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A SURVEY OF WILD SWINE IN THE UNITED STATES FOR EVIDENCE OF HOG CHOLERA

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ABSTRACT: The results of surveillance for hog cholera (HC) in wild swine (*Sus scrofa*) collected from throughout the United States from 1979 to 1987 are presented. Sera collected from 1,218 wild swine and tissues from 637 were evaluated for HC antibodies and virus, respectively. Included within this surveillance were samples from Santa Cruz and Santa Rosa Islands, California, where HC virus had been deliberately introduced into wild swine during the 1950's in attempts to eradicate these animals. All evaluations were considered negative for HC. It appears that the HC virus does not maintain itself in dispersed swine populations and that wild swine have not remained a reservoir of HC since its eradication in domestic swine in the United States.

Key words: *Sus scrofa*, wild swine, hog cholera, surveillance, serologic survey.

INTRODUCTION

The United States was declared free of hog cholera (HC) on 31 January 1978; however, HC remains widespread in domestic swine throughout most of the rest of the world (Trevino, 1985). Wild swine (*Sus scrofa*) are fully susceptible (Brugh et al., 1964); HC has been reported in wild swine in the United States (Shaw, 1941; Hanson and Karstad, 1959) and occurs at present in wild swine in Europe (International Disease Surveillance, 1988). Epizootics in European wild boar (*Sus scrofa*) typically have begun through exposure of wild swine to discarded HC-infected domestic swine carcasses. Infection of domestic swine through the feeding of infected wild swine carcasses to domestic swine also has been reported (Hutter, 1953; Spaa, 1955; Spiecker, 1969).

Throughout the eradication program in the United States, wild swine were suspected as a possible reservoir of the virus. The initial objective of this survey was to determine if wild swine in the United States had evidence of HC infection following its eradication from domestic swine. During the survey, we learned that wild swine on Santa Cruz and Santa Rosa Islands, California, had been intentionally infected with HC during the 1950's. A second objective, therefore, was to determine the fate of the HC virus following its introduction into these two insular wild swine populations.

MATERIALS AND METHODS

The survey for evidence of HC in wild swine was conducted throughout the geographic range of wild swine in the United States from January 1979 to April 1987 (Table 1). Animals were live-trapped or collected by shooting. Immediately after euthanasia or death by gunshot, a blood sample was drawn via heart puncture. Serum was collected, centrifuged and frozen until evaluated by fluorescent antibody serum neutralization (National Veterinary Services Laboratories, 1981) at the National Veterinary Services Laboratories (NVSL; Veterinary Services, Animal and Plant Health Inspection Service, U.S. Department of Agriculture, Ames, Iowa 50010, USA). Titers $<1{:}16$ were considered negative. Samples positive at a titer of $\geq 1{:}16$ also were tested by serum neutralization for antibodies against bovine viral diarrhea (BVD) virus. A low HC antibody titer coupled with an equivalent or higher BVD antibody titer was interpreted as a cross reaction resulting from BVD virus exposure, not as evidence of HC virus exposure (Stewart et al., 1971; Carbrey et al., 1976).

Abbreviated necropsies were conducted on all animals. Spleen and tonsil samples were collected, frozen and submitted to NVSL for HC virus isolation attempts by cell culture inoculation and/or direct fluorescent antibody tests. Suspensions of spleen and/or tonsil were inoc-

TABLE 1. Surveillance for hog cholera in wild swine in the United States, January 1979 through April 1987. Sera were tested by serum neutralization. Tissues were evaluated by cell culture virus isolation and/or the direct fluorescent antibody test.

