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SEROLOGIC SURVEY FOR EVIDENCE OF EXPOSURE TO VESICULAR STOMATITIS VIRUS, PSEUDORABIES VIRUS, BRUCELLOSIS AND LEPTOSPIROSIS IN COLLARED PECCARIES FROM ARIZONA

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ABSTRACT: Two hundred eighteen usable serum samples were collected from hunter-killed collared peccaries (*Tayassu tajacu*) during March 1986, in three areas of Arizona. Evaluations for antibodies against vesicular stomatitis virus (VSV) New Jersey (NJ) type, VSV Indiana type, pseudorabies virus, brucellosis, and leptospirosis revealed positive test results in 8%, 0%, <1%, 0%, and 23% of the sera, respectively. Exposure of peccaries to VSV (NJ) was widespread, but variation in the prevalence of seropositive peccaries was not found between the three areas sampled. The exposure of peccaries to VSV (NJ) probably was related to the recent epizootics in livestock in the vicinity. Exposure to *Leptospira interrogans* serovars also was widespread, and geographic variation in the prevalence of peccaries with antibodies against *L. interrogans* was found.

Key words: *Tayassu tajacu*, collared peccary, vesicular stomatitis, pseudorabies, brucellosis, leptospirosis, serology, survey.

INTRODUCTION

Peccaries and pigs belong to the same taxonomic order (Artiodactyla) but different families, Tayassuidae and Suidae, respectively (Sowls, 1984). The collared peccary (*Tayassu tajacu*) is a small artiodactyl which is present in southern, western, and parts of central Texas, southwestern New Mexico, and southeastern to central Arizona. The range extends southward from these states to northern Argentina (Sowls, 1978). Peccaries are generalist herbivores that feed on cacti, grasses, browse, herbs, and insects in amounts which vary by season and availability (Sowls, 1984).

The status of free-ranging collared peccaries in regard to infectious diseases is not known. Under laboratory conditions, collared peccaries were found to be susceptible by inoculation and/or direct contact to vesicular stomatitis (VS), foot-and-mouth disease, vesicular exanthema of swine, rinderpest (Dardiri et al., 1969), hog cholera (Loan and Storm, 1968; Dardiri et al., 1969), and pseudorabies (Crandell et al., 1986), but not African swine fever (Dardiri et al., 1969). A few limited surveys for evidence of infection in free-ranging peccaries have been published. Sera from 12 of 30 (40%) peccaries from southern Texas sampled prior to the 1971 Venezuelan equine encephalitis virus (VEEV) outbreak were found to neutralize VEEV and/or western equine encephalitis virus (Smart et al., 1975). Also in southern Texas, three peccaries were negative serologically for *Coxiella burnetti* and *Brucella canis* (Randhawa et al., 1977). In New Mexico, 20 peccaries were negative serologically for antibodies against the following disease agents: *C. burnetti*; *Leptospira interrogans* serovars *autumnalis, canicola, grippotyphosa, hardjo, icterohaemorrhagiae,* and *pomona*; *B. abortus*; influenza myxoviruses A/Swine/1967/31 and A_2/Japan/170/62; and the SF_4 strain of bovine myxovirus parainfluenza 3 (Woods et al., 1968). In Arizona, two cases in which free-ranging peccaries apparently had died of salmonellosis were reported (Sowls, 1984). A few studies have reported on the parasites of free-ranging peccaries (Samson and Donaldson, 1968; Samuel and Low, 1970;

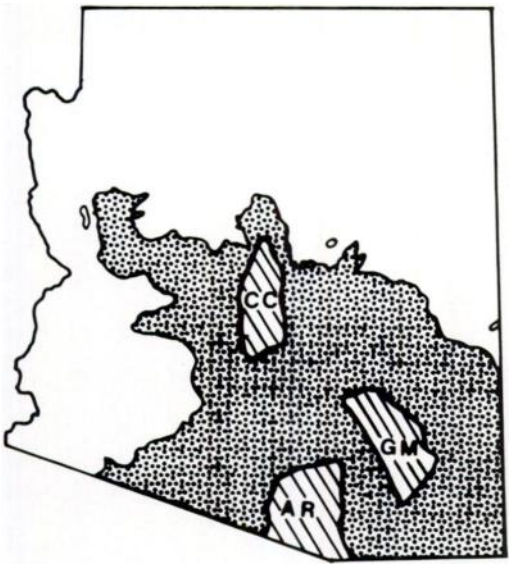

FIGURE 1. Areas in Arizona from which collared peccary sera were collected. The dotted region indicates the range of collared peccaries in Arizona in 1985. Lined regions are the areas where samples were collected. Codes for the areas sampled are as follows: CC, Cave Creek; AR, Arivaca; GM, Galiuro Mountains. (Range map courtesy of the Arizona Game and Fish Department.)

