Contact transmission of vesicular stomatitis virus New Jersey in pigs

David E. Stallknecht, PhD; Deborah E. Perzak, BS; Leslie D. Bauer, BS; Molly D. Murphy, BS; Elizabeth W. Howerth, DVM, PhD

Objective—To determine how viral shedding and development or lack of clinical disease relate to contact transmission of vesicular stomatitis virus New Jersey (VSV-NJ) in pigs and determine whether pigs infected by contact could infect other pigs by contact.

Animals—63 pigs.

Procedure—Serologically naive pigs were housed in direct contact with pigs that were experimentally inoculated with VSV-NJ via ID inoculation of the apex of the snout, application to a scarified area of the oral mucosa, application to intact oral mucosa, or ID inoculation of the ear. In a second experiment, pigs infected with VSV-NJ by contact were moved and housed with additional naive pigs. Pigs were monitored and sampled daily for clinical disease and virus isolation and were serologically tested before and after infection or contact.

Results—Contact transmission developed only when vesicular lesions were evident. Transmission developed rapidly; contact pigs shed virus as early as 1 day after contact. In pens in which contact transmission was detected, 2 of 3 or 3 of 3 contact pigs were infected.

Conclusions and Clinical Relevance—Transmission was lesion-dependent; however, vesicular lesions often were subtle with few or no clinical signs of infection. Contact transmission was efficient, with resulting infections ranging from subclinical (detected only by seroconversion) to clinical (development of vesicular lesions). Long-term maintenance of VSV-NJ via contact transmission alone appears unlikely. Pigs represent an efficient large-animal system for further study of VSV-NJ pathogenesis and transmission. (*Am J Vet Res* 2001;62:516–520)

Vesicular stomatitis (VS), which is caused by related viruses of the genus Vesiculovirus in the family Rhabdoviridae, is a disease of cattle, horses, and swine. Two of these viruses, including **vesicular stomatitis virus New Jersey (VSV-NJ)** and Indiana, have been associated with VS in the United States as recently as 1997 and 1998, respectively.[1]

Although VS may be associated with substantial direct and indirect economic losses to livestock producers,[2] the epidemiologic features of this disease are not well defined. Transmission of VSV-NJ may occur via biological vectors,[3-7] mechanical vectors,[8] and animal-to-animal contact,[9-11] but the relative importance of these routes in the spread of this virus among livestock populations is unclear. In addition, possible relationships between the presence of clinical disease, characterized by vesicular lesions, and VSV transmission have received limited attention. Because VS control and eradication are based on the detection of infection through identification of clinically affected animals and the elimination of transmission via herd quarantine, an understanding of transmission potential is required.

Results of studies of VSV-NJ in naturally infected feral swine indicate that most of these infections are subclinical.[12-14] Experimental infections indicate that swine may be infected by various routes compatible with mechanical and contact transmission, with as little as 1×10^2 **median tissue culture infective doses ($TCID_{50}$)**, and that the clinical outcome (specifically the development of vesicular lesions) is dependent on route of inoculation and viral dose.[15,16] In natural and experimental infections, VSV-NJ was isolated from tonsil and nasal swab specimens from animals with and without clinical disease, and viral titers associated with these swab specimens exceeded the minimum dose required for infection.[17] However, a viremia was not detected in these animals. To date, viremia has not been reported in pigs or any domestic animal species, suggesting that domestic animals represent a dead-end host in a transmission cycle involving a biological vector. If this is the case, contact and mechanical transmission may represent the most important transmission routes after this virus is introduced into a domestic animal population.

The study reported here attempted to develop a model using domestic pigs to study the pathogenesis and transmission of VSV-NJ. The objectives of the study were to determine how viral shedding and development or lack of clinical disease relate to contact transmission of VSV-NJ in pigs and determine whether pigs infected by contact could infect other pigs by contact.

Materials and Methods

Swine—Sixty-three commercially available mixed-breed pigs, weighing approximately 10 kg were studied. All pigs were seronegative for VSV-NJ as indicated by results of serum neutralization tests (< 8) and were housed in insect-proof isolation units in groups of 3 to 5.

