BioOne COMPLETE

ANTIBODY PREVALENCE OF EIGHT RUMINANT INFECTIOUS DISEASES IN CALIFORNIA MULE AND BLACK-TAILED DEER (ODOCOILEUS HEMIONUS)

Authors: Chomel, Bruno B., Carniciu, Marius L., Kasten, Rickie W., Castelli, Paolo M., Work, Thierry M., et al.

Source: Journal of Wildlife Diseases, 30(1) : 51-59

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-30.1.51

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

Journal of Wildlife Diseases, 30(1), 1994, pp. 51–59
© Wildlife Disease Association 1994

ANTIBODY PREVALENCE OF EIGHT RUMINANT INFECTIOUS DISEASES IN CALIFORNIA MULE AND BLACK-TAILED DEER (*ODOCOILEUS HEMIONUS*)

Bruno B. Chomel,[1] Marius L. Carniciu,[1] Rickie W. Kasten,[1] Paolo M. Castelli,[1] Thierry M. Work,[2] and David A. Jessup[2]

[1] University of California, School of Veterinary Medicine, Department of Epidemiology and Preventive Medicine, Davis, California 95616, USA

[2] California Department of Fish and Game, Wildlife Investigation Laboratory, 1701 Nimbus Road, Suite D, Rancho Cordova, California 95670, USA

ABSTRACT: We tested 276 sera from 18 free-ranging black-tailed and mule deer (*Odocoileus hemionus*) herds in California (USA) collected from 1987 to 1991 in five biogeographical habitat types, for antibodies against eight infectious disease agents. Overall antibody prevalence was 56% for *Anaplasma marginale*, 31% for *Borrelia burgdorferi*, 16% for bluetongue virus serotype 17, 15% for epizootic hemorrhagic disease virus, 7% for *Coxiella burnetii* and *Toxoplasma gondii*, respectively, and 0% for bovine leukosis virus and caprine arthritis/encephalitis virus, respectively. Antibodies against Lyme borreliosis and anaplasmosis were found in deer throughout California, but antibodies against bluetongue and epizootic hemorrhagic disease were most prevalent in deer from southern California.

Key words: Deer, *Odocoileus hemionus*, California, serologic survey, anaplasmosis, bluetongue virus, bovine leukosis, caprine arthritis/encephalitis-ovine progressive pneumonia, epizootic hemorrhagic disease, Lyme borreliosis, Q fever, toxoplasmosis.

INTRODUCTION

In many areas, deer (*Odocoileus* spp.) and domestic ruminants share the same habitat and are exposed to the same infectious agents. Ranching of deer adjacent to or in common with livestock is increasing in the United States. Infectious agents may not only be transmitted from wildlife to livestock but also from livestock to wildlife, and occasionally to humans. The ecological and geographical circumstances that lead to transmission may vary considerably from one area to another. Wild ruminants and other wildlife may serve as sentinels or reservoirs for diseases of cattle and small ruminants (Trainer, 1970).

Several surveys for a variety of agents already have been conducted in free-ranging California (USA) deer populations (Behymer et al., 1989; Campbell et al., 1989; Enright et al., 1971). However, most either were geographically limited or directed towards bacterial infections. Furthermore, investigation of exposure to retroviral agents in livestock has never been reported in free-ranging deer in California. Thus, our purpose was to determine the prevalence of antibodies to eight viral and bacterial infectious agents in 18 herds of black-tailed deer (*Odocoileus hemionus columbianus*), California mule deer (*O. hemionus californicus*), Rocky Mountain mule deer (*O. hemionus hemionus*) and southern mule deer (*O. hemionus fulginatus*) in California. Most herds had not been tested previously for these various agents. We also sought to determine the prevalence of exposure by herd location and subspecies, and to determine temporal trends by comparison with previous surveys conducted in similar areas.

MATERIALS AND METHODS

Two hundred and seventy-six serum samples were collected from 18 deer herds located in 15 California counties (Fig. 1) between 1987 and 1991. There were 143 sera from black-tailed deer (*Odocoileus hemionus columbianus*), 60 from Rocky Mountain mule deer (*O. hemionus hemionus*), 47 from southern mule deer (*O. hemionus fulginatus*) and 26 from California mule deer (*O. hemionus californicus*). Samples were collected from hunter-killed and live-trapped deer by California State Department of Fish and Game personnel. Bleeding was by cardiac puncture in hunter-killed deer and jug-

