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## ANTIBODIES TO VESICULAR STOMATITIS VIRUS IN POPULATIONS OF FERAL SWINE IN THE UNITED STATES

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**ABSTRACT:** From 1979 to 1985, 941 feral swine (*Sus scrofa*) from 53 locations in 15 states were serologically tested for antibodies to vesicular stomatitis virus (VSV). Antibodies to New Jersey serotype VSV were present in 75 swine from five locations in Arkansas, Florida, Georgia, and Louisiana. Within these populations, antibody prevalences ranged from 10 to 100%. No antibodies to Indiana serotype were detected.

### INTRODUCTION

Vesicular stomatitis (VS) was first reported in domestic swine (*Sus scrofa*) in 1943 (Schoening, 1943). Evidence of VS initially was detected in feral swine during serologic surveys of wildlife in Georgia associated with a clinical outbreak in livestock during 1952-1954 (Hanson and Karstad, 1956). In these surveys, feral swine showed a very high prevalence of antibody to VS virus. Similar results were reported during a comprehensive serologic survey for serum neutralizing antibodies to VS virus (VSV) among wild and domestic animals of Ossabaw Island, Georgia (Fletcher et al., 1985).

Lesions attributable to VS were first suspected in feral swine in June 1957 in four small pigs from Champney Island, Georgia. Although not confirmed by virus isolation, antibodies to VSV were detected in these animals 2 wk after vesicular lesions were observed (Hanson and Karstad, 1959a). The first isolations of virus from feral swine were made during 1983 from two swine with vesicular lesions on Ossa-

baw Island, Georgia (Stallknecht et al., 1985). To date, all seropositive and culture-positive results in feral swine from the United States have been New Jersey (NJ) serotype.

The high prevalence (86%) of antibodies to VSV in feral swine coupled with a range which conformed to the recognized enzootic areas in the southeastern United States led Hanson and Karstad (1959b) to suggest that swine were "an important factor in perpetuation of VS." Although there has been no additional evidence to prove this, the value of this species as a sensitive indicator of VSV activity has been established through serial bleeding of sentinel wild swine (Hanson and Karstad, 1957; Stallknecht et al., 1985).

Feral swine are present in 19 states primarily in the Southeast, Southwest, California, and Hawaii. This study reports on a serologic survey of populations of wild swine throughout the United States for VSV antibodies to both New Jersey and Indiana serotypes.

### MATERIALS AND METHODS

Wild swine were collected with live traps, catch-dogs, and firearms. Serum was collected from all animals. Swine were assigned to one of four age classes using age criteria described previously for domestic (Sisson and Grossman, 1953) and wild swine (Matschke, 1967).

Frozen sera were submitted to the Diagnostic Virology Laboratory, National Veterinary Services Laboratories (NVSL), Animal and Plant Health Inspection Service, United States Department of Agriculture, Ames, Iowa. Sera were tested serologically for antibodies to both New Jersey and Indiana serotypes of VSV utilizing

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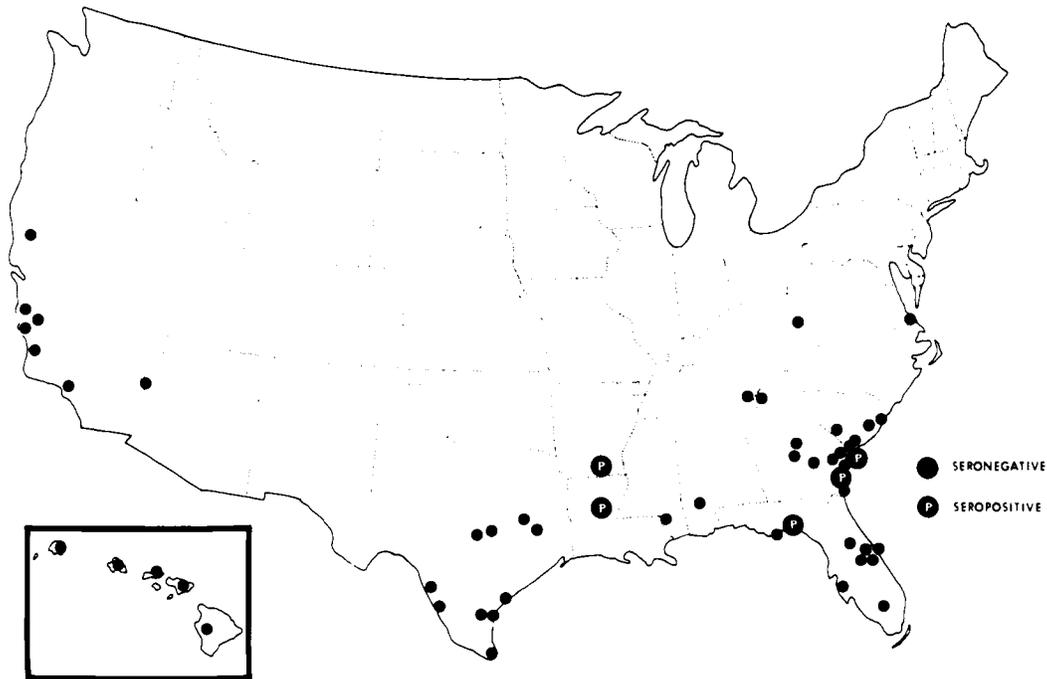