Experiment 1 study design—Four inoculation routes that experimentally induce a full range of clinical and subclinical VSV-NJ infections in inoculated pigs were used.[16,17] These included ID inoculation of the apex of the snout, application of virus to a scarified area of the oral mucosa,

Received Mar 20, 2000.
Accepted May 30, 2000.
From the Southeastern Cooperative Wildlife Disease Study (Stallknecht, Perzak, Bauer) and the Department of Pathology (Howerth, Murphy), College of Veterinary Medicine, University of Georgia, Athens, GA 30602.
Supported by a grant from the National Research Initiative Competitive Grants Program, United States Department of Agriculture.
The authors thank Tara Hoppes for technical assistance.

application of virus to intact oral mucosa, and ID inoculation of the ear. Previous work indicated that infections by ID inoculation of the snout and application of virus to scarified oral mucosa would result in development of vesicular lesions, viral shedding via tonsil and nasal cavity, and seroconversion. Infection by application to intact oral mucosa would result in viral shedding and seroconversion, and infection via ID inoculation of the ear would be detected only by seroconversion. The inoculum consisted of 100 µl (10^6 $TCID_{50}$ of VSV-NJ) of harvested vesicular fluid from a pig inoculated ID in the snout with a sand fly isolate of VSV-NJ from Ossabaw Island, Ga. This virus was originally isolated in Vero cells (ATCC CCL81) and had been passaged once in this cell line prior to infection of the pig.

Two replicates, consisting of 1 experimentally infected pig housed with 3 contact pigs, initially were done for each route of infection. To reduce the required number of pigs, 2 additional replicates per route of infection were done only for those routes of infection for which contact transmission was not detected by virus isolation or seroconversion. With the exception of 3 of the trials involving application of virus to intact oral mucosa, the 3 contact pigs were added to the pen with the inoculated animal 24 hours after the primary experimental infection.

All pigs were examined daily for clinical signs of disease, and swab specimens of the nasal cavity and tonsil of the soft palate were collected in 1 ml of transport medium consisting of **minimum essential medium (MEM)** supplemented with antimicrobials (1,000 U of penicillin G, 1 mg of streptomycin, 0.25 mg of gentamicin sulfate, 0.5 mg of kanamycin monosulfate, and 2.5 µg of amphotericin B per ml).[a] Inoculated pigs were examined and sampled on **postinoculation days (PID)** 1 through 14. Contact pigs were examined and samples were obtained on **postcontact days (PCD)** 1 through 15. To avoid pig-handling-related transmission in this and subsequent experiments, samples were obtained from all contact pigs in each pen before samples were obtained from the known infected pig. Serum for serologic testing was collected from all swine prior to infection or contact with infected pigs and just prior to euthanasia. Inoculated and contact pigs were euthanatized on PID 14 and PCD 15, respectively, by IV administration of an overdose of sodium pentobarbital.

Experiment 2 study design—The study design for objective 2 was illustrated (**Fig 1**). Two pigs (pen A) were inoculated ID in the apex of the snout with 100 µl of vesicular fluid containing 10^6 $TCID_{50}$ VSV-NJ. After 24 hours, 4 primary contacts (pigs 427, 438, 439, 440) were added to pen A. Inoculated pigs were examined daily, and swab samples of the tonsil and nasal cavity were collected for virus isolation on PID 1 through 10. Virus isolation was attempted daily from tonsil and nasal swab specimens from primary contact pigs starting on PCD 1. Immediately upon detection of virus (PCD 2 and 3), these pigs were moved to additional pens that each contained 3 secondary contact pigs. Primary contacts were examined, and samples were obtained for 10 additional days after this movement. Secondary contact pigs were examined, and samples were obtained daily from PCD 1 through 14. Serum for serologic testing was collected from all swine prior to infection or contact with infected pigs and just prior to euthanasia. Pigs were euthanatized on the final day of sampling by IV administration of an overdose of sodium pentobarbital.

