



**PATHOGEN EXPOSURE IN FERAL SWINE  
POPULATIONS GEOGRAPHICALLY ASSOCIATED WITH  
HIGH DENSITIES OF TRANSITIONAL SWINE PREMISES  
AND COMMERCIAL SWINE PRODUCTION**

Authors: Corn, Joseph L., Cumbee, James C., Barfoot, Rendall, and Erickson, Gene A.

Source: Journal of Wildlife Diseases, 45(3) : 713-721

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-45.3.713>

---

BioOne Complete ([complete.BioOne.org](https://complete.BioOne.org)) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at [www.bioone.org/terms-of-use](https://www.bioone.org/terms-of-use).

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

---

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

# PATHOGEN EXPOSURE IN FERAL SWINE POPULATIONS GEOGRAPHICALLY ASSOCIATED WITH HIGH DENSITIES OF TRANSITIONAL SWINE PREMISES AND COMMERCIAL SWINE PRODUCTION

Joseph L. Corn,<sup>1,4</sup> James C. Cumbee,<sup>1</sup> Rendall Barfoot,<sup>2</sup> and Gene A. Erickson<sup>3</sup>

<sup>1</sup> Southeastern Cooperative Wildlife Disease Study, Department of Population Health, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602, USA

<sup>2</sup> Veterinary Services, Animal and Plant Health Inspection Service, United States Department of Agriculture, 9600 Two Notch Road, Suite 10, Columbia, South Carolina 29223, USA

<sup>3</sup> Rollins Animal Disease Diagnostic Laboratory, North Carolina Department of Agriculture and Consumer Services, 2101 Blue Ridge Road, Raleigh, North Carolina 27607, USA

<sup>4</sup> Corresponding author (email: jcorn@uga.edu)

**ABSTRACT:** Surveys for evidence of exposure to pseudorabies virus (PRV), *Brucella suis*, swine influenza virus (SIV; human-like H1N1, reassortant type H1N1, H1N2-like H1N1 and H3N2), porcine circovirus 2 (PCV 2), and porcine respiratory and reproductive syndrome virus (PRRSV) in feral swine (*Sus scrofa*) were conducted in areas where feral swine were geographically associated with high densities of transitional swine premises in South Carolina and high densities of commercial swine production in North Carolina. In South Carolina, 10/50 (20.0%), 7/50 (14.0%), and 29/49 (59.2%) feral swine tested antibody positive for PRV, *B. suis*, and PCV-2, respectively. Antibodies to PRRSV (0/49) and SIV (0/49) were not detected. In North Carolina, antibodies to PRV and *B. suis* were not detected in serum samples from 120 feral swine; however, antibodies to PRRSV (1/120 [0.8%]), PCV-2 (86/120 [71.7%]); these included 80 positives plus six suspects), and SIV (108/119 [90.7%]) were present. The presence of PRV and *B. suis* in South Carolina may have been due to the introduction of infected feral swine into the area or to a previous association of feral swine with infected transitional swine. Their absence in the North Carolina populations may have been due to the absence of these disease agents in the feral swine originally introduced into the area and the lack of a potential for contact with infected commercial swine. Feral swine associated with commercial swine in North Carolina may have been exposed to SIV subtypes circulating in commercial swine via airborne spread of SIV from commercial swine facilities. Feral swine seropositive for PCV-2 were prevalent in both states, which may indicate efficient transmission from commercial swine and transitional swine, or that PCV-2 is widespread in feral swine. The low prevalence of animals with antibodies against PRRSV may indicate a less-than-efficient means of transmission from commercial to feral swine. Additional epidemiologic studies are needed to understand the risks and mechanisms of transmission of disease agents among commercial, transitional, and feral swine, and the role of feral swine as reservoirs of these disease agents.

**Key words:** *Brucella suis*, domestic swine, feral swine, porcine circovirus-2, porcine respiratory and reproductive syndrome, pseudorabies, swine influenza.