FIGURE 1. Feral swine collection sites in United States showing areas of vesicular stomatitis virus NJ seropositive results.

the microtiter serum neutralization test (NVSL, 1981). Antibody titers of 1:32 or greater were considered positive.

### RESULTS

From January 1979 to September 1985, 941 serum samples were obtained from wild swine collected from 53 locations in 15 states (Table 1, Fig. 1). Of these, 382 were examined during necropsy for signs of vesicular lesions. Serologic results are presented in Table 1. Antibodies to VSV (New Jersey serotype) were observed in five populations in four states, viz., Arkansas, Florida, Georgia, and Louisiana. Within these five populations, antibody prevalences ranged from 10 to 100% with 51.7% of the combined swine within these areas testing seropositive. High antibody titers ( $\geq 1:256$ ) were observed in 92% of seropositive swine. Antibody titers varied greatly between and within locations, with lower titers (1:32–1:128) restricted to se-

ropositive swine from St. Marks National Wildlife Refuge, Wakulla County, Florida, and Ossabaw Island, Chatham County (1979), and Rhetts Island, McIntosh County, Georgia. Age class data are presented in Table 2.

No antibodies to Indiana serotype VSV were detected. No lesions compatible with clinical VS were observed.

### DISCUSSION

Antibodies to VSV (NJ) in feral swine were restricted to five locations in the southeastern coastal plain, an area where VS has been reported to be enzootic (Hanson and Karstad, 1956). Our data would suggest that VSV activity may be very localized since antibodies were detected in only 5 of 32 (16%) coastal plain populations surveyed. This localized distribution of VS has been reported previously during clinical outbreaks in livestock (Jonkers, 1967).

TABLE 1. Results of serologic tests for vesicular stomatitis virus (New Jersey and Indiana serotypes) among wild swine of the United States.

State	County	Location(s)	Year(s)	Serologic results <sup>a</sup>	
Alabama	Clarke	Fred T. Stimpson/Hal's Lake	1980	0/7	
Arizona	Mohave	Havasu N.W.R.	1983	0/13	
Arkansas	Union	Felsenthal N.W.R.	1980	10/10 (NJ)	
California	Merced	Cottonwood Creek W.M.A.	1979-1981	0/7	
	Monterey	Deer Valley Ranch	1979-1981	0/48	
	San Luis Obispo	Hardens Wildlife Management, Inc.	1979-1981	0/20	
	Santa Clara	Private Land, Mt. Hamilton Range	1979-1981	0/17	
	Tehama	Dye Creek Preserve	1979-1981	0/62	
	Ventura	San Clemente Island, U.S.N.	1979-1981	0/18	
	Florida	Sarasota	Myakka State Park	1979	0/24
Lake		E.K. Ranch	1980	0/1	
Orange		Tosohatchee	1980	0/10	
			1981	0/6	
Osceola		Deseret Ranch	1980	0/10	
Osceola		Prairie Lakes State Park	1980	0/10	
Brevard		Merritt Island	1981	0/10	
Franklin		St. Vincent N.W.R.	1981	0/10	
Hendry		Alico Ranch	1981	0/10	
Wakulla		St. Marks N.W.R.	1981	4/12 (NJ)	
Georgia		Chatham	Ossabaw Island	1979 <sup>b</sup>	5/10 (NJ)
				1980 <sup>c</sup>	48/92 (NJ)
		Wilkinson	Napier Plantation	1979	0/5
		Camden	Cumberland Island	1980	0/15
	Telfair	Horse Creek W.M.A.	1980	0/10	
	Liberty	Thompson Pasture	1980	0/12	
	Liberty	Fort Stewart	1980-1981	0/12	
	McIntosh	Rhetts Island	1981	1/12 (NJ)	
	Chatham	Savannah N.W.R.	1984	0/5	
	Liberty	St. Catherines Island	1985	0/13	
	Houston	Oaky Woods	1985	0/9	
Hawaii	Maui	Molokai	1980	0/11	
			1983	0/66	
			1985	0/10	
		Hawaii	Hawaii	1983	0/62
		Honolulu	Oahu	1985	0/10
		Kauai	Kauai	1985	0/10
		Maui	Maui	1985	0/10
Louisiana	Grant	Georgetown	1980	7/10 (NJ)	
Mississippi	Pearl River	Pearl River	1980	0/10	
North Carolina	Swain	Great Smoky Mountains N.P.	1980	0/8	
South Carolina	Georgetown	Hobcaw Barony	1979	0/21	
			1980	0/24	
		Berkeley	Francis Marian N.F.	1980	0/10
		Jasper	Palmetto Bluff	1980	0/9
		Aiken	Savannah River Plant	1984	0/10
	1985			0/8	
	Tennessee	Blount	Great Smokey Mountains N.P.	1979	0/11
Texas	Anderson	Valley View Cattle Company	1985	0/12	
	Aransas	Aransas N.W.R.	1985	0/13	
	Burnet	Goodrich/Turbiville	1985	0/11	
	Cameron	Laguna Atascosa N.W.R.	1985	0/11	
	Dimmitt	Piloncillo Ranch	1985	0/14	