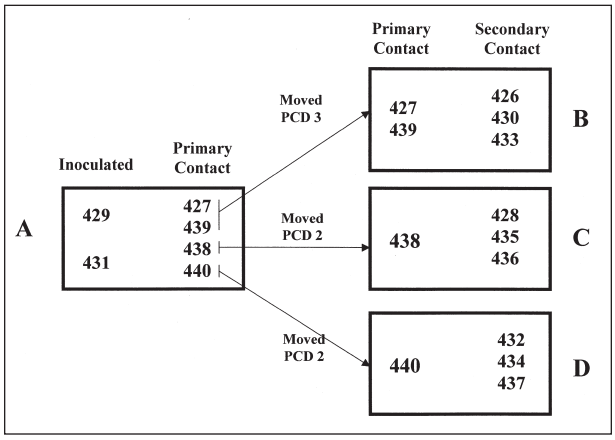

Figure 1—Schematic diagram of arrangement and movement of pigs in a study of contact transmission of vesicular stomatitis virus. A,B,C,D = Pens. PCD = Postcontact day. Three-digit numbers identify individual pigs.

Virus isolation and serologic testing—Virus isolation from swab specimens was attempted using Vero cells on 12-well culture plates. Briefly, swab specimens in transport medium were vortexed and centrifuged at 1,500 × g for 15 minutes. Individual wells, containing confluent monolayers of Vero cells in 2 ml of maintenance medium consisting of MEM supplemented with 3% fetal bovine serum and antimicrobials, were inoculated with 100 µl of supernatant from swab specimens. All virus isolations were completed immediately after sample collection, and residual supernatant from the samples was frozen at –70 C. If cytopathic effect was not observed within 72 hours, results for the sample were considered negative. If cytopathic effect was observed, cell culture fluids were collected and tested by use of immunohistochemistry, using hyperimmune mouse ascites fluid to confirm the presence of VSV-NJ.[15,16,b] All samples with positive results were quantified by endpoint titration in Vero cells using the stored frozen supernatant.[15,16] Serum was tested for antibodies against VSV-NJ by use of serum neutralization as described.[15] A 4-fold or greater increase in antibody titer was considered to indicate seroconversion.

Statistical analysis—Fisher exact tests were used to test whether transmission to contact pigs was dependent on detection of virus from the inoculated pig or dependent on the presence of lesions.[18] $P < 0.05$ was considered significant.

Results

Experiment 1—Clinical response, viral shedding, and seroconversion results for the inoculated pigs were consistent with our previous study[17] (**Table 1**). Vesicular lesions were restricted to pigs inoculated ID in the snout and by scarification of the oral mucosa. In pigs inoculated ID, vesicles developed at the site of inoculation on PID 2. The vesicles ruptured on PID 3, and there was subsequent loss of the necrotic epithelium that formed the vesicle roof and healing by granulation. Vesicles were almost healed by the day of euthanasia (PID 14). A secondary vesicle formed around the nostril of 1 pig on PID 5. In those infected by oral scarification, white exudate covered the scarified area on PID 1, and by PID 2, ≤ 1-mm vesicles had formed in the scarified area. By PID 3 to 6, large expanding vesicles had developed in the area of scarification. These vesicles had ruptured and were characterized by loss of their covering epithelium centrally and a peripheral rim of vesiculation. Resolution began on PID 7, with complete healing by PID 12.

Virus was isolated from swab specimens collected from pigs in all treatment groups except for those inoculated ID in the ear. For the 2 pigs inoculated ID in the

snout, virus was isolated from tonsil and nasal swab specimens on PID 2 through 5. Maximum VSV-NJ titers for tonsil and nasal swab specimens were 3.3 and 2.3 $TCID_{50}$, respectively. The VSV-NJ was isolated from PID 1 through 6 from both pigs inoculated by application of virus to scarified areas of the oral mucosa. Maximum viral titers for tonsil and nasal swab specimens in these pigs were ≥ 5.1 and 2.3 $TCID_{50}$, respectively. For the 4 pigs infected by application of virus to the intact oral mucosa, VSV-NJ was isolated from 4 to 6 consecutive days starting at PID 1. Isolations were restricted to tonsil swab specimens, with a maximum titer of ≥ 5.1 $TCID_{50}$. All of the inoculated pigs seroconverted.