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

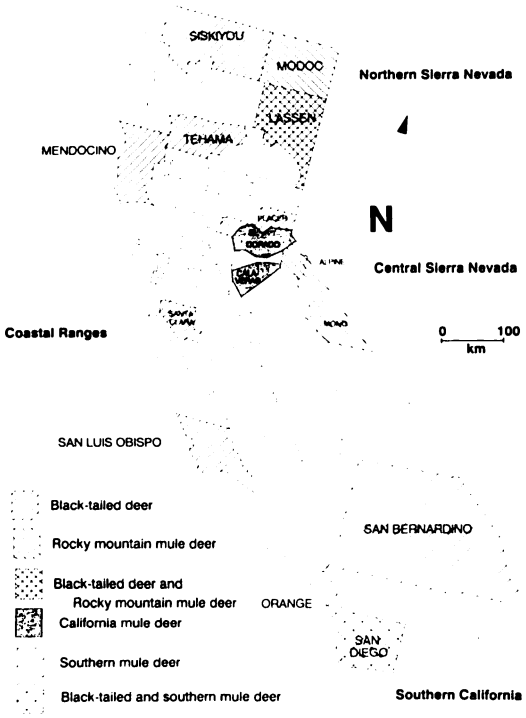


FIGURE 1. Deer herd locations by county and subspecies, California, 1987 to 1991.

ular venipuncture in live-trapped deer. Blood samples were stored at 4 C shortly after collection and centrifuged within 24 hr. Sera were stored at −20 C until tested. Sex was noted for each animal. Dates of collection were grouped into spring (April, May, June), summer (July, August, September), fall (October, November, December), and winter (January, February, March) for each year. Age was estimated by tooth wear (Robinette et al., 1957).

The survey was conducted among 18 herds in 15 counties that were grouped into four distinct ecosystems.

In the Coastal Ranges (34°N to 41°N, 119°W to 124°W), 18 black-tailed deer came from Mendocino County (Mendocino herd), 15 black-tailed deer came from Santa Clara County (Mount Hamilton herd), and 11 black-tailed deer came from San Luis Obispo County (Adelaida herd). In southern California (33°30'N to 36°N, 114°W to 119°W), 17 southern mule deer came from Orange County (Santa Ana mountains herd), 14 southern mule deer came from San Bernardino County (San Bernardino Mountains herd), 16 southern mule deer and 16 black-tailed deer came from San Diego county (San Diego herds). In the northern Sierra (40°N to 42°N, 120°W to 124°W), 16 black-tailed deer came from Tehama County (East Tehama herd), 36 black-tailed deer came from Siskiyou County (19 from the Happy Camp herd and 17 from the McCloud Flat herd), 20 black-tailed deer and 17 Rocky Mountain mule deer came from Lassen County (Doyle herd and West Lassen herd, respectively), and 12 Rocky mountain deer came from Modoc County (Adin herd). In central Sierra (36°N to 40°N, 117°W to 123°W), 14 Rocky Mountain mule deer came from Alpine County (Carson River herd), 17 Rocky Mountain mule deer came from Mono County (Round Valley herd), 13 California mule deer came from Calaveras County (Mother Lode herd), 13 California mule deer came from El Dorado County (Pacific herd) and 11 black-tailed deer came from Placer County (Blue Canyon herd).

Sera were tested for antibodies against bovine leukemia virus (BLV), caprine arthritis/encephalitis virus (CAEV), epizootic hemorrhagic disease (EHD) virus, bluetongue (BT) virus, *Toxoplasma gondii*, *Coxiella burnetii*, *Anaplasma marginale* and *Borrelia burgdorferi*.

We tested for antibodies against BLV, CAEV and EHD using commercial agar gel immunodiffusion test (AGID) kits (BLV antibody test kit: Leukassay B, Pittman-Moore, Inc., Washington Crossing, New Jersey, USA; CAEV and EHD antibody kits, Veterinary Diagnostic Technology, Inc., 4890 Van Gordon St. Suite 101, Wheat Ridge, Colorado, USA). For these qualitative tests, results were reported as negative or positive.

Antibodies to *T. gondii* were detected using a commercial indirect latex agglutination test kit (Toxotest-MT "Eiken," Eiken Chemical Co., Ltd., 33-8, Hongo 1-chome, Bunkyo-ku, Tokyo, Japan). Any serum with a titer ≥1:32 was considered as positive (1:32 = low positive, 1:64 to 1:512 = positive, ≥1:1,024 = high positive).

