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## FERAL SWINE AS A POTENTIAL AMPLIFYING HOST FOR VESICULAR STOMATITIS VIRUS NEW JERSEY SEROTYPE ON OSSABAW ISLAND, GEORGIA

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**ABSTRACT:** Sentinel feral swine (*Sus scrofa*) on Ossabaw Island, Georgia (USA), were serologically monitored for antibodies to vesicular stomatitis New Jersey serotype (VSNJ) virus from 17 April to 27 August 1990. Seroconversions to VSNJ virus were detected in 24% of swine island-wide. Differences in the incidence of seroconversion were detected between swine sampled in the Pleistocene and Holocene formations of the island suggesting that the presence of virus is forest type dependent. Based on the consistency in onset and spatial distribution of seroconversions with data from 1981 to 1985, this is a very stable host-parasite system.

Sequential virus isolation attempts from nasal swabs, tonsil swabs, and blood were made on a subsample of 54 sentinel swine from 9 May to 4 July 1990. The VSNJ virus was isolated from five swine from 16 May to 20 June. Vesicular lesions were detected on only two of these animals. Although infections in these feral swine were short-lived (<7 days) and were followed by a strong neutralizing antibody response, VSNJ virus was detected in a single group of swine for a period exceeding 1 month. From these data, it appears that feral swine could provide a source of virus to feeding arthropods for extended periods of time. The failure to detect a viremia in these animals, however, indicates that a source other than blood may be required for transmission to occur.

**Key words:** Epizootiology, feral swine, Ossabaw Island, seroconversion, *Sus scrofa*, vesicular stomatitis virus, virus isolation.

### INTRODUCTION

Vesicular stomatitis (VS), a disease of cattle, horses, swine, and humans is caused by related viruses in the genus *Vesiculovirus* of the family Rhabdoviridae. In the United States, epizootics in livestock have been associated with two serotypes of vesicular stomatitis virus, New Jersey (VSNJ) and Indiana (VSI). Of these serotypes, VSNJ virus has occurred more frequently with major clinical outbreaks in livestock in the United States reported as recently as 1982-83 (U.S. Department of Agriculture, 1982; Jenney et al., 1984). The complete maintenance and transmission cycle for VSNJ virus, however, remains undefined.

Serological evidence of VSNJ virus first was detected from feral swine (*Sus scrofa*) between 1952 and 1954 in Georgia (USA) (Hanson and Karstad, 1956). Although serum neutralizing antibodies to VSNJ virus subsequently have been detected in feral

swine populations in Arkansas, Florida, Georgia, and Louisiana (USA) (Stallknecht et al., 1986), Ossabaw Island, Georgia (31°47'N, 81°07'W) represents the only enzootic area in the United States where VSNJ virus infections have been confirmed through virus isolation (Stallknecht et al., 1985; Corn et al., 1990).

Following epizootiological studies of VSNJ virus on Ossabaw Island using both feral and domestic sentinel swine, we observed both seasonal and geographical patterns of infection consistent with arthropod transmission (Stallknecht et al., 1987). Subsequent field studies with *Lutzomyia shannoni*, the only species of phlebotomine sand fly reported from Ossabaw Island, resulted in VSNJ virus isolations from both male and female sand fly pools (Corn et al., 1990; Comer et al., 1992). Based on experimental studies of VSNJ virus in *L. shannoni*, we also demonstrated virus replication, transovarial transmission, and bite



transmission of the virus to hamsters and suckling mice (Comer et al., 1990). Although these observations support the hypothesis that *L. shannoni* is a capable biological vector, the low frequency of transovarial transmission (<2%) observed in these experiments is evidence that periodic viral amplification is needed for VSNJ virus maintenance.

In a serological survey of 24 species of wild and domestic vertebrates on Ossabaw Island, Fletcher et al. (1985) reported that feral swine had the highest prevalence (53%) of antibodies to VSNJ virus. The primary objective of the present study was to determine the extent and duration of VSNJ virus shedding or viremia in naturally infected feral swine on this island. Since recent work with white-tailed deer (*Odocoileus virginianus*) on Ossabaw Island indicated a strong relationship between VSNJ virus antibody prevalence and geological formation of the island (Fletcher et al., 1991), a secondary objective was to test this relationship in swine.