## INTRODUCTION

The Pseudorabies Eradication Program Standards (US Department of Agriculture, 2003) define swine (*Sus scrofa*) in three categories: commercial, transitional, or feral. Commercial swine are those swine that are continuously managed and have adequate facilities and practices to prevent exposure to either transitional or feral swine. Transitional swine are those feral swine that are captive or swine that have reasonable opportunities to be exposed to

feral swine. Feral or wild swine are those swine that are free ranging. Feral swine serve as a reservoir for disease agents that affect commercial and transitional swine, including pseudorabies virus (PRV) and *Brucella suis*. These pathogens have been detected in feral swine populations throughout their range in the United States (Nettles and Erickson, 1984; Corn et al., 1986; van der Leek et al., 1993) and feral swine populations have been shown to maintain PRV over time (Corn et al., 2004). Feral swine may also play impor-

tant roles in the epidemiology of other swine disease agents that affect commercial swine in the United States, including swine influenza (SIV), porcine circovirus 2 (PCV-2), and porcine respiratory and reproductive syndrome (PRRSV), as well as foreign animal diseases such as classical swine fever and foot-and-mouth disease. However, data are lacking on the prevalence of these agents in feral swine populations. Antibodies against SIV H1N1 were reported in 13/117 feral swine in Oklahoma (Saliki et al., 1998), 15/20 feral swine in Kansas (Gipson et al., 1999), 11/387 feral swine in Texas (Hall et al., 2008), and in 4% of 78 European wild boar in Spain (Vicente et al., 2002). Antibodies against SIV H3N2 were found in 64/387 feral swine in Texas, 5/94 in California, and 1/99 in Mississippi (Hall et al., 2008). Porcine circovirus 2 was isolated from Eurasian wild boar raised in free-range conditions on pasture in western Canada during an outbreak of multisystemic disease (Ellis et al., 2003). Antibodies against PRRSV were found in 2/117 feral swine in Oklahoma (Saliki et al., 1998), 2/659 European wild boar in Germany (Oslage et al., 1994), and in 33/909 European wild boar in France (Albina et al., 2000). Additionally, Bonilauri et al. (2006) reported positive PCR results for PRRSV from a road-killed wild boar in Italy.

Risks for transmission of disease agents among commercial, transitional, and feral swine are not presently understood, and this greatly limits the ability to devise and evaluate prevention and control strategies. Disease agents may be transmitted at various interfaces, including direct contact (Hahn et al., 1997), via contamination of food, water, or fomites (Pritchard et al., 2005), and by aerosols (Gillespie and Hill, 1996; Albina, 1997). The objectives of this study were 1) to identify feral swine populations in geographic association with areas of high density commercial swine production in North Carolina, 2) to identify feral swine populations in areas

with an abundance of transitional swine premises in South Carolina, and 3) to determine if PRV, *B. suis*, SIV, PRRSV, and PCV-2 were present in feral swine associated with either high-density commercial swine production or transitional swine premises.

## MATERIALS AND METHODS

### Feral swine distribution

The Southeastern Cooperative Wildlife Disease Study (SCWDS) produced national feral swine distribution maps in 1982, 1988, and 2004. These maps were prepared with the use of data provided by state wildlife management agencies in the United States. Each individual state provided data on the distribution of established feral swine populations in their respective state for the given year, and these data were then collated into a national map (SCWDS, 2004; Corn et al., 2005).

### Targeting feral swine associated with high-density commercial swine production in North Carolina

In 2003, SCWDS developed Geographic Information System (GIS)-based maps for prioritization of surveillance for PRV and *B. suis* in feral swine in Georgia. These maps identified counties where commercial swine occurred in Georgia at that time, and provided a ranking of counties based on the relative abundance of commercial swine (George et al., 2003). Using the formula developed for Georgia, relative abundance maps for commercial swine were prepared for 28 states where feral swine were reported during 2004. Counties that reported large numbers of high-output commercial swine farms ranked the highest. Eight of the 10 highest-ranked counties were in North Carolina (Corn et al., 2005). To assess the risk of transmission of disease agents between commercial and feral swine we combined the North Carolina commercial swine relative abundance map with the 2004 North Carolina feral swine distribution map. The North Carolina map combines relative abundance rankings for commercial swine for every county within the distribution of feral swine from 2004 (Fig. 1).