State mo./yr	County or parish	Area	Number of sera	Number of tissues
Alabama				
4/80	Clarke	Fred T. Stimpson/Hal's Lake	8	8
Arizona				
1/83	Mojave	Havasu National Wildife Refuge	13	13
Arkansas				
10/80	Union	Felsenthal National Wildife Refuge	10	10
California				
2/83	San Luis Obispo	Hardens Wildlife Management, Inc.	20	0
2/83	Los Angeles	San Clemente Island, U.S. Navy	18	0
2/83	Santa Clara	Mt. Hamilton Range	18	0
2/83	Tehama	Dye Creek Preserve	39	11
2/83	Merced	Cottonwood Creek Wildlife Management Area	6	0
5/83	Monterey	Deer Valley Ranch	38	38
5/83	Santa Clara	Mt. Hamilton Range	10	10
5/83	Tehama	Dye Creek Preserve	23	10
12/86	Santa Clara	Guadalupe Dump	10	0
2/87	Santa Barbara	Santa Cruz Island	31[a]	31
2/87	Santa Barbara	Santa Rosa Island	60	61
Florida				
12/79	Sarasota/Manatee	Myakka River State Park	24	10
2/80	Orange/Brevard	Tosohatchee/Merritt	10	10
2/80	Lake	E. K. Ranch	1	0
3/80	Osceola	Deseret Ranch	10	10
7/80	Osceola	Prairie Lakes State Park	10[a]	10
1/81	Orange	Tosohatchee State Preserve	6	5
3/81	Wakulla	St. Marks National Wildlife Refuge	12	12
5/81	Brevard	Merritt Island National Wildife Refuge	10	10
5/81	Franklin	St. Vincent National Wildife Refuge	10	10
6/81	Hendry	Alico Ranch	10[b]	10
Georgia				
1/79 and 7/80	Chatham	Ossabaw Island	104	10
6/79 and 8/80	Camden	Cumberland Island	15	10
7/79	Wilkinson	Napier Plantation	5	5
7/80	Telfair	Horse Creek Wildlife Management Area	10	10
11/80 and 4/81	Liberty	Fort Stewart	10	10
11/80 to 12/80	Liberty	Thompson's Pasture	12	10
5/81	McIntosh	Rhetts Island	12	10
9/81	Chatham	Ossabaw Island	10	10
12/84	Liberty	St. Catherines Island	6	6
3/85	Liberty	St. Catherines Island	8	5
7/85	Houston	Oaky Woods Wildlife Management Area	9	0
Hawaii				
6/80 to 9/80	Maui	Puu-O-Hoku Ranch, Molokai	59	8
6/83 to 7/83	Maui	Molokai	68	0
8/83 to 11/83	Hawaii	Hawaii	133	0
6/85 to 7/85	Maui	Molokai	30	10
7/85 to 8/85	Honolulu	Oahu	11	10
8/85	Maui	Maui	9	10

TABLE 1. Continued.

State mo/yr	County or parish	Area	Number of sera	Number of tissues
9/85	Kauai	Kauai	9	10
3/87 to 4/87	Maui	Molokai	28	0
Louisiana				
11/80	Grant	Georgetown	9	10
Mississippi				
9/80	Pearl River	Poplarville	10[b]	10
North Carolina				
1/80 to 3/80	Swain	Great Smoky Mountains National Park	8	11
South Carolina				
1/79 and 5/80	Georgetown	Hobcaw Barony	45	10
4/80	Beaufort	Palmetto Bluff	9	9
5/80	Berkeley	Francis Marion National Forest	10	10
11/84	Aiken	Savannah River Plant	10	10
3/85	Aiken	Savannah River Plant	8	8
Tennessee				
3/79 to 3/80	Blount	Great Smoky Mountains National Park	11	14
Texas				
1/85	Cameron	Laguna Atascosa National Wildife Refuge	11	10
2/85	Webb	Callaghan Ranch	20	10
2/85	Kleberg	King Ranch (Santa Gertrudis)	10[a]	10
2/85	Kleberg	King Ranch (Laureles)	13	10
2/85	Aransas	Aransas National Wildife Refuge	13	10
4/85	Burnet	Lake Buchanan	11	10
4/85	Llano	Granite Hills Herford Ranch	10	10
4/85	Dimmit	Piloncillo Ranch	14	10
4/85	Anderson	Valley View Cattle Company	12	10
4/85	Trinity	Temple Eastex	10	10
Virginia				
10/80	Princess Anne	False Cape/Back Bay	5	5
West Virginia				
11/79 and 1/80	Boone/Logan	Boar Hunting Area	6	11
11/81	Boone/Logan	Bear Tree Hollow	0	4
11/81	Boone/Logan	Bear Tree Hollow	7	9
11/81	Boone/Logan	Bear Tree Hollow	1	3

[a] One animal had a titer of 1:16 for hog cholera and 1:16 for bovine viral diarrhea.
[b] One animal had a titer of 1:16 for hog cholera and 1:256 for bovine viral diarrhea.

ulated into fetal porcine kidney (FPK) stationary roller tube cell cultures. Two 7-day passages were made in FPK cell cultures, and after the second passage, the cultures were freeze-thawed and subinoculated into FPK Leighton tube cell cultures (Stewart et al., 1975). After 48- to 72-hr incubation, the FPK cultures were stained with a polyvalent viral antiserum and an anti-porcine immunoglobulin fluorescent antibody conjugate for immunofluorescence assay (Stewart et al., 1975).