TABLE 1. Continued.

State	County	Location(s)	Year(s)	Serologic results <sup>a</sup>
	Kleberg	King Ranch (Santa Gertrudis)	1985	0/10
		King Ranch (Laureles)	1985	0/13
	Llano	Granite Hills Ranch	1985	0/9
	Trinity	Temple-Eastex	1985	0/10
	Webb	Callaghan Ranch	1985	0/20
Virginia	Princess Anne	False Cape N.W.R.	1980	0/5
West Virginia	Boone/Logan	Bear Tree Hollow	1979–1981	0/13
				75/941

<sup>a</sup> Number seropositive/number tested.

<sup>b</sup> Jenney et al., 1980, op. cit.

<sup>c</sup> Fletcher et al., 1985, op. cit.

Prior serologic results are available on only two of the wild swine populations sampled and both have a history of VSV (NJ) activity. Antibodies to VSV (NJ) were reported from white-tailed deer (*Odocoileus virginianus*) from Ossabaw Island in 1965 (Jenney et al., 1970) and from feral swine in 1969 (Jenney and Brown, 1972) and 1978 (Jenney et al., 1980). Likewise, antibodies to VSV and clinical disease in swine were reported during the mid 1950's from the Altamaha Wildlife Management Area which includes adjacent Rhetts and Champney islands (Hanson and Karstad, 1959a). It is interesting that despite the persistence of VS within these areas, it has not spread to adjacent populations of swine or white-tailed deer occupying similar habitats (Stallknecht and Erickson, 1986). This implies either that a precise set of ecological conditions must be met for this virus to exist in a given area or that potential reservoirs would have to be very short-ranged.

Variations in prevalences of antibody in the five seropositive populations may be attributable to fluctuations in annual activity or to the seasonal nature of viral activity. Both of these variances have been reported from Ossabaw Island (Stallknecht et al., 1985). The absence of VSV antibodies in younger age classes collected at St. Marks National Wildlife Refuge and

Rhetts Island (Table 2) may have resulted from sampling in years of little or no viral activity at these sites. Furthermore, since these populations were sampled during the winter and early spring months, swine less than 8 mo of age may not have had an opportunity for viral exposure. Except for these two areas, antibody prevalence in seropositive populations equaled or exceeded 50%.

In seropositive populations, prevalences were age-related (Table 2) and reached 86% in the 24+-mo age class. In three populations, 100% of swine were seropositive at this age. These high antibody prevalences coupled with observed high titers reinforce previous reports that wild swine represent an excellent indicator species for delineation and detection of VS NJ foci. This age relationship further indicates that age structure of the sample could easily bias prevalence estimates due to antibody persistence.

The absence of lesions was not unexpected. In all, only 54 of 382 necropsied swine were from seropositive herds. This relatively small sample size was taken without regard for the seasonal nature of viral activity (Clower and Mikel, 1953; Hanson and Karstad, 1959b). Furthermore, as evident by the fact that only two vesicular lesions were observed in 111 seroconverting swine on Ossabaw Island,

TABLE 2. Prevalence of seropositive swine by age class sampled from areas of detected vesicular stomatitis virus (New Jersey serotype) activity.