Tests for antibodies to bluetongue, Q fever, Lyme disease and anaplasmosis were performed using an enzyme linked immunosorbent assay (ELISA). The antigens used to sensitize microtiter plates for testing black-tailed deer sera by ELISA were as follows: *A. marginale* was obtained from the U.S. Department of Agriculture; it was a complement fixation antigen and was tested at a > 1:200 dilution. The *C. burnetii* antigen was from a trichloroacetic acid extract antigen and used at a ≥1:50 dilution. The *B. burgdorferi* antigen was from a sonicated extract antigen and used at a ≥1:200 dilution. We used a BT virus 17 sonicated extract antigen at a ≥1:400 dilution. Antigens were tested with known positive serum samples from naturally or experimentally infected (anaplasmosis, bluetongue and Lyme borreliosis) or vaccinated (Q fever) deer and titrated for optimal working dilution. Negative controls were constituted by a pool of sera from serologically negative deer

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

herds, defined as having optical density (OD) values <20% of the OD value of the positive control serum.

The ELISA was based on the official United States Department of Agriculture (USDA) method for pseudorabies (Snyder et al., 1977; Behymer et al., 1985), modified as follows. Briefly, 50 μl per well of the optimal antigen dilution in carbonate bicarbonate buffer, pH 9.6, was bound to 96-well flat bottom microtitration plates (Linbro/Titertek, Flow Laboratories, Inc., McLean, Virginia, USA) by overnight incubation at 4 C. Sera were diluted 1:100 (Q fever, bluetongue and anaplasmosis) or 1:200 (Lyme disease) in tris buffer (pH 7.4) containing 0.05% tween 20 and 0.01% bovine serum albumin fraction V (Sigma Chemical Co., St. Louis, Missouri, USA). The peroxidase conjugate was a rabbit anti-deer antibody at 1:1,000 (rabbit anti-deer IgG, Kirkegaard & Perry Laboratories, Inc., Gaithersburg, Maryland, USA). The substrate was 2,2′-azino-bis(3-ethylbenzthiazoline-6-sulfonic acid; ABTS) (Sigma Chemical Co., St. Louis, Missouri). The reaction was stopped after 30 min with 100 μl of a 0.1 M solution of hydrofluoric acid (pH 3.3).

Each plate contained known positive and negative control sera. Each serum was tested in duplicate and the mean of the two absorbance values calculated. If the standard deviation between the duplicate values exceeded 0.05 percent, the sample was retested. If the absorbance of the positive control divided by the absorbance of the negative control (P/N ratio) was less than five, the test was repeated.

Microtitration plates were read at 410 and 450 nm wavelengths respectively for test and reference on a microelisa autoreader (MR 5000, Dynatech Laboratories, Inc., Chantilly, Virginia). The cut-off point (COP) for a positive test was determined as the mean optical density (OD) of the negative control population plus three standard deviations (SD) (Richardson et al., 1983; Magnarelli et al., 1991). The COP for the various antigens were respectively OD = 0.200 for *C. burnetii*, OD = 0.400 for *A. marginale* and OD = 0.450 for *B. burgdorferi* and BT virus 17.

For *C. burnetii* antibodies, an OD > 0.200 and <0.250 was considered as low positive; high positive sera had an OD ≥ 0.300. For *A. marginale* and *B. burgdorferi* antibodies, sera with OD ≥ 0.600 were considered as high positive; low positive sera had an OD > 0.400 and <0.500. Threshold values for bluetongue virus were respectively OD > 0.450 and OD < 0.500 for the low positive samples; sera with an OD ≥ 0.650 were considered high positive sera.

The data were compiled and analyzed using Epi Info version 5.01 (Dean et al., 1990). Frequency distributions were obtained from Analysis program (Epi Info subdirectory) and chi-square statistics for 2 × 2 contingency tables were calculated to obtain measures of association, and the statistical significance of such associations. Odds ratios also were calculated with this program.

RESULTS

The overall antibody prevalences ranged from a high of 56% for *A. marginale* to 0% for BLV and CAEV. Over 30% of the deer had antibodies against anaplasmosis and Lyme borreliosis. High antibody titers (OD ≥ 0.600 for *A. marginale* and *B. burgdorferi*; OD ≥ 0.650 for BT virus) were observed primarily against *Anaplasma* (30%), *B. burgdorferi* (17%) and bluetongue virus (13%).

Black-tailed deer, the largest subspecies group tested, had the highest antibody prevalences against *A. marginale* (63%) and *C. burnetii* (10%). Based on an odds ratio (OR), black-tailed deer were more likely (OR = 3.8; 95% confidence interval (CI) = 1.2, 16.0; $P = 0.03$) to have *C. burnetii* antibodies than any other subspecies.

The highest prevalence of antibodies against *B. burgdorferi* (50%) and *T. gondii* (15%) were observed in California mule deer. This subspecies was the only one negative for antibodies against BT and EHD and had very low prevalence (3.8%) of Q fever antibodies.

Southern mule deer had the highest prevalence of antibodies for BT (38%) (OR = 4.6; 95% CI = 2.1, 10.0; $P < 0.001$) and EHD (45%) (OR = 8; 95% CI = 3.6, 17.6; $P < 0.001$). The southern mule deer also was the only subspecies in which there was no evidence of Q fever antibody.

