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Prevalence and Transmission of Pseudorabies Virus in an Isolated Population of Feral Swine

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ABSTRACT: Six hundred sixty-one feral swine (*Sus scrofa*) from Ossabaw Island, Georgia (USA) were captured, bled, and their sera tested for pseudorabies virus (PRV) antibody during a 6 yr period. Prevalence of seroconversion in females was somewhat higher than in males (10% versus 7%), but the difference was not statistically significant. Adults had a significantly higher prevalence than juveniles (29% versus 1%). An important finding in this study was that seroconversion occurred primarily in the adult feral swine.

Key words: Feral swine, *Sus scrofa*, pseudorabies virus, seroconversion, serologic survey, prevalence.

Feral swine (*Sus scrofa*) are present in 22 states of the United States (Wood and Barrett, 1979; Mayer, 1983), and Georgia is among those states. In Georgia, feral swine are present in several areas of the mainland and on at least five islands of the coast.

Feral swine are considered a reservoir of pseudorabies virus (PRV) (Nettles and Erickson, 1984). The Southeastern Cooperative Wildlife Disease Study (SCWDS; College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602, USA) conducted a serologic survey of feral swine for antibodies against PRV using the virus neutralization test; the prevalence of reactors from 16 enzootic sites in 11 southeastern states was 39% (93 of 325 swine) (Nettles and Erickson, 1984). Results of a survey in Texas indicated that 36% of 124 feral swine evaluated had antibodies against PRV as determined by the enzyme-linked immunosorbent assay (Corn et al., 1986). In another study, feral swine were experimentally infected by oral exposure with PRV and placed in contact with susceptible feral swine (Tozzini et al., 1982). The exposed animals did not show clinical signs but did shed virus in saliva for 7 to 12 days postexposure, and PRV was isolated from tonsils at slaughter. The contact animals became infected and all swine seroconverted with antibodies against PRV. The purpose of the present study was to determine the prevalence and transmission spread of PRV in a closed, isolated (insular) population of feral swine not exposed to PRV-infected domestic swine. Ossabaw Island was chosen as a study area because of its isolated feral swine population and the absence of domestic swine on the island.

The terrestrial habitat of Ossabaw Island (31°46′N to 81°05′W) is approximately 4,370 ha, but the exact population of feral swine on the island is not known. Feral swine were live-trapped in pen or box traps baited with shelled corn. Swine were trapped from March through September in 1981 through 1986. When first trapped, swine were bled for serum, eartagged and weighed. Age estimates of swine were made; those <8-mo-old were classified as juveniles, and those >8-mo-old were adults. Many adults were removed from the island after trapping, but most juveniles were released. Some feral swine were retrapped and bled from two to 14 times during the study period. Frozen sera labeled with a code number and year of collection were sent to the National Animal Disease Center (NADC, Ames, Iowa 50010, USA) for serology. After all testing was completed, test results were matched with ear tag numbers to identify swine and the number of times they were bled. There were no new introductions of swine to the Ossabaw swine herd during the course of the study, and no brucellosis-

TABLE 1. Sex and age distribution and PRV antibody prevalence in feral swine trapped on Ossabaw Island, Georgia from 1981 to 1986.

Sex[a]	Age[b]	Year trapped						Total
		1981	1982	1983	1984	1985	1986	
Number trapped								
M	J	12	64	69	45	59	1	250
M	A	8	15	17	15	14	3	72
F	J	13	55	60	65	46	0	239
F	A	11	25	28	15	17	4	100
M		20	79	86	60	73	4	322
F		24	80	88	80	63	4	339
	J	25	119	129	110	105	1	489
	A	19	40	45	30	31	7	172
Number positive								
M	J	0	1	3	0	1	0	5
	A	1	0	6	0	8	1	16
F	J	0	1	0	0	0	0	1
	A	4	8	7	2	11	1	33
Prevalence %								
M	J, A	5	1	11	0	12	25	7
F	J, A	17	11	8	3	18	25	11
M, F	J	0	2	2	0	1	0	1
M, F	A	26	20	30	7	61	29	29
All swine		11	6	9	1	15	25	8

[a] M, male; F, female.
[b] J, juvenile; A, adult.

and PRV-free breeding boars were introduced into the herd during the previous 10 yr.

Six hundred sixty-one feral swine sera were initially screened for antibodies against PRV using the microimmunodiffusion test (MIDT) (Gutekunst et al., 1978). PRV latex agglutination antibody test (LAT) kits (Viral Antigens, Inc., 5171 Wilfong Road, Memphis, Tennessee 38134, USA) became available approximately 4 mo into the study and subsequently were used to test sera. All sera positive by the MIDT were also tested with the LAT. Finally, sera positive by either or both screening tests were quantitated using the microtitration virus neutralization test (MVNT) (Hill et al., 1977).