The commercial swine relative abundance/feral swine distribution map for North Carolina was used to select counties for surveys for selected disease agents in feral swine. With the use of this map we determined the 15 counties with the highest ranks for commercial

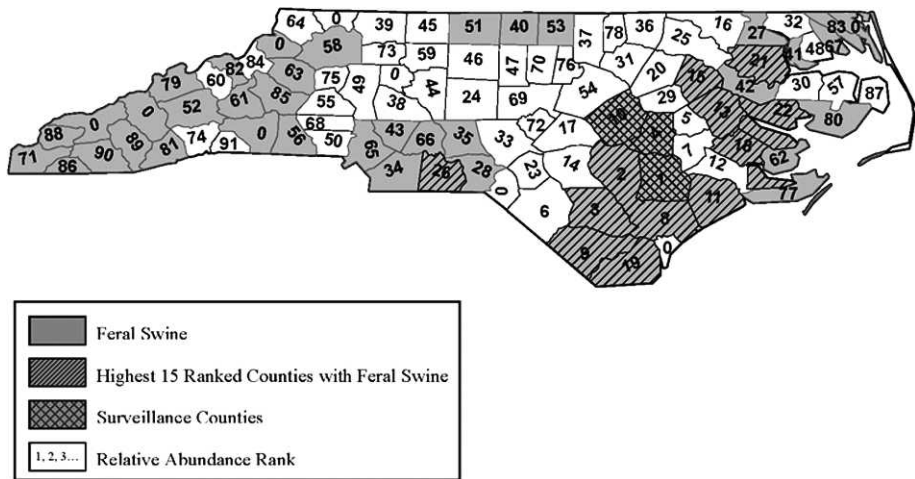


FIGURE 1. Relative abundance of domestic swine production and the distribution of feral swine in North Carolina.

swine abundance that also included reports of populations of feral swine (in order of most number of farms to least: Duplin, Sampson, Bladen, Wayne, Pender, Columbus, Johnston, Onslow, Pitt, Edgecombe, Craven, Brunswick, Bertie, Beaufort, and Anson). According to the census data these 15 counties included 1,388 farms that produced 7,228,969 commercial swine during 2002 (USDA, 2002). During the 2006–2007 trapping season we conducted surveys in Duplin, Johnston, and Wayne counties. These counties were selected because they ranked high in our relative abundance calculations for high densities of commercial swine farms and had relatively large, viable, and accessible feral swine populations. Census data for these three counties documented a combined presence of 583 farms that produced 2,920,451 commercial swine (USDA, 2002) in an area of 5,624 sq km for an average of 0.1 swine farms/sq km and 519.3 domestic swine/sq km.

#### Targeting feral swine associated with transitional swine production in South Carolina

Counties with large numbers of low-output farms were considered to be counties where transitional swine premises might be common. A map of potential transitional swine premises in South Carolina was developed with the use of a combination of county-level inventory and swine farm data from the 2002 Census of Agriculture (USDA, 2002), additional field observations, and data collected from livestock sales and other sources. Confirmation of the status of these premises was via observations at the premises and objective determinations as

to the potential for direct contact between feral and transitional swine at each premises. We investigated 593 potential transitional swine farms in South Carolina and classified 306 farms as active transitional swine farms. These data were combined in the GIS and used to map the distribution of transitional swine premises in South Carolina (Fig. 2).