Age and sex were available for 254 (92%) of the deer population tested. Adult deer (≥2 yr-old) of all subspecies were more likely (OR = 3; 95% CI = 1.25, 8.0; $P = 0.01$) to have antibodies against *A. marginale* (48%) than deer <2 years old (23%). Overall, adult deer had a higher prevalence of antibodies than the young deer against all agents except for Lyme borre-

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

liosis. There were no significant differences by sex with respect to antibodies against any of the organisms tested.

Antibody prevalence against *T. gondii* was highest in winter (11%, $n = 122$) and fall (6.8%, $n = 44$), compared with spring (2.0%, $n = 49$) and summer (1.6%, $n = 61$). Deer were more likely (OR = 3.8; 95% CI = 1.3, 14.0; $P = 0.01$) to have *T. gondii* antibodies if they were bled during the winter compared with the three other seasons. Antibody prevalences were the highest for anaplasmosis and Lyme borreliosis in summer and spring; whereas, antibody prevalences for bluetongue, EHD and Q fever were the highest in the fall (27%, 20%, and 14%, respectively).

Exposure to Lyme borreliosis was found to be widespread among the four areas of the state sampled. The antibody prevalence to *B. burgdorferi* also was widespread and ranged from 28% to 34%. Among individual herds, prevalence ranged from 6% in the Round Valley herd (Mono County) to 77% in the Pacific herd (El Dorado County). Antibody prevalence was similar for winter (34%), spring (37%), and summer (34%), but much lower during the fall (14%).

The prevalence of *A. marginale* antibodies ranged from 40% in the central Sierra to 75% in the Coastal Ranges. Deer from the central Sierra were more likely to be free of antibodies than the deer of the other geographical areas (OR = 0.42, 95% CI = 0.23, 0.76; $P = 0.003$). Among individual herds, antibody prevalence ranged from 0% in the Carson River herd (Alpine County) to 100% in the Mendocino herd (Mendocino County). Eight of the 18 herds had antibody prevalences of ≥60%, indicating widespread exposure in California.

Antibodies against BT virus and EHD virus were observed mainly in southern California where, respectively 44% and 48% of the deer had antibodies. Deer in southern California were more likely to have BT (OR = 9.2; 95% CI = 4.3, 19.8; $P < 0.001$) and EHD (OR = 15.2; 95% CI = 6.7, 35.6; $P < 0.001$) antibodies than those from the three other areas. Antibodies against BT and EHD were more limited geographically than anaplasmosis; five of the 18 herds had no evidence of exposure.

Antibodies to *C. burnetii* were observed primarily in the herds from the northern Sierra (12%; $n = 101$) and the Coastal Ranges (9%, $n = 44$). Deer in the northern Sierra were more likely (OR = 3; 95% CI = 1.1, 10.0; $P = 0.02$) to have antibodies to *C. burnetii* than the deer from the three other areas. Exposure to *Coxiella burnetii* was evident in nine of the 18 herds, and antibody prevalence was the highest (30%; $n = 20$) in the Doyle herd (Lassen County).

Herds with antibodies against *T. gondii* were mainly from the central Sierra (10%, $n = 68$) and southern California (9.5%, $n = 63$). Half of the 18 herds had antibodies to *T. gondii*. The highest antibody prevalences were observed in the Pacific herd (31%, $n = 13$) (El Dorado County), and the Doyle herd (20%, $n = 20$) (Lassen County).

Serum cross-reactivity between the BT antigen and the EHD antigen was observed as animals were positive to both tests with a frequency much higher than expected if the two variables were independent (OR = 54.4 95% CI = 20.3, 149.2; $P < 0.001$).

DISCUSSION

No herds had antibodies against the retrovirus causing bovine leukemia in cattle, or the lentiviruses causing caprine arthritis and encephalitis (CAEV) in goats or ovine progressive pneumonia (OPP) in sheep. In California, bovine leukemia infection has been reported in dairy cattle (Thurmond et al., 1985), with an antibody prevalence of 10% to 30% in slaughtered Holstein cattle from the southern San Joaquin valley. Deer do not commonly overlap with cattle in these areas and under these husbandry conditions.

In Europe, two cases of leukosis tumor were reported in Germany in roe deer (*Ca-*

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

preolus capreolus) (Brömel and Zettl, 1973) and two deer had antibodies to BLV: a roe deer near Frankfurt, Germany (Weber et al., 1978) and a roe deer near Haute Marne, France (Baradel et al., 1988). However, no BLV antibodies were detected in Germany among 255 deer, including roe deer, red deer (*Cervus elaphus*), fallow deer (*Dama dama*), and mouflons (*Ovis ammon musimon*) (Weber et al., 1978), or in France among 193 roe deer (Blancou, 1983), respectively. Our results are in agreement with these previous findings; BLV does not appear to be present in wild deer in California.