The age and sex distribution and PRV antibody prevalence in feral swine trapped on Ossabaw Island are shown in Table 1. Retrapped juveniles are only entered in the table at the time of their first capture. The ratio of males to females (322:339) did not differ significantly from unity (χ^2 = 0.44, df = 1, $P > 0.05$). The ratio of juveniles to adults was higher in males (1:3.5) than in females (1:2.4), and the difference was statistically significant (χ^2 = 4.3, df = 1, $P < 0.05$).

Only one of 215 retrapped juvenile feral swine seroconverted to PRV within 6 mo after initial trapping, whereas 17 of 76 feral swine seroconverted as adults when retrapped 12 mo or more after initial trapping as juveniles. The large increase of seroconversion from 6 to 12 mo was statistically significant (χ^2 = 74.5, df = 1, $P < 0.0001$). Adults had a statistically higher prevalence than juveniles (29% versus 1%), and the difference was statistically significant (χ^2 = 171, df = 1, $P < 0.001$). Yearly prevalences differed significantly (χ^2 = 21, df = 6, $P < 0.001$) but this difference was entirely due to the difference between the 1984 and 1985 adult rates. When these 2

yr were combined, the year differences no longer exhibited any significant deviations ($\chi^2 = 2.5$, df = 5, $P > 0.05$). The sex differences were small and not statistically significant ($\chi^2 = 1.5$, df = 1, $P > 0.05$). Virus neutralization (VN) titers of feral swine that seroconverted ranged from 1:4 to 1:512, with the greatest percentage being 1:128 and 1:256. Titers of ≥1:4 were accepted as evidence of antibody responses resulting from infection with PRV.

The results of this study indicate that PRV was being continuously spread in the feral swine herd on Ossabaw Island. This is based on the fact that there were seroconversions in adult swine against PRV each year of the study.

The fact that neither seroconversion nor maternal antibody to PRV was detected in juvenile feral swine by LAT or VN tests has important implications in determining whether a feral swine herd is infected with PRV. Results of the present study indicate that the presence of infection with PRV is best determined by testing adult feral swine sera for antibodies against PRV.

One method for eliminating PRV from swine herds is testing for antibodies against PRV and removing seropositive swine from the herd (Thawley et al., 1982). This method is based on the assumption that seropositive swine are latently infected with and potential shedders of PRV. Seropositive feral swine are therefore suspect as potential shedders of PRV, and their indiscriminate dissemination should be avoided because of possible adverse impact on the pseudorabies eradication effort presently underway in the United States.

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LITERATURE CITED

CORN, J. L., P. K. SWIDEREK, B. O. BLACKBURN, G. A. ERICKSON, A. B. THIERMANN, AND V. F. NETTLES. 1986. Survey of selected diseases in wild swine in Texas. Journal of the American Veterinary Medical Association 189: 1029–1032.

GUTEKUNST, D. E., E. C. PIRTLE, AND W. L. MENGELING. 1978. Development and evalution of a microimmunodiffusion test for detection of antibodies to pseudorabies virus in swine serum. American Journal of Veterinary Research 39: 207–210.

HILL, H. T., R. A. CRANDELL, C. L. KANITZ, J. P. MCADARAGH, G. L. SEAWRIGHT, R. F. SOLORZANO, AND W. C. STEWART. 1977. Recommended minimum standards for diagnostic tests employed in the diagnosis of pseudorabies (Aujeszky's disease). Proceedings, Annual Meeting of the American Association of Laboratory Diagnosticians 20: 375–390.

MAYER, J. J. 1983. The history, comparative morphology, and current status of wild pigs in the United States. Ph.D. Dissertation. The University of Connecticut, Storrs, Connecticut, 368 pp.

NETTLES, V. F., AND G. A. ERICKSON. 1984. Pseudorabies in wild swine. Proceedings of the United States Animal Health Association Annual Meeting 88: 505–506.

THAWLEY, D. G., D. P. GUSTAFSON, AND G. W. BERAN. 1982. Procedures for the elimination of pseudorabies virus from herds of swine. Journal of the American Veterinary Medical Association 181: 1513–1518.

TOZZINI, F., P. ALESSANDRO, AND G. D. CROCE. 1982. Experimental infection of European wild swine (*Sus scrofa* L.) with pseudorabies virus. Journal of Wildlife Diseases 18: 425–428.

WOOD, G. W., AND R. H. BARRETT. 1979. Status of wild pigs in the United States. The Wildlife Society Bulletin 7: 237–246.

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