To assess the association of transitional and feral swine we combined the South Carolina transitional swine map with the 2004 South Carolina feral swine distribution map (Fig. 2). Based on a clustering of transitional swine farms near the Congaree and Wateree river drainages, and the relatively large geographic distribution of feral swine in this area, we selected the Congaree and Wateree river drainages, including the Congaree National Park, as the survey site for feral swine in South Carolina. These river drainages are located in Calhoun, Richland, and Sumter counties. In these three counties we identified 122 potential transitional swine farms; 70 of these showed evidence of active transitional swine farms during this survey. Census data documented that these three counties included 75 farms that produced 7,286 domestic swine (USDA, 2002) in an area of 4,781 sq km for an average of 0.02 swine farms/sq km and 1.5 domestic swine/sq km.

#### Surveys

Surveys were conducted in North Carolina in Duplin, Wayne, and Johnston counties from January to April in 2006 and 2007. Surveys in South Carolina were conducted in Calhoun, Richland, and Sumter counties from January

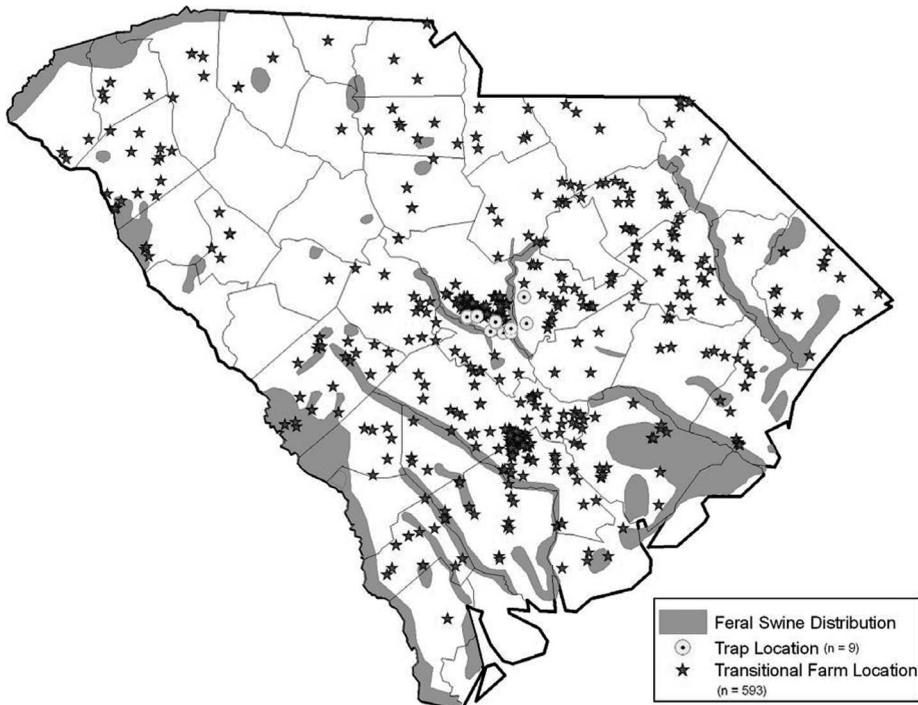


FIGURE 2. Distribution of transitional swine premises and feral swine in South Carolina.

to September 2006. Feral swine were trapped with walk-in drop door traps, including  $1.3 \times 2 \times 1$ -m box-style traps and  $6 \times 6 \times 2$ -m corral-type traps. All traps were baited with soured corn and used a root stick to trigger the door. We also collected samples via night hunting on private properties that had large bait piles for wildlife or agricultural fields that were being damaged by feral swine. Some of the properties in North Carolina, particularly in Wayne and Duplin counties, had commercial swine operations on site. At two of these sites we found evidence of rooting/foraging and successfully trapped feral swine within 100 m of the commercial swine facilities. All trapping sites in South Carolina were located within 5 km of the large cluster of transitional swine farms identified in the transitional swine mapping project (Fig. 2).