Transmission to contact pigs was detected by use of virus isolation and serologic tests (Table 1). Virus was isolated from nasal and tonsil swab specimens collected from all 6 of the contact pigs associated with pigs inoculated ID in the snout. Contact-infected pigs shed virus for 6 consecutive days starting on PCD 2. Maximum titers for tonsil and nasal swab specimens were ≥ 5.1 and 2.3 $TCID_{50}$, respectively. These same VSV-NJ titers were observed in swab specimens collected from 3 contact pigs associated with 1 pig in which VSV-NJ was applied to scarified areas of the oral mucosa. Virus was detected in these contact pigs for 6 consecutive days starting on PCD 2. A single isolate came from a nasal swab specimen from a pig in contact with a pig that had been infected by applying the virus to intact oral mucosa. The titer of this swab specimen was ≤ 2.3 $TCID_{50}$, and on the basis of lack of seroconversion, it was believed that this represented surface contamination rather than infection. Contact transmission was dependent on detection of virus in the inoculated pigs ($P = 0.005$) and on the presence of vesicular lesions on the inoculated pigs ($P < 0.001$). No lesions were detected in any of the 12 pigs infected by contact.

Experiment 2—Transmission from inoculated pigs to primary contacts and from primary to secondary contact pigs was observed (**Table 2**). Virus was isolated from primary contacts as early as PCD 1. On this day, large vesicles had formed at the site of inoculation on the apex of the snout of both inoculated pigs. These vesicles ruptured by PID 4, with subsequent granulation and almost complete healing of the lesion by day of euthanasia (PID 10). Secondary vesicular lesions also developed in these pigs on PID 5 and con-

Table 1—Clinical, virus isolation, and serologic results for pigs in contact with pigs that were experimentally infected with vesicular stomatitis virus-New Jersey (VSV-NJ)

		Inoculated pig (n = 1)			Contact pigs (n = 3)		
Route	**Replicate**	**Lesions**	**Virus isolation**	**Seroconversion***	**Lesions**	**Virus isolation**	**Seroconversion***
ID snout	1	Yes	Yes (N,T)	Yes (≥ 256)	0	3 (N,T)	3 (≥ 256)
	2	Yes	Yes (N,T)	Yes (≥ 256)	0	3 (N,T)	3 (≥ 256)
Oral scarification	1	Yes	Yes (N,T)	Yes (≥ 256)	0	3 (N,T)	3 (32–≥ 256)
	2	Yes	Yes (N,T)	Yes (64)	0	0	2 (32–≥ 256)
Oral	1	No	Yes (T)	Yes (≥ 256)	0	1(N)	0
	2†	No	Yes (T)	Yes (≥ 256)	0	0	0
	3†	No	Yes (T)	Yes (≥ 256)	0	0	0
	4†	No	Yes (T)	Yes (≥ 256)	0	0	0
ID ear	1	No	No	Yes (128)	0	0	0
	2	No	No	Yes (≥ 256)	0	0	0
	3	No	No	Yes (32)	0	0	0
	4	No	No	Yes (32)	0	0	0

*Seroconversion (titer). †Inoculated pigs housed with contact pigs on postinoculation day 2.
N = Nasal swab specimen. T = Tonsil swab specimen.

Table 2—Clinical, virus isolation, and serologic results for VSV-NJ-infected primary and secondary contact pigs

				Virus isolation tonsil		Virus isolation nasal		
Treatment group	**Pig No.**	**Pen**	**Lesions**	**Maximum titer**	**Duration**	**Maximum titer**	**Duration**	**Seroconversion***
Inoculated	429	A	S,CB	2.6	PID 3-5	4.1	PID 2-6	Yes
	431	A	S,CB	2.9	PID 3-5	< 2.3	PID 2-5	Yes
Primary contact	427	A,B	CB	3.6	PCD 2-7	< 2.3	PCD 1-4	Yes
	439	A,B	CB	3.9	PCD 1-6	< 2.3	PCD 1-4	Yes
	438	A,C	S	4.3	PCD 1-7	< 2.3	PCD 1-2	Yes
	440	A,D	None	4.6	PCD 1-7	< 2.3	PCD 2	Yes
Secondary contact	426	B	None	3.6	PCD 2-5	< 2.3	PCD 1	Yes
	430	B	CB	3.6	PCD 1-5	No titer	NA	Yes
	433	B	CB	3.1	PCD 1-5	No titer	NA	Yes
	428	C	None	No titer	NA	< 2.3	PCD 2	Yes
	435	C	None	3.3	PCD 2,5-8	< 2.3	PCD 2	Yes
	436	C	None	< 2.3	PCD 2	< 2.3	PCD 2	No
	432	D	None	No titer	NA	No titer	NA	No
	434	D	None	No titer	NA	No titer	NA	No
	437	D	None	No titer	NA	No titer	NA	No