Included within the survey were wild swine collected on Santa Cruz and Santa Rosa Islands, California. Santa Cruz and Santa Rosa are two of a group of eight islands off the southern California coast that collectively are known as the Channel Islands. Santa Cruz lies 40 km south of the mainland coast of Santa Barbara; Santa Rosa

lies 67 km southwest of the same location. Both islands are included within Santa Barbara County, California.

During February 1987 interviews were conducted with persons who had been involved with or had knowledge of the HC introductions on Santa Cruz and Santa Rosa. Information gathered indicated that HC virus had been introduced into the Santa Cruz wild swine population at least three times. The first introduction occurred sometime prior to 1944 when wild swine were trapped, inoculated, and released (Wheeler, 1944). Virus was reintroduced on at least two subsequent occasions, once in 1950 and once in the early to mid-1950's. Both times an unknown number of pigs kept in a pen at the main ranch were inoculated and set free. Each time large numbers of wild swine were observed dying or dead, but within a few years most evidence of the disease disappeared (C. Stanton, Natural Reserve System, Marine Science Institute, University of California, Santa Barbara, California 93106, USA, pers. comm.).

Interviews with longtime residents of Santa Rosa revealed that HC virus was released into the wild swine population of Santa Rosa twice, once in 1949 and again in either 1952 or 1953. Both times, 20 to 40 animals were roped, inoculated and set free. Wild swine were abundant at the time of the first introduction and about 80% of the island population died. The population was smaller when the second introduction occurred and a smaller percentage of wild swine died. Within a few years of each introduction, evidence of the disease in the wild swine population disappeared.

RESULTS

Sixty-seven wild swine collections were made in 15 states. Serum samples from 1,218 animals and tissues from 637 animals were negative for HC antibodies (Table 1). Included within these figures are the 31 wild swine from Santa Cruz and 61 from Santa Rosa. Three wild swine, one each from Santa Cruz Island, California, Osceola County, Florida, and Kleberg County, Texas, had equivocal HC-BVD antibody titers of 1:16. Individual wild swine from Hendry County, Florida, and Pearl River County, Mississippi, had HC antibody titers of 1:16 but higher BVD titers (Table 1). All five wild swine with equivocal HC-BVD antibody titers or higher BVD titers were negative for HC virus by cell culture inoculation and/or the direct fluorescent antibody test of tonsil and/or spleen.

DISCUSSION

Results of serologic evaluations and virus isolation attempts on samples from wild swine from Santa Cruz and Santa Rosa Islands were considered indicative of no HC virus activity on these islands. Although it would not be possible to determine a zero infection rate without collecting samples from all of the wild swine on each island, negative HC tests from 92 animals provided a good statistical inference for this conclusion. Tabulations using a binomial distribution, as provided by the Southeastern Cooperative Wildlife and Fisheries Statistics Project (North Carolina State University, Raleigh, North Carolina 27695, USA) indicated that in large populations, as are found on both Santa Cruz and Santa Rosa, the collection of 29 animals negative for HC would indicate that the upper limit of the proportion of the population infected was 10% with a confidence level of 95%. If the sample size were doubled (59 animals) and all evaluations were negative for HC, it would indicate that the upper limit of proportion infected did not exceed 5% with a confidence level of 95% (Snedecor and Cochran, 1967). Combined results from both islands (92 animals) indicated that the maximum infection rate possible was <3.3%. Such low rates of infection would be unlikely considering the infectious nature of the HC virus. These statistics, combined with the fact that evidence of active HC infections had not been observed in wild swine on either island since the period of time when virus was introduced, predicate the conclusion that the HC virus did not remain active on either Santa Cruz or Santa Rosa Islands.

The above conclusion has national significance. The fact that the virus did not persist in these two island populations after its deliberate introduction, combined with the negative results from nationwide HC surveillance of the additional wild swine

from 15 states (Table 1) presents considerable evidence to support the position that HC virus does not remain viable in a feral population. This should negate any fears that HC virus is active but undetected in wild swine populations of the United States or that low density populations of wild swine could serve as a long-term reservoir in the event that HC virus was reintroduced into the United States.

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