#### MATERIALS AND METHODS

Free-ranging sentinel feral swine (<8 mo of age) were trapped, ear-tagged, and serologically monitored for antibodies to VSNJ virus as described by Stallknecht et al. (1985, 1987). Traps were located throughout the island (Fig. 1). When possible, sentinel swine were bled once every week. In addition to the collection of serum samples, all animals were checked for vesicular lesions when bled.

Serum samples were tested for antibodies to VSNJ and VSI viruses by serum neutralization tests at the National Veterinary Services Laboratories, Ames, Iowa (USA) (National Veterinary Services Laboratories, 1981). Serum samples which neutralized virus at  $\geq 1:32$  dilution were considered positive. A sentinel animal was defined as any animal that tested negative (<1:32) for antibodies to VSNJ virus at one or more previous captures. Seroconversions in these sentinel swine were recognized only if a rise in antibody titer ( $\geq 1:32$ ) was observed and maintained throughout all subsequent samplings. Differences in incidence of seroconversion by location and geological formation were tested using the G-statistic with Yates' correction for continuity (Sokal and Rohlf, 1981).

In addition to serological testing, nasal swabs,

tonsil swabs, and heparinized blood samples were collected from a subsample of sentinel swine from seven trap sites (Fig. 1) from 9 May to 4 July 1990. Sites and sample period for virus isolation attempts were selected based on the high incidence of seroconversion previously observed in sentinel swine during May and June in the area (Stallknecht et al., 1985, 1987; Corn et al., 1990). Samples for virus isolation were collected one day each week, were stored on wet ice, and were evaluated within 24 hr of collection. Swabs were collected in 2 ml of transport medium consisting of tryptose broth (Difco Laboratories, Detroit, Michigan, USA) supplemented with antibiotics (1,000 units penicillin G, 1 mg streptomycin, 0.25 mg gentamicin sulfate, 0.5 mg kanamycin monosulfate, and 50 units mycostatin/ml) (Sigma Chemical Company, St. Louis, Missouri, USA). If suspected vesicular lesions were detected, swabs and skin samples from the affected area also were collected in transport medium.

For virus isolation, swab samples were mixed thoroughly and centrifuged at  $1,500 \times g$  for 15 min. Blood samples were diluted 1:10 in Dulbecco's phosphate buffered saline solution (D-PBS) (Sigma). Tissue samples were triturated in a tissue grinder and resuspended in 1 ml D-PBS. All samples (100  $\mu$ l) were inoculated onto monolayers of Vero cells (American Type Culture Collection, Rockville, Maryland, USA) prepared in 6-well cell culture plates. Remaining portions of all samples were frozen at  $-70^\circ\text{C}$ . Inoculum was absorbed for 1 hr at  $33.5^\circ\text{C}$ , and plates were rocked at 15-min intervals. Inoculated cell cultures then were washed with 2 ml D-PBS. Maintenance medium (3 ml) consisting of Eagle's minimum essential medium (MEM) with Earle's salts supplemented with 1% heat inactivated fetal bovine serum and antibiotics (50 units penicillin G, 50  $\mu$ g streptomycin, and 0.125  $\mu$ g amphotericin B/ml) (Sigma) was added to each well. Plates were incubated at  $33.5^\circ\text{C}$  in a humidified 5%  $\text{CO}_2$  atmosphere for 5 days. A second passage was done on day 5.

Medium was collected from all cell cultures exhibiting cytopathic effect. The presence of a rhabdovirus was confirmed by negative staining and electron microscopy (Doan and Anderson, 1987). Samples containing rhabdovirus were submitted to NVSL for VSNJ virus confirmation through the direct fluorescent antibody test using fluorescein conjugated hyperimmune bovine serum.