Corn et al., 1985), and there also are reports of causes of debilitation and death in captive peccaries (Hellgren et al., 1984a, b; Sowls, 1984; Hannon et al., 1985; Lochmiller et al., 1985). One case of rabies has been reported in a collared peccary (Centers for Disease Control, 1986).

The relative lack of information available on diseases of the collared peccary and their disease susceptibility, the extent of the range of this species, and the fact that this animal commonly inhabits livestock range provide grounds for disease studies. The objective of this survey was to determine the prevalence and distribution of collared peccaries in Arizona with antibodies against selected major livestock diseases.

MATERIALS AND METHODS

Blood samples were obtained from hunter-killed collared peccaries during Arizona's 1-wk-long firearms hunt. Animals sampled were from three Arizona Game and Fish Department Management Units which are referred to subsequently as Arivaca, Cave Creek, and the Galiuro Mountains (Fig. 1). These three areas were chosen because they represented three widely separated areas within the range of the collared peccary in Arizona. Also, relatively large numbers of hunter permits were available for these areas, and the hunter success rates were usually above average. The Arivaca area is bordered to the west by the Papago Indian Reservation, the north by Highway 86, the east by Interstate 19, and the south by the United States-Mexico border. Southeast Pima and western Santa Cruz counties are included. The Cave Creek area is bordered to the west by Interstate 17, the east by the Verde River, and extended north from Phoenix to Camp Verde. Northeast Maricopa and southeast Yavapai counties are included. The Galiuro Mountains area is bordered by the San Pedro River to the southwest, the Aravaipa Valley to the northeast, and runs from just north of Mammoth southeast to Interstate 10. Extreme southeast Pinal, extreme northeast Pima, southwest Graham, and northwest Cochise counties are included.

Hunters with permits for the three management units were mailed blood collection tubes, maps describing the locations of the check stations, and a letter asking their cooperation. Hunters were asked to collect blood while field dressing their animals by either cutting into the heart and draining blood directly into a blood collection tube or by scooping blood from the chest cavity. Hunters were instructed to keep the blood samples as still as possible and cool until they were delivered to a check station. Blood samples were collected from hunters at six check stations, two in each of the three management units during 14–16 March 1986. Most of the returned blood samples had separated and had been stored in ice chests. Blood was centrifuged the day it was received and the serum was collected, frozen on dry ice, and sent for serologic evaluation at the National Veterinary Services Laboratories (NVSL) (Veterinary Services, United States Department of Agriculture, Ames, Iowa 50010, USA).

Antibodies against vesicular stomatitis virus (VSV) and pseudorabies virus (PRV) were determined by use of serum neutralization (National Veterinary Services Laboratories, 1981). Tests were conducted for both the New Jersey (NJ) and Indiana (IN) VSV types. Titers $\geq$1:32 against VSV were considered indicative of prior exposure to VSV. Titers $\geq$1:8 against PRV were considered indicative of prior exposure to PRV.

Serum samples were evaluated for *Brucella* spp. by the brucellosis plate and brucellosis buffered acidified plate antigen tests (United States Department of Agriculture, 1965). Titers of 1:25 in the plate test and 1:25 in the buffered acidified plate antigen test were considered positive for antibodies against brucellosis. Sera were evaluated for antibodies against *L. interrogans* serovars by the microscopic agglutination test (Cole et al., 1973). A titer of ≥1:100 was considered positive for antibodies against a given serovar. Antigens of the following 13 *L. interrogans* serovars were used: *australis, autumnalis, ballum, bataviae, bratislava, canicola, grippotyphosa, hardjo, icterohaemorrhagiae, pomona, pyrogenes, tarassovi,* and *wolffi*.

Data were analyzed with the chi-square test. Yate's correction was used for the 2 × 2 contingency tables (Steel and Torrie, 1980).

RESULTS

Blood samples from 323 collared peccaries were delivered by hunters to the check stations. The voluntary hunter participation accounted for approximately 40% of the peccaries harvested from the three areas. Hemolyzed and contaminated samples were discarded, leaving 218 (68%) usable serum samples. Varied amounts of serum were obtained and some samples were not of sufficient volume to test for all disease agents.