Area	Age class (mo)			
	<8	8-14	14-24	24+
Arkansas				
Felsenthal National Wildlife Refuge	3/3	—	3/3	4/4
Florida				
St. Marks National Wildlife Refuge	0/3	—	1/6	3/3
Georgia				
Ossabaw Island	7/40	1/6	26/34	19/22
Rhetts Island	0/6	—	0/2	1/3
Louisiana				
Grant Parish	—	—	3/6	4/4
Total	10/52 (19%)	1/6 (17%)	33/51 (65%)	31/36 (86%)

clinical VS can be uncommon even in areas of high viral exposure (Stallknecht et al., 1985).

The possibility that wild swine are a component in the maintenance cycle of enzootic VS in the Southeast cannot at this point be supported or dismissed. Wild swine generally have a higher prevalence of antibodies to VSV than other mammalian wildlife. Furthermore, extensive serologic surveillance of white-tailed deer populations support the observed localized distribution of VS within the Southeast and to date, all seropositive results from deer came from areas where wild swine were present (Jenney et al., 1970; Jenney and Brown, 1972; Stallknecht and Erickson, 1986). This evidence however is still circumstantial, and if a relationship does indeed exist, it may be that wild swine simply occupy habitat types where VSV is likely to be maintained.

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#### LITERATURE CITED

- CLOWER, T. B., AND C. J. MIKEL. 1953. Vesicular stomatitis in swine. *Proc. U.S. Livestock Sanit. Assoc.* 57: 320-321.
- FLETCHER, W. O., D. E. STALLKNECHT, AND E. W. JENNEY. 1985. Serologic surveillance for vesicular stomatitis on Ossabaw Island, Georgia. *J. Wildl. Dis.* 21: 100-104.
- HANSON, R. P., AND L. H. KARSTAD. 1956. Enzootic vesicular stomatitis. *Proc. U.S. Livestock Sanit. Assoc.* 60: 228-292.
- , AND ———. 1957. Further studies on enzootic vesicular stomatitis. *Proc. U.S. Livestock Sanit. Assoc.* 61: 300-307.
- , AND ———. 1959a. Feral swine in the southeastern United States. *J. Wildl. Manage.* 23: 64-74.
- , AND ———. 1959b. Feral swine as a reservoir of vesicular stomatitis virus in the southeastern United States. *Proc. U.S. Livestock Sanit. Assoc.* 62: 309-315.
- JENNEY, E. W., AND C. L. BROWN. 1972. Surveillance for vesicular stomatitis in the United States—January, 1968 through July, 1972. *Proc. U.S. Anim. Health Assoc.* 76: 183-193.
- , G. A. ERICKSON, W. W. BUISCH, W. C. STEWART, AND M. A. MIXON. 1980. Surveillance for vesicular stomatitis in the United States 1972 through 1979. *Proc. Am. Assoc. Vet. Lab. Diagnost.* 23: 83-89.
- , F. A. HAYES, AND C. L. BROWN. 1970. Survey for vesicular stomatitis virus neutralizing an-

- tibodies in serums of white-tailed deer (*Odocoileus virginianus*) of the southeastern United States. *J. Wildl. Dis.* 6: 488–493.
- JONKERS, A. H. 1967. The epizootiology of the vesicular stomatitis virus: A reappraisal. *Am. J. Epidemiol.* 86: 286–291.
- MATSCHKE, G. H. 1967. Aging European wild hogs by dentition. *J. Wildl. Manage.* 31: 109–113.
- NATIONAL VETERINARY SERVICES LABORATORIES. 1981. Serologic microtitration techniques. USDA, APHIS, Nat. Vet. Serv. Lab., Ames, Iowa, 48 pp.
- SCHOENING, H. W. 1943. Vesicular stomatitis in swine. *Proc. U.S. Livestock Sanit. Assoc.* 47: 85–86.
- SISSON, S., AND J. D. GROSSMAN. 1953. *The Anatomy of the Domestic Animals*. W. B. Saunders Co., Philadelphia, Pennsylvania, 972 pp.
- STALLKNECHT, D. E., AND G. A. ERICKSON. 1986. Antibodies to vesicular stomatitis New Jersey type virus in a population of white-tailed deer. *J. Wildl. Dis.* 22: 250–254.
- , V. F. NETTLES, W. O. FLETCHER, AND G. A. ERICKSON. 1985. Enzootic vesicular stomatitis New Jersey type in an insular feral swine population. *Am. J. Epidemiol.* 122: 876–883.

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