Caprine arthritis encephalitis virus (CAEV) is widespread in dairy goats in the United States (Crawford and Adams, 1981). In California, East et al. (1987), reported that 53% of goats in 13 goat dairies had antibodies to CAEV by the AGID test. Most infected flocks are found in the Central Valley and contact between wild ruminants and dairy goats is very unlikely. Ovine progressive pneumonia (OPP) also is common in sheep flocks in the United States (Bulgin, 1990; Cutlip et al., 1992). Our results are in agreement with those of Cutlip et al. (1991) who did not find any seropositive white-tailed deer (*Odocoileus virginianus*) among 217 deer tested for OPP from different regions of the United States. Furthermore, their attempt to experimentally infect four white-tailed deer was not successful, and is evidence for a lack of susceptibility of deer to this lentivirus. Similarly, Clark et al. (1985) and Cutlip et al. (1991) did not detect any antibodies against OPP among the California and Wyoming (USA) bighorn sheep (*Ovis canadensis*) tested.

The most common infectious agent detected among the California deer tested in this study was *A. marginale*. Deer and other wild ruminants are probable reservoirs of *A. marginale* infection (Christensen et al., 1960). The Pacific Coast tick (*Dermacentor occidentalis*) appears to be the most efficient vector of anaplasmosis in California, feeding on both wild ruminants and cattle (Christensen et al., 1962). An antibody prevalence of 56% was found in our study, the highest percentage being in black-tailed deer (63%), especially these living in the coastal ranges and northern Sierra Nevada, as previously reported by Christensen and McNeal (1967), and Utterback et al. (1972). More specifically, in the five herds (Adelaida, Carson River, East Tehama, Mendocino and Mount Hamilton) tested in our study and tested a few years earlier by Behymer et al. (1989), prevalence was higher in three (Adelaida, East Tehama, Mendocino) of the five herds in our study, and the Carson River herd remained anaplasmosis free (Table 1). Thus there is evidence that in recent years, the diffusion of anaplasmosis within the herds has been active. However, such differences also could be related to the size of the sample from each herd and the time of the year the sampling was performed. As reported by Kuttler (1984), of the three subspecies of deer in the U.S., the black-tailed deer appears to be most susceptible to *A. marginale*. Anaplasmosis antibody prevalence also was greater in summer; this corresponds to the active period of infection by ticks (Howe, 1981). Prevalence was much lower in young deer than in adults; this is similar to what has been reported for black-tailed deer (Behymer et al., 1989) and for white-tailed deer (Smith et al., 1982).

Bluetongue and EHD are closely-related diseases of North American domestic and wild ruminants. In California, workers have reported antibody prevalences from 12% (Trainer and Jochim, 1969) to 60% in some deer herds (Jessup, 1985). The percentage of reactors to BT and EHD in this study was much higher in adults (18% (BTV) and 17% (EHDV)) than in young deer (6% (BTV) and 3% (EHDV)); this supports previous studies in deer in California (Trainer and Jochim, 1969) and other states (Johnson et al., 1986; Stallknecht et al., 1991). A high prevalence of BT and EHD antibodies was found in the southern mule deer populations, which could result

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

TABLE 1. Comparison of antibody prevalence in five California black-tailed deer herds for anaplasmosis, Q fever and toxoplasmosis, 1979 to 1991.

Herd	1979 to 1986[a]				1987 to 1991[b]			
	Number tested	Number positive			Number tested	Number positive		
		Anaplasma marginale	*Coxiella burnetii*	*Toxoplasma gondii*		*Anaplasma marginale*	*Coxiella burnetii*	*Toxoplasma gondii*
Adelaida	28	9	3	1	11	6	0	0
Carson River	21	0	0	0	14	0	0	2
East Tehama	12	6	1	0	16	11	2	0
Mendocino	42	23	1	4	18	18	3	1
Mt. Hamilton	109	70	2	13	15	9	10	0

[a] From Behymer et al. (1989).
[b] From the present study.

from a recent outbreak of BT or EHD in these herds, as bluetongue is endemic in southern and central California (Stott et al., 1981). The viruses of BT and EHD are antigenically similar, and cross reactions between the various serological tests are expected, as supported by our results. Therefore, the development of more specific and inexpensive tests is necessary to clearly differentiate the viruses in wildlife studies. However, antibodies to both EHDV and BTV in deer also may result from dual infections (Stallknecht et al., 1991).