Positive VSV (NJ) antibody titers were detected in 18 of 218 (8%) sera evaluated. Antibody titers of 1:32, 1:64 and 1:256 against VSV (NJ) were found in 14 (6%), 3 (1%), and 1 (<1%) animals, respectively. Geographically, antibody titers were found in nine of 106 (9%), three of 64 (5%) and six of 48 (13%) animals from the Arivaca, Cave Creek, and Galiuro Mountains areas, respectively, but these differences were not significant ($P > 0.05$). Positive VSV (IN) antibody titers were not found.

Sera from 192 peccaries were evaluated for antibodies against PRV. Due to frequent toxicity to the cell culture system at the 1:4 dilution in the serum neutralization test, sera were diluted to 1:8 before evaluation. One peccary from the Arivaca area had a positive PRV antibody titer of 1:16.

Sera from 194 peccaries were evaluated for *Brucella* spp. Results were negative for all peccaries on both the plate and buffered acidified plate antigen tests.

Positive antibody titers against one or more *L. interrogans* serovars were found in sera from 48 (23%) of the 213 collared peccaries evaluated (Table 1). Of the seropositives, 22 (46%) had titers against only one serovar, 11 (23%) had titers against two serovars, eight (17%) had titers against three serovars, three (6%) had titers against four serovars, two (4%) had titers against five serovars, and one each (2%) had titers against six and seven serovars. Titers were as high as >1:12,800 (Table 2).

Geographically, antibody titers against *L. interrogans* serovars were found in 15 of 100 (15%), 25 of 69 (36%) and eight of 44 (18%) peccaries from the Arivaca, Cave Creek, and Galiuro Mountains areas, respectively. The prevalences of animals from Arivaca and the Galiuro Mountains with positive antibody titers were not different ($P > 0.05$). The prevalence of animals from Cave Creek with positive antibody titers was greater than those with positive antibody titers from Arivaca ($P < 0.005$) and the Galiuro Mountains ($P < 0.10$).

DISCUSSION

Although many sources and vectors of VSV (NJ) have been suggested, the basic transmission cycle and virus reservoir remain unknown (Hanson, 1984). The role of wildlife in the maintenance and dissemination of VSV (NJ) is unclear; however, serologic evidence of wildlife exposure is considerable. Fletcher et al. (1985) summarized reports of naturally occurring antibodies against VSV (NJ) in wildlife. Vesicular stomatitis (NJ) is enzootic in the coastal plains of the southeastern United States and also occurs in widespread epizootics (Karstad, 1981). Recent epizootics occurred in the western United States during 1982–1983 (Jenney et al., 1984) and 1985 (Hall, 1985). Wildlife surveillance during the 1982–1983 epizootic demonstrated the occurrence of antibody titers

TABLE 1. Prevalence of collared peccaries from Arizona with positive titers (≥1:100) against *Leptospira interrogans* serovars.

Serovar	Number of positive sera and prevalence			
	Arivaca (n = 100)	Cave Creek (n = 69)	Galiuro Mountains (n = 44)	Total (n = 213)
australis	3 (3%)	0	2 (5%)	5 (2%)
autumnalis	4 (4%)	4 (6%)	3 (7%)	11 (5%)
ballum	1 (1%)	0	0	1 (<1%)
bataviae	0	0	0	0
bratislava	7 (7%)	12 (17%)	6 (14%)	25 (12%)
canicola	2 (2%)	0	0	2 (1%)
grippotyphosa	3 (3%)	0	1 (2%)	4 (2%)
hardjo	8 (8%)	3 (4%)	2 (5%)	13 (6%)
icterohaemorrhagiae	1 (1%)	2 (3%)	1 (2%)	4 (2%)
pomona	4 (4%)	24 (35%)	8 (18%)	36 (17%)
pyrogenes	1 (1%)	0	0	1 (<1%)
tarassovi	0	0	0	0
wolffi	1 (1%)	0	0	1 (<1%)

in rodents, elk (*Cervus elaphus*), mule deer (*Odocoileus hemionus*) (Webb, 1984), and pronghorn antelope (*Antilocapra americana*) in Colorado (Thorne, 1983; Webb, 1984). In view of this information, it was not surprising that evidence of exposure of collared peccaries to VSV (NJ) was found through serologic evaluations.

The initial reports of clinical VS (NJ) in livestock from the two most recent VS (NJ) epizootics came from Arizona during May 1982 (Orrell, 1983) and from Arizona and New Mexico during June 1985 (Hall, 1985). Since VSV (NJ) is not known to be enzootic in Arizona, the occurrence of positive antibody titers in peccary sera in the present study probably resulted from exposure to the virus during one of these recent epizootics. Although only 8% of the peccaries were seropositive, the positive samples came from peccaries from throughout the three areas sampled. This suggests that VSV (NJ) had been active previously throughout southeast and central Arizona.