Positive = ≥ 32
S = Vesicular lesion on snout. CB = Vesicular lesion on coronary band. PID = Postinoculation day. PCD = Postcontact day. NA = Not applicable.

sisted of new vesicles on the apex of the snout, nasal planum, and lower lip in pig 429 and vesicle formation along the coronary bands and in the interdigital space of the rear feet of pigs 429 and 431. On PID 7, pig 429 developed a new vesicle along the coronary band of 1 front claw. By euthanasia (PID 10), the snout lesions were healing by granulation and affected feet had healing by granulation along the coronary bands and interdigital space and sloughing of heel bulbs. Lesions developed in 3 of 4 primary contacts. Pig 438 developed a large vesicle around a cut just behind the apex of the snout on PCD 5. This vesicle ruptured by PCD 7 and was healing by granulation on the day of euthanasia. Pigs 427 and 439 developed lesions along the coronary bands on PCD 5 and 7, respectively. This was characterized by swelling and scabbing along the coronary bands of 2 feet in pig 427 and vesiculation along the coronary band of 1 claw in pig 439. These lesions were healing and the heel bulbs of affected feet were sloughing by the day of euthanasia. Virus also was isolated from the secondary contacts as early as PCD 1. Two secondary contacts in 1 pen developed foot lesions. Pig 433 developed a vesicle along the coronary band of 1 claw on PID 5, and pig 430 had swelling and cracking along the coronary band of 1 claw on PID 6. These lesions were healing and the heel bulb of the affected claw was sloughing by the day of euthanasia.

Discussion

Contact transmission of VSV-NJ developed only when vesicular lesions were present or subsequently developed in the infected pig. Transmission was observed when these lesions were either associated with the snout, oral cavity, or coronary band. Although it is unknown whether transmission developed as a result of direct contact or indirectly through contact with a contaminated environment, the observed relationship between transmission and vesicular lesions probably relates to the high viral titers, exceeding 10^8 $TCID_{50}$/ml, that have been reported from vesicular fluid from VSV-NJ-infected swine.[15] The observed relationship between vesicular lesions and contact transmission has important implications in the control of this disease in the United States, because current VSV-NJ surveillance measures for domestic livestock are based on vesicular disease detection. Although lesions were associated with all pigs in which contact transmission developed, these lesions, especially when associated with the oral cavity, often were quite subtle. In addition, clinical signs such as excess salivation and anorexia were not apparent and provided no indication that vesicular lesions were present. In pigs, this implies that many pigs that are capable of transmitting VSV-NJ by contact would not be detected on the basis of clinical signs and subsequent examination for vesicular lesions alone. Subclinical VSV-NJ infections are not limited to swine and also have been reported in naturally infected cattle[19] and horses.[20]

Contact with VSV-NJ-infected pigs provided an efficient means of VSV-NJ transmission within the experimental groups used in experiments 1 and 2. In pigs in which vesicular lesions developed on the snout, 12 of 13 contact pigs became infected. When oral lesions and coronary band lesions were detected, 5 of 6 and 7 of 7 contact pigs, respectively, were infected. The route by which these contact pigs became infected is not known, but results are consistent with previous experimental infections in which pigs were infected orally or by application of VSV-NJ to an abrasion or a break in the skin.[16,17] In the former situation, infected pigs did not develop clinical disease, but infection was detected by use of virus isolation from the tonsil, seroconversion, or both. This outcome was observed in all pigs infected by contact in experiment 1. In contrast, VSV-NJ infection was confirmed by the development of clinical disease, virus isolations, and serologic results in many of the pigs infected by contact in experiment 2. This was observed in primary and secondary contacts and is consistent with application of VSV-NJ to a break in the skin or mucosal epithelium. Unlike experiment 1, most of the pigs used in experiment 2 had noticeable skin abrasions and lacerations associated with fighting. This observation is consistent with studies in which development of lesions and contact transmission to domestic swine could be enhanced by abrasions of the skin.[9,10]