To verify infectivity of positive samples, original field samples were thawed, and a second VSNJ virus isolation was attempted as previously described. Viral titers of these samples were determined at this time by endpoint titration in 96-well cell culture plates. For each ti-

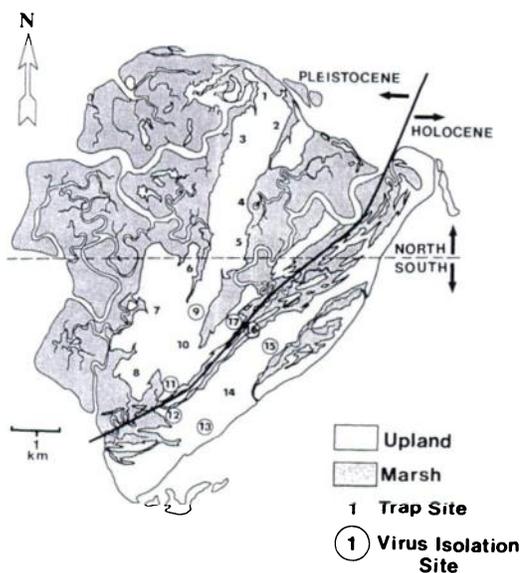


FIGURE 1. Locations of sentinel swine traps on Ossabaw Island, Georgia showing north/south and Pleistocene/Holocene divisions. Bar = 2 km.

tration, eight replicate wells were inoculated at 10-fold dilutions ranging from  $1 \times 10^{-1}$  to  $1 \times 10^{-11}$ . Microtiter plates were read at 48 hr, and viral titers were calculated as described by Reed and Muench (1938). To validate negative virus isolation results, a second virus isolation from frozen samples was attempted from all virus isolation-negative samples that had been collected from swine 1 wk before or at the time of a detected seroconversion.

**RESULTS**

From 17 April to 27 August 1990, 243 feral swine were serologically monitored and 1,564 serum samples were collected (6.4 samples/sentinel swine). Seroconver-

TABLE 2. Isolation attempts for vesicular stomatitis virus New Jersey serotype from 54 free-ranging sentinel swine on Ossabaw Island, Georgia, 9 May to 4 July 1990.

Trap location	Number of sentinel swine	Number (%) of seroconversions	Number of virus isolation attempts*	Number of VSNJ virus isolations
9	6	0 (0%)	23	0
11	5	1 (20%)	14	0
12	8	8 (100%)	33	0
13	13	10 (77%)	76	5
15	7	2 (29%)	36	0
16	6	0 (0%)	17	0
17	9	0 (0%)	36	0
Total	54	21 (39%)	235	5

\* Each virus isolation attempt included nasal swab, tonsil swab, blood, plus lesion material (swab and skin samples) when present.

sion was initially detected on 16 May. As of 27 August, 24% of the sentinel swine had seroconverted (Table 1). Most seroconversions were detected between 6 June and 7 July.

Seroconversions were detected in swine surveyed at locations 10 through 15 (Fig. 1). Using a Chi-square test, differences ( $P < 0.0001$ ) in incidence of seroconversion at the end of the sample period (27 August 1990) were detected between the north (0%) and south (32%) areas and between Pleistocene (2%) and Holocene (62%) areas.

Virus isolations from swab, blood, and lesion samples were attempted from a subsample of 54 swine on 235 occasions (4.3

TABLE 1. Incidence of seroconversion to vesicular stomatitis virus New Jersey serotype in free-ranging sentinel feral swine on Ossabaw Island, Georgia, by area and geological formation, 1990.

Area	Trap location number	Sample periods				
		17 April to 6 May	7 May to 5 June	6 June to 3 July	4 July to 29 July	30 July to 27 August
North	(1 to 5)	—	0/50 (0%)*	0/49 (0%)	0/37 (0%)	0/19 (0%)
South	(6 to 17)	0/36 (0%)	11/120 (9%)	33/109 (30%)	31/91 (34%)	19/60 (32%)
Pleistocene	(1 to 11, 17)	0/18 (0%)	2/133 (2%)	6/110 (5%)	6/93 (6%)	1/50 (2%)
Holocene	(12 to 16)	0/18 (0%)	9/57 (16%)	27/48 (56%)	25/35 (71%)	18/29 (62%)
Island-wide	(1 to 17)	0/36 (0%)	11/170 (6%)	33/158 (21%)	31/128 (24%)	19/79 (24%)

\* Number seroconverted/number sentinels captured (% seroconverted).