Peccaries have developed clinical VS (IN) after both inoculation and direct contact; however, the course of the disease was not severe (Dardiri et al., 1969). Information is not available on the susceptibility of peccaries to VSV (NJ). Clinical VS (NJ)

TABLE 2. The number of collared peccaries with positive antibody titers against each *Leptospira interrogans* serovar present.

Serovar	Titer								
	1:100	1:200	1:400	1:800	1:1,600	1:3,200	1:6,400	1:12,800	>1:12,800
australis	3	0	2	0	0	0	0	0	0
autumnalis	3	3	1	2	1	0	0	1	0
ballum	0	0	0	0	1	0	0	0	0
bratislava	9	7	4	0	3	2	0	0	0
canicola	0	1	0	0	0	0	0	1	0
grippotyphosa	0	1	0	1	0	0	1	0	1
hardjo	6	3	1	2	1	0	0	0	0
icterohaemorrhagiae	1	1	0	2	0	0	0	0	0
pomona	5	7	7	3	2	1	1	3	7
pyrogenes	0	0	0	1	0	0	0	0	0
wolffi	0	1	0	0	0	0	0	0	0

and VS (IN) are similar in livestock (Seymour and Yuill, 1981). Although the effect that a VS (NJ) epizootic would have on a peccary population is not known, extrapolation from the above information suggests that while peccaries could be involved in an epizootic, the effect of the disease on the population probably would not be significant.

Crandell et al. (1986) found evidence of peccary susceptibility to PRV infection in an experimental trial. Two captive peccaries inoculated with PRV did not develop clinical disease, but virus was isolated from one animal from a nasal swab taken 7 days postinoculation and a tonsil specimen taken 15 days postinoculation. Both animals had serum neutralization antibody titers of 1:4 at 15 days postinoculation. Because other information is not available on PRV in peccaries, it is premature to attach any significance to the single PRV seropositive peccary found in the present survey. Pseudorabies is generally a disease of domestic swine and swine serve as the principal reservoir (Gustafson, 1975). Feral swine also may be important sources of PRV (Nettles and Erickson, 1984; Corn et al., 1986), but neither domestic nor feral swine were known to exist in the area where the seropositive peccary was killed. Additional research and surveillance are necessary to determine the importance, if any, of peccaries to PRV epizootiology.

Most of the range of peccaries in the southwestern United States is shared with cattle and other livestock, and the fact that peccaries in the present study were not found to have *Brucella* spp. antibodies indicates that brucellosis was not enzootic in peccaries in Arizona. This finding does not prove that peccaries in other areas could not be involved, since there may have been little opportunity for infection. Arizona has been declared free of swine brucellosis, and less than 1% of the cattle herds are infected (Frye, 1985).

Serologic evaluations indicated that *L. interrogans* serovars *pomona* and *bratislava* were the most prevalent in these peccaries from Arizona. Striped skunks (*Mephitis mephitis*) are considered to be the principal reservoir of serovar *pomona* (Shotts, 1981). Although attempts to culture leptospires were not made, the prevalences of peccaries seropositive for the various serovars did not indicate that they were of singular importance to the epizootiology of these serovars in Arizona. However, peccaries may aid in maintenance and transmission. Contaminated water and food, especially infected prey species in the case of carnivores, are the most important modes of transmission of leptospires (Redetzke and McCann, 1980; Shotts, 1981). Since peccaries are generalist herbivores, they probably were exposed to the leptospires through contact with contaminated water. Geographic variation in the prevalence of seropositive peccaries probably was due to variation in the environment relative to conditions necessary for the survival and transmission of leptospires. No information is available on what effects *L. interrogans* infection would have on peccaries.

Surveys such as the present study are limited in that they do not indicate the actual presence of infected animals. However, such surveys do demonstrate the prevalence of exposure of wildlife species to selected pathogens. Vesicular stomatitis virus (NJ) activity had been widespread in Arizona as evidenced by seropositive peccaries from all areas sampled. A possibility of peccary involvement in PRV epizootiology was suggested by the finding of one PRV seropositive peccary, but further study is needed. The fact that no peccaries were seropositive for *Brucella* spp. demonstrated that brucellosis was not enzootic in peccaries in Arizona. The widespread prevalence of peccaries seropositive for *L. interrogans* serovars demonstrated broad *L. interrogans* contamination. While evidence for exposure of peccaries to some of the above pathogens was found, these data

did not suggest that peccaries were reservoirs of these diseases or that they were of singular importance to the maintenance of the diseases.

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