Lyme borreliosis is a tickborne illness caused by the spirochete *Borrelia burgdorferi*. The vector tick, *Ixodes pacificus* is found in 53 of the 58 counties in California (Anonymous, 1991) and black-tailed deer are its major host (Lane and Burgdorfer, 1986). In the coastal ranges of California, antibodies to *B. burgdorferi* were detected in 39% of the black-tailed deer tested, and antibody prevalence varied significantly according to sex and season of collection (Lane and Burgdorfer, 1986). Our study provided support to the wide distribution of *B. burgdorferi* antibodies in the California black-tailed deer population and its seasonal trends. Based on serology, there seems to be a high rate of exposure in deer, but all attempts to isolate spirochetes from these hosts have been unsuccessful (Brown and Lane, 1992). In the coastal ranges, black-tailed deer also serve as hosts of a spirochete resembling the recently described *Borrelia coriaceae*, which cross reacts with *B. burgdorferi* and is transmitted by the soft tick *Ornithodoros coriaceus* (Lane and Burgdorfer, 1988). Similarly, strong cross-reactivity with *Borrelia hermsii*, the causative agent of tickborne relapsing fever can occur (Magnarelli et al., 1986). Therefore, until a more specific serologic test for spirochetal antibody surveys of wildlife is available, the results of studies such as ours should be interpreted cautiously.

Coxiella burnetii, the infectious agent of Q fever, can occur in a variety of wildlife animals (Bell, 1981). There is serological evidence for infection in deer in Europe (Weber et al., 1978, Blancou, 1983; Baradel et al., 1988), especially roe deer with prevalence rates of 1.7 to 3.7%. In California, infection also has been reported in the black-tailed deer population (Enright et al., 1971; Behymer et al., 1989) with a range of infection from 6% to 51%. When comparing the five herds tested by Behymer et al. (1989) in 1979 to 1986 (Table 1), the Carson River herd remained free of *C. burnetii* antibody and prevalence decreased in the Adelaida herd from 11 to 0%. In the three other herds, prevalence was higher in our study. Prevalence also was highest in black-tailed deer in the coastal ranges where sheep infection is known to be high (Enright et al., 1971).

Nearly all animal species are susceptible

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

to *Toxoplasma gondii* (Dreesen, 1990). In the U.S., there is serological evidence that deer may serve as reservoirs for the tissue cyst stage of *T. gondii*. In California, past antibody prevalences of 14 to 29% have been reported in black-tailed deer (Franti et al., 1975; Behymer et al., 1989); this is much higher than the 7% found in our study. In the five herds tested for *T. gondii* infection by Behymer et al. (1989), prevalence decreased in three herds (Adelaida, Mendocino, Mount Hamilton) and increased in one herd (Carson River) (Table 1). The East Tehama herd was free of antibodies in both studies. However, our small sample sizes could explain such differences in prevalence. A higher prevalence was observed in our study during the winter season (11%). Such results need to be confirmed in future studies in order to assess this seasonal trend, as no specific risk factor could be associated.

Serologic analyses can provide important information about disease distribution and seasonal trends. In wildlife surveillance programs, the use of herd antibody profiles offers an economic and easy approach to monitor infections and their epidemiologic patterns. Serologic surveillance programs are even more easily applied to deer raised on game farms. This approach can serve as a monitor for disease transmission between free-ranging deer, confined and privately owned deer and domestic livestock.

ACKNOWLEDGMENTS

The authors acknowledge the support of Karen Jones and many biologists of the California Department of Fish and Game (CDFG) and funding from the CDFG Wildlife Management Division.

LITERATURE CITED

ANONYMOUS. 1991. The surveillance of Lyme disease in California. California Morbidity November 29, 1991, Numbers 47/48, 1 p.

BARADEL, J. M., J. BARRAT, J. BLANCOU, J. M. BOUTIN, C. CHASTEL, G. DANNACHER, D. DELORME, Y. GERARD, J. M. GOURREAU, U. KIHM, B. LARENAUDIE, C. LE GOFF, P. P. PASTORET, P. PERREAU, A. SCHWERS, E. THIRY, D. TRAP, G. UILENBERG, AND P. VANNIER. 1988. Results of a serological survey of wild mammals in France. Revue scientifique et technique de l'Office international des Epizooties No. 7. Office International de Epizooties, Paris, France, pp. 873–883.

BEHYMER, D. E., R. RUPPANNER, D. BROOKS, J. C. WILLIAMS, AND C. E. FRANTI. 1985. Enzyme immunoassay for surveillance of Q fever. American Journal of Veterinary Research 46: 2413–2417.

———, D. JESSUP, K. JONES, C. E. FRANTI, H. RIEMANN, AND A. BAHR. 1989. Antibodies to nine infectious disease agents in deer from California. Journal of Zoo and Wildlife Medicine 20: 297–306.