TABLE 3. Virus isolation and serologic results from five sentinel feral swine from which vesicular stomatitis virus New Jersey serotype was isolated on Ossabaw Island, Georgia, 1990. Antibody titers  $\geq 32$  were considered positive.

Swine number	Date sampled							
	9 May	16 May	23 May	30 May	6 June	13 June	20 June	27 June
343								
Virus isolation	Neg	Pos	Neg	Neg	— <sup>a</sup>	—	—	Neg
Serology <sup>b</sup>	Neg	Neg	512	128	256	128	—	128
331								
Virus isolation	Neg	Neg	Pos	Neg	Neg	Neg	—	Neg
Serology	Neg	Neg	Neg	256	1,024	1,024	—	512
389								
Virus isolation	Neg	Neg	—	Pos	Neg	Neg	Neg	Neg
Serology	Neg	Neg	Neg	Neg	512	256	$\geq 8,192$	1,024
387								
Virus isolation	Neg	Neg	Neg	—	Pos	Neg	—	Neg
Serology	Neg	128	256	64	Neg	1,024	—	512
309								
Virus isolation	Neg	—	—	—	Neg	Neg	Pos	Neg
Serology	Neg	Neg	Neg	Neg	Neg	Neg	Neg	$\geq 8,192$

<sup>a</sup> —, not tested.

<sup>b</sup> Titers reported as reciprocal of final dilution giving a positive reaction.

isolation attempts/sentinel swine) (Table 2). Seroconversion incidence at the seven trap sites monitored by virus isolation ranged from 0 to 100%. The VSNJ virus was isolated from nasal and tonsil swabs collected from five swine at trap site 13 (Table 3), but was not isolated from any of the 235 blood samples. Isolations of VSNJ virus were made from this group of animals from 16 May to 20 June.

All isolations were detected by cytopathic effect in cell culture by day two on initial virus isolation attempt. All swine from which virus was recovered seroconverted 1 wk after VSNJ virus was detected. Virus was not isolated from any of the five swine more than once, even though three and five of these animals were sampled 1 wk before and 1 wk after the virus isolation, respectively.

Vesicular lesions were detected on only two of five swine from which VSNJ virus was isolated (Table 4). In both cases, VSNJ virus also was isolated both from lesion swabs and from tissue samples associated with these lesions.

Only one virus isolation was not con-

firmed through a second isolation from frozen material (Table 4). Viral titers of these samples generally were low, but ranged up to  $1 \times 10^{5.3}$  CCID<sub>50</sub>/ml transport media and  $1 \times 10^{7.8}$  CCID<sub>50</sub>/2 × 3 mm of skin sample for lesion swabs and skin samples, respectively (Table 4). Thirty-three additional virus isolation attempts were made from previously negative samples which had been collected 1 wk before or at the time of a detected seroconversion. All samples were negative on these second isolation attempts.

#### DISCUSSION

The date of the initially detected seroconversion (16 May) was consistent with results reported during 1982, 1983 (Stallknecht et al, 1985), 1984, 1985 (Stallknecht et al., 1987), and 1988 (Corn et al., 1990); thus this is a very stable host-parasite system. Likewise, incidence of seroconversion differed greatly between the north and south areas, as had been reported for 1982 through 1985 (Stallknecht et al., 1985; 1987). As suggested by Fletcher et al. (1991) in an analysis of white-tailed deer serologic

TABLE 4. Sentinel feral swine swab and tissue samples from which vesicular stomatitis virus New Jersey serotype was isolated.