Results indicated that transmission occurred rapidly, with contact pigs shedding virus as early as PCD 1. In those contact-infected pigs that developed vesicular lesions (experiment 2), infection was confirmed by use of virus isolation from 4 to 6 days prior to the detection of lesions. In addition, as observed in a previous study,[10] VSV-NJ transmission in experiment 2 often was detected by use of virus isolation in the secondary contact pigs prior to detection of lesions in the primary pigs. This is in contrast to those pigs that were infected by inoculation of the snout or application of the virus to scarified areas of the oral mucosa. In those pigs, detection of virus and the development of lesions coincided. This may indicate that the vesicular lesions that developed on contact-infected pigs developed secondarily to actual infection and may have resulted from autogenous spread to preexisting breaks in skin epithelium.

Although results suggest that VSV-NJ could be maintained in swine solely through contact transmission, the seasonal pattern of VSV-NJ observed on Ossabaw Island, Ga,[12,13] and the western United States,[2,21] suggests that long-term viral maintenance by contact transmission alone is unlikely. Three observations from this study explain why VSV-NJ contact transmission may be self-limiting. First, VSV-NJ may be efficiently transmitted within an individual herd, and it is likely that most of that herd could be infected in a short period of time. In this instance, the susceptible pigs needed for continued transmission would be quickly eliminated. This pattern has been observed in field studies in which high proportions of pigs in infected herds had antibodies against VSV-NJ.[3,19,20] Second, viral shedding from infected pigs observed in this and other experimental studies[15-17] rarely persists for more than 7 days and usually ends with seroconversion. This provides a relatively narrow window for contact transmission to develop, and, to date, there is no evidence of persistent infection in swine or any other domestic animal species. Finally, transmission in

our experiments was lesion-dependent, and the development of lesions appeared to require preexisting skin abrasions. This implies that animal husbandry methods or environmental conditions that promote abrasions or breaks in skin or mucosal surfaces could play an important role in determining the frequency of clinical VS and subsequent contact transmission.

Our results indicate that contact transmission of VSV-NJ alone may not result in long-term viral maintenance in domestic animal populations; however, it may represent the major route of transmission after the virus is introduced into a domestic animal herd. Such transmission, especially if clinical disease develops, also could serve to locally amplify the virus, increasing availability of VSV-NJ to mechanical vectors. Although a viremia has not been reported from VSV-NJ-infected pigs or other livestock species, such amplification also may provide an initial viral source for biological vectors. This could be important in situations in which subsequent viral transmission within the insect population develops via cofeeding. Contact transmission may result in clinical and subclinical infections, and results from our study suggest that development of vesicular lesions is an important risk factor for development of contact transmission. Therefore, it is important to understand why vesicular lesions, which may be detected in only a small proportion of animals, develop. Without an understanding of basic pathogenesis, especially with regard to understanding the potential for viral transmission via contact, mechanical, or biological vectors, it will be difficult to development and implement effective VSV-NJ control strategies. Although pigs have not been involved in recent outbreaks of VSV-NJ in the western United States, they represent an excellent model system to gain this knowledge, and it is hoped that this will greatly minimize costs and time associated with the additional research needed to understanding this disease in horses and cattle.

[a]Sigma chemical Co, St Louis, Mo.

[b]Courtesy of Dr. Robert B. Tesh, Department of Pathology, The University of Texas Medical Branch, Galveston, Tex.

References

1. Schmidtmann ET, Tabachnick WJ, Hunt GL, et al. 1995 epizootic of vesicular stomatitis (New Jersey serotype) in the western United States: an entomologic perspective. *J Med Entomol* 1999;36:1–7.

2. Bridges VE, McCluskey BJ, Salmon MD, et al. Review of the 1995 vesicular stomatitis outbreak in the western United States. *J Am Vet Med Assoc* 1997;211:556–560.