During the trapping seasons of 2006 and 2007 we collected samples from 120 feral swine in North Carolina and 50 feral swine in South Carolina (Table 1). Field necropsies were conducted on all feral swine collected. Whole blood was harvested directly from the heart. Serum (5 ml) was collected from the whole blood by centrifugation and frozen on dry ice in the field until being stored in a freezer at  $-20$  C. Sera from South Carolina

were tested for *B. suis* at the Athens Diagnostic Laboratory (Athens, Georgia, USA) with the card test, and positives were confirmed by the Georgia State–Federal Brucellosis Laboratory (Atlanta, Georgia, USA) via the brucellosis card test, buffered acidified plate antigen (BAPA) test, and the rivanol test. Sera from North Carolina were tested for *B. suis* at the Rollins Animal Disease Diagnostic Laboratory (RADDL; Raleigh, North Carolina, USA) with the BAPA test. All other serologic tests were conducted at the RADDL. Samples were evaluated for PRV by latex agglutination with the use of the PRV gp1 Antibody Test Kit (IDEXX Laboratories, Inc., Westbrook, Maine, USA). Samples were evaluated for PCV-2 via ELISA with the use of the SERELISA PCV2 Ab Mono Blocking Kit (Synbiotics Europe, Lyon, France). Samples were evaluated for PRRSV via ELISA with the use of the PRRSV Antibody Test Kit (IDEXX Laboratories.). Samples were evaluated for four types of swine influenza (human-like H1N1, reassortant type H1N1, H1N2-like H1N1 and H3N2) via hemagglutination inhibition (HI). The viruses used for the SIV HI tests were Hu-H1N1 SIV, A/Sw/NC/8912-2005; rH1N1 SIV, A/Sw/NC/36883-2000; H1N2-like H1N1 SIV, A/Sw/NC/3422-2006;



TABLE 1. Survey for selected disease agents in feral swine geographically associated with high densities of transitional swine premises in South Carolina and high densities of commercial swine production in North Carolina.<sup>a</sup>

County	<i>Brucella suis</i>	PRV	PCV-2	PRRS	Hu H1N1	rH1N1	H1N1	H3N2
Duplin <sup>b</sup>	0/50	0/50	48/50 (96%)	1/54 (2%)	31/50 (64%)	4/50 (8%)	9/50 (18%)	28/50 (56%)
Wayne <sup>b</sup>	0/25	0/25	11/25 (44%)	0/25	16/25 (64%)	1/25 (4%)	7/25 (28%)	10/25 (40%)
Johnston <sup>b</sup>	0/45	0/45	27/45 (60%)	0/41	40/44 (91%)	3/40 (8%)	1/44 (2%)	18/44 (41%)
Total <sup>b</sup>	0/120	0/120	86/120 (72%)	1/120 (1%)	87/119 (73%)	8/119 (7%)	17/119 (14%)	56/119 (47%)
Richland <sup>c</sup>	4/40 (10%)	8/40 (20%)	24/40 (60%)	0/40	0/40	0/40	0/40	0/40
Sumter <sup>c</sup>	3/8 (38%)	1/8 (20%)	4/8 (50%)	0/8	0/8	0/8	0/8	0/8
Calhoun <sup>c</sup>	0/2	1/2 (50%)	1/1 (100%)	0/1	0/1	0/1	0/1	0/1
Total <sup>c</sup>	7/50 (14%)	10/50 (20%)	29/49 (59%)	0/49	0/49	0/49	0/49	0/49

<sup>a</sup> PRV = pseudorabies virus, PCV-2 = porcine circovirus 2, PRRS = porcine respiratory and reproductive syndrome virus, Hu H1N1 = human-like H1N1, rH1N1 = reassortant type H1N1, H3N2 = an H3N2-like H1N1.

<sup>b</sup> North Carolina counties.

<sup>c</sup> South Carolina counties.

and H3N2