHER, AND J. MATHEWS. 1976. Serum dilution neutralization test for California Group virus identification and serology. *Journal of Clinical Microbiology* 4: 503–510.
- MASON, J. 1978. The epidemiology of vesicular stomatitis. *Boletín de la Centro Panamericana Fiebre Afosa* 29–30: 35–53.
- MCLEAN, R. G., D. B. FRANCY, G. S. BOWEN, R. E. BAILEY, C. H. CALISHER, AND A. M. BARNES. 1981. The ecology of Colorado tick fever in Rocky Mountain National Park in 1974. I. Objectives, study design and summary of principal findings. *American Journal of Tropical Medicine and Hygiene* 30: 483–489.
- PECK, J. M. 1982. Elk. *In* Wild mammals of North America: Biology, management, economics. J. A. Chapman and G. A. Feldhamer (eds.). Johns Hopkins University Press, Baltimore, Maryland, pp. 851–861.
- SCHMIDT, R. 1976. Baiting bighorn sheep with apple pulp and trapping with a drop net. *Proceedings of the Biennial Symposium of the Northern Wild Sheep Council*, pp. 26–34.
- SORENSEN, D. K., T. L. CHOW, T. KOWALCZYK, R. P. HANSON, AND C. A. BRANDLY. 1958. Persistence in cattle of serum neutralizing antibodies to vesicular stomatitis. *American Journal of Veterinary Research* 19: 74–77.
- STALLKNECHT, D. E., AND G. A. ERICKSON. 1986. Antibodies to vesicular stomatitis New Jersey type virus in a population of white-tailed deer. *Journal of Wildlife Diseases* 22: 250–254.
- THORNE, E. T., E. S. WILLIAMS, W. J. ADRIAN, AND C. M. GILLIN. 1983. Vesicular stomatitis in pronghorn antelope: Serologic survey and artificial infection. *Proceedings of the 87th Annual Meeting of the U.S. Animal Health Association*, Las Vegas, Nevada, pp. 638–643.
- TRAINER, D. O., AND R. P. HANSON. 1969. Serologic evidence of arbovirus infections in wild ruminants. *American Journal of Epidemiology* 90: 354–359.
- WALTON, T. E., P. A. WEBB, W. L. KRAMER, G. C. SMIT, T. DAVIS, F. R. HOLBROOK, C. G. MOORE, T. J. SCHIEFER, R. H. JONES, AND G. C. JANNEY. 1987. Epizootic vesicular stomatitis in Colorado, 1982: Epidemiologic and entomologic studies. *American Journal of Tropical Medicine and Hygiene* 36: 166–176.

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