BELL, J. F. 1981. Q (Query) Fever. *In* Infectious diseases of wild mammals, 2nd ed., J. W. Davis, L. H. Karstad, and D. O. Trainer (eds.). Iowa State University Press, Ames, Iowa, pp. 388–397.

BLANCOU, J. 1983. Serologic testing of wild roe deer (*Capreolus capreolus L.*) from the Trois Fontaines forest region of eastern France. Journal of Wildlife Diseases 19: 271–273.

BRÖMEL, J., AND K. ZETTL. 1973. Untersuchungen von Wild im Regierungsbezirk Kassel. Deutsche Tierärztliche Wochenschrift 80: 41–45.

BROWN, R. N., AND R. S. LANE. 1992. Lyme disease in California: A novel enzootic transmission cycle of *Borrelia burgdorferi*. Science 256: 1439–1442.

BULGIN, M. S. 1990. Ovine progressive pneumonia, caprine arthritis-encephalitis, and related lentiviral diseases of sheep and goats. Veterinary Clinics of North America: Food Animal Practice 6: 691–704.

CAMPBELL, G. L., B. F. ELDRIDGE, J. L. HARDY, W. C. REEVES, D. A. JESSUP, AND S. B. PRESSER. 1989. Prevalence of neutralizing antibodies against California and Bunyamwera serogroup viruses in deer from mountainous areas of California. American Journal of Tropical Medicine and Hygiene 40: 428–437.

CHRISTENSEN, J. F., AND D. W. MCNEAL. 1967. *Anaplasma marginale* infection in deer in the Sierra Nevada foothill area of California. American Journal of Veterinary Research 28: 599–601.

———, J. W. OSEBOLD, J. B. HARROLD, AND M. N. ROSEN. 1960. Persistence of latent *Anaplasma marginale* infection in deer. Journal of the American Veterinary Medical Association 136: 426–427.

———, ———, AND J. R. DOUGLAS. 1962. Bovine anaplasmosis in the coast range area of California. Journal of the American Veterinary Medical Association 141: 952–957.

CLARK, R. K., D. A. JESSUP, M. D. KOCK, AND R. A. WEAVER. 1985. Survey of desert bighorn sheep in California for exposure to selected infectious diseases. Journal of the American Veterinary Medical Association 187: 1175–1179.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

CRAWFORD, T. B., AND D. S. ADAMS. 1981. Caprine arthritis-encephalitis: Clinical features and presence of antibody in selected goat populations. Journal of the American Veterinary Medical Association 178: 713–719.

CUTLIP, R. C., H. D. LEHMKUHL, K. A. BROGDEN, AND M. J. F. SCHMERR. 1991. Seroprevalence of ovine progressive pneumonia in various domestic and wildlife animal species, and species susceptibility to the virus. American Journal of Veterinary Research 52: 189–191.

———, ———, J. M. SACKS, AND A. L. WEAVER. 1992. Seroprevalence of ovine progressive pneumonia virus in sheep in the United States as assessed by analyses of voluntarily submitted samples. American Journal of Veterinary Research 53: 976–979.

DEAN, A. G., J. A. DEAN, A. H. BURTON, AND R. C. DICKER. 1990. Epi-Info version 5.01: A word processing, database, and statistics program for epidemiology on microcomputers. USD, Inc., Stone Mountain, Georgia, 384 pp.

DREESEN, D. W. 1990. *Toxoplasma gondii* infections in wildlife. Journal of the American Veterinary Medical Association 196: 274–276.

EAST, N. E., J. D. ROWE, B. R. MADEWELL, AND K. FLOYD. 1987. Serologic prevalence of caprine arthritis-encephalitis virus in California goat dairies. Journal of the Ameican Veterinary Medical Association 190: 182–185.

ENRIGHT, J. B., C. E. FRANTI, D. E. BEHYMER, W. M. LONGHURST, V. J. DUTSON, AND M. E. WRIGHT. 1971. *Coxiella burnetii* in a wildlife-livestock environment. Distribution of Q fever in wild mammals. American Journal of Epidemiology 94: 79–90.

FRANTI, C. E., G. E. CONNOLLY, H. P. RIEMANN, D. E. BEHYMER, R. RUPPANNER, C. M. WILLADSEN, AND W. LONGHURST. 1975. A survey for *Toxoplasma gondii* antibodies in deer and other wildlife on a sheep range. Journal of the American Veterinary Medical Association 167: 565–568.

HOWE, D. L. 1981. Anaplasmosis. *In* Infectious diseases of wild mammals, 2nd ed., J. W. Davis, L. H. Karstad, and D. O. Trainer (eds.). Iowa State University Press, Ames, Iowa, pp. 407–414.