Swine number	Vesicular lesions present	Original VSNJ virus isolation	Confirmatory reisolation result	Virus titer
343	No	Nasal swab	Positive	$<10^{1.5}/\text{ml}$
		Tonsil swab	Positive	$<10^{1.5}/\text{ml}$
331	No	Nasal swab	Negative	$<10^{1.5}/\text{ml}$
389	Yes	Nasal swab	Positive	$10^{3.5}/\text{ml}$
		Lesion swab	Positive	$10^{1.9}/\text{ml}$
		Lesion tissue	Positive	$10^{5.4}/2 \times 3 \text{ mm skin}$
387	Yes	Nasal swab	Positive	$10^{2.2}/\text{ml}$
		Tonsil swab	Positive	Not done
		Lesion swab	Positive	$10^{5.3}/\text{ml}$
		Lesion tissue	Positive	$10^{7.4}/2 \times 3 \text{ mm skin}$
309	No	Nasal swab	Positive	$<10^{1.5}/\text{ml}$

data from Ossabaw Island, these spatial differences probably relate to variation in forest types associated with the Pleistocene and Holocene formations of the Island. It is possible that the old-growth maritime forest type associated with the Holocene formation provides necessary habitat for *L. shannoni* which has been shown to be a biological vector of VSNJ virus (Comer et al., 1990). When the swine data were grouped by these geologic areas, differences in incidence of seroconversion were extreme, ranging from 2% for swine sampled in the Pleistocene formation to 62% in swine sampled from the Holocene formation. As most of the Holocene area is included in the southern area (Fig. 1), the higher incidence of seroconversion associated with this geologic area also is explained by this previously detected north/south variation.

Although virus was isolated from both nasal and tonsil swabs of swine with and without vesicular lesions, availability of virus from these sites was short-lived and there was no indication of viremia. These field infections were followed by a strong neutralizing antibody response which could be detected within 7 days of virus isolation. These observations are consistent with results from experimental infections in swine (van der Maaten, 1984, 1986; Redelman

et al., 1989) and other vertebrates (Tesh et al., 1969). However, although these infections were detectable in individual swine over a very short period, we isolated virus from a single comingling group of these animals for a period exceeding 1 mo. Such an extended period of infection in this small group of animals could provide ample opportunity for transmission of VSNJ virus to biting arthropods, provided that the virus is actually available to such potential vectors.

The failure to consistently demonstrate viremia in this and other studies remains as the major argument against an arthropod-based maintenance cycle (Jonkers, 1967). Viral transmission to feeding arthropods in the absence of detectable viremia, however, has been reported with Thogoto virus and *Rhipicephalus appendiculatus* (Jones et al., 1987). We observed high viral titers in skin samples associated with vesicular lesions. Likewise, high VSNJ virus titers have been reported from vesicular fluid (Hanson and Brandley, 1957). Whether *L. shannoni* or other biting arthropods actively feed at these sites or become infected through ingestion of infected tissue fluids, however, remains to be demonstrated.

A great deal of circumstantial evidence links feral swine to the epizootiology of

VSNJ virus in the southeastern United States. During the 1950's and 1960's, VS was common in domestic and free-ranging swine throughout this area (Clower and Mikel, 1953; Schoening, 1954; Hanson and Karstad, 1956; Jenney, 1967). In an early investigation of VS, Hanson and Karstad (1959a, b) concluded that feral swine were likely to be "an important factor in the perpetuation of VS." Even today, all six areas where VSNJ virus neutralizing antibodies have been detected in white-tailed deer (Jenney et al., 1970; Stallknecht and Erickson, 1986; Fletcher et al., 1991) are characterized by the presence of feral swine populations. The Ossabaw strain of VSNJ virus is very similar genetically to those strains circulating in swine populations in the southeastern United States during the 1950's (Bilsel et al., 1990; Vernon et al., 1990); thus the virus is well adapted to this host system. Finally, feral swine population dynamics are compatible with a *L. shannoni*/feral swine VSNJ virus maintenance cycle. With peak breeding and farrowing occurring in late fall and early winter, respectively, introduction of susceptible pigs without maternal antibodies into the population coincides with the precise time that *L. shannoni* populations are emerging and expanding (Corn et al., 1990). Furthermore, the reproductive potential and relatively high turnover in this swine population assures that a large proportion of these animals will be susceptible to VSNJ virus annually.

Actual transmission of VSNJ virus between swine and *L. shannoni* must be demonstrated before such a maintenance cycle can be proven. Such a relationship between this vector and a potential amplifying host would be significant since it would represent a very recent and habitat dependent adaptation between an introduced Old World species and a New World vector and virus.

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