3. Walton TE, Webb PA, Kramer WL, et al. Epizootic vesicular stomatitis in Colorado, 1982: epidemiologic and entomologic studies. *Am J Trop Med Hyg* 1987;36:166–167.

4. Francy DB, Moore CG, Smith GC, et al. Epizootic vesicular stomatitis in Colorado, 1982: isolations of virus from insects collected along the northern Colorado Rocky Mountain front range. *J Med Entomol* 1984;25:343–347.

5. Kramer WL, Jones RH, Holbrook FR, et al. Isolation of arboviruses from *Culicoides* midges (Diptera: Ceratopogonnidae) in Colorado during an epizootic of vesicular stomatitis New Jersey. *J Med Entomol* 1990;25:487–493.

6. Comer JA, Tesh RB, Modi B, et al. Vesicular stomatitis virus New Jersey serotype: replication in and transmission by *Lutzomyia shannoni* (Diptera: Psychodidae). *Am J Trop Med Hyg* 1990;42:483–490.

7. Mead, DG, Mare CJ, and Ramberg FB. Bite transmission of vesicular stomatitis virus (New Jersey Serotype) to laboratory mice by *Simulium vittatum* (Diptera: Simuliidae). *J Med Entomol* 1999; 36:410–413.

8. Ferris DH, Hanson RP, Dicke RJ, et al. Experimental transmission of vesicular stomatitis by Diptera. *J Infect Dis* 1955;96:184–191.

9. Shahan MS, Frank AH, Mott LO. Studies on vesicular stomatitis with special reference to a virus of swine origin. *J Am Vet Med Assoc* 1946;108:5–19.

10. Patterson WC, Jenney EW, Holbrook AA. Experimental infections with vesicular stomatitis in swine I. Transmission by direct contact and feeding infected meat scraps. *US Livestock Sanit Assoc* 1955;59:368–379.

11. Cotton WE. Vesicular stomatitis in its relation to the diagnosis of foot-and-mouth disease. *J Am Vet Med Assoc* 1926;69:313–332.

12. Stallknecht DE, Nettles VF, Fletcher WO, et al. Enzootic vesicular stomatitis New Jersey type in an insular feral swine population. *Am J Epidemiol* 1985;122:876–883.

13. Stallknecht DE, Fletcher WO, Erickson GA, et al. Antibodies to vesicular stomatitis New Jersey type virus in feral and domestic sentinel swine. *Am J Epidemiol* 1987;125:1058–1065.

14. Stallknecht DE, Kavanaugh DM, Corn JL, et al. Feral swine as a potential amplifying host for vesicular stomatitis virus New Jersey serotype on Ossabaw Island, Georgia. *J Wildl Dis* 1993; 29:377–383.

15. Clarke GR, Stallknecht DE, Howerth EW. Experimental infection of swine with a sandfly (*Lutzomyia shannoni*) isolate of vesicular stomatitis virus New Jersey serotype. *J Vet Diagn Invest* 1996;8:105–108.

16. Howerth EW, Stallknecht DE, Dorminy M, et al. Experimental vesicular stomatitis in swine: effects of route of inoculation and steroid treatment. *J Vet Diagn Invest* 1997;9:136–142.

17. Stallknecht DE, Howerth EW, Reeves CL, et al. Potential for contact and mechanical vector transmission of vesicular stomatitis virus New Jersey in pigs. *Am J Vet Res* 1999;60:43–48.

18. Sokal RR, Rohlf FJ. Analysis of frequencies. In: *Biometry*. New York: WH Freeman & Co, 1981;731–746.

19. Webb PA, Monath TP, Reif JS, et al. Epizootic vesicular stomatitis in Colorado, 1982: epidemiologic studies along the northern Colorado front range. *Am J Trop Med Hyg* 1987;36:183–188.

20. Mumford EL, McCluskey BJ, Traub-Dargatz JL, et al. Serologic evaluation of vesicular stomatitis virus exposure in horses and cattle in 1996. *J Am Vet Med Assoc* 1998;213:1265–1269.

21. McCluskey BJ, Hurd HS, Mumford EL. Review of the 1997 outbreak of vesicular stomatitis in the western United States. *J Am Vet Med Assoc* 1999;215:1259–1262.