JESSUP, D. A. 1985. Epidemiology of two orbiviruses in California's native wild ruminants: Preliminary report. *In* Bluetongue and related orbiviruses, T. L. Barber and M. M. Jochim (eds.). Alan R. Liss, Inc., New York, New York, pp. 53–65.

JOHNSON, J. L., T. L. BARBER, M. L. FREY, AND G. NASON. 1986. Serologic survey of selected pathogens in white-tailed and mule deer in western Nebraska. Journal of Wildlife Diseases 22: 515–519.

KUTTLER, K. L. 1984. Anaplasma infections in wild and domestic ruminants: A review. Journal of Wildlife Diseases 20: 12–20.

LANE, R. S., AND W. BURGDORFER. 1986. Potential role of native and exotic deer and their associated ticks (Acari: *Ixodidae*) in the ecology of lyme disease in California, USA. Zentralblatt für Bakteriologie und Hygiene A 263: 55–64.

———, AND ———. 1988. Spirochetes in mammals and ticks (Acari: *Ixodidae*) from a focus of Lyme borreliosis in California. Journal of Wildlife Diseases 24: 1–9.

MAGNARELLI, L. A., J. F. ANDERSON, C. S. APPERSON, D. FISH, R. C. JOHNSON, AND A. CHAPPELL. 1986. Spirochetes in ticks and antibodies to *Borrelia burgdorferi* in white-tailed deer from Connecticut, New York State, and North Carolina. Journal of Wildlife Diseases 22: 178–188.

———, J. H. OLIVER, JR., H. J. HUTCHESON, AND J. F. ANDERSON. 1991. Antibodies to *Borrelia burgdorferi* in deer and raccoons. Journal of Wildlife Diseases 27: 562–568.

RICHARDSON, M. D., A. TURNER, D. W. WARNOCK, AND P. A. LLEWELLYN. 1983. Computer assisted rapid enzyme linked immunosorbent assay (ELISA) in the serological diagnosis of Aspergillosis. Journal of Immunological Methods 56: 201–207.

ROBINETTE, W. L., D. A. JONES, G. ROGERS, AND J. S. GASHWILER. 1957. Notes on tooth development and wear for Rocky Mountain mule deer. The Journal of Wildlife Management 21: 134–153.

SMITH, R. D., A. WOOLF, L. L. HUNGERFORD, AND J. P. SUNDBERG. 1982. Serologic evidence of *Anaplasma marginale* infection in Illinois white-tailed deer. Journal of the American Veterinary Medical Association 181: 1254–1256.

SNYDER, M. L., W. C. STEWART, AND E. A. CARBREY. 1977. A procedural guide for enzyme immunoassay in pseudorabies diagnosis. The National Veterinary Laboratories, Animal and Plant Health Inspection Team, U.S. Department of Agriculture, Ames, Iowa, 38 pp.

STALLKNECHT, D. E., J. L. BLUE, E. A. ROLLOR, III, V. F. NETTLES, W. R. DAVIDSON, AND J. E. PEARSON. 1991. Precipitating antibodies to epizootic hemorrhagic disease and bluetongue viruses in white-tailed deer in the southeastern United States. Journal of Wildlife Diseases 27: 238–247.

STOTT, J. L., K. C. ELSE, B. MCGOWAN, L. K. WILSON, AND B. I. OSBURN. 1981. Epizootiology of bluetongue virus in western United States. Proceedings of the United States Animal Health Association 85: 170–180.

THURMOND, M. C., C. H. HOLMBERG, AND J. P. PICANSO. 1985. Antibodies to bovine leukemia virus and presence of malignant lymphoma in slaughtered California dairy cattle. Journal of the National Cancer Institute 74: 711–714.

TRAINER, D. O. 1970. The use of wildlife to monitor zoonoses. Journal of Wildlife Diseases 6: 397–401.

———, AND M. M. JOCHIM. 1969. Serologic evi-

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

dence of bluetongue in wild ruminants in North America. American Journal of Veterinary Research 30: 2007–2011.

UTTERBACK, W. W., L. M. STEWART, T. L. BEALS, AND C. E. FRANTI. 1972. Anaplasmosis survey in northern California, 1969–1970; Prevalence of complement-fixing antibodies in cattle by herd location. American Journal of Veterinary Research 33: 257–267.

WEBER, A., J. PAULSEN, AND H. KRAUSS. 1978. Seroepidemiologische Untersuchungen zum Vorkommen von Infectionskrankheiten bei einheimischen Schalenwild. Der Praktische Tierarzt 59: 353–357.

Received for publication 27 January 1993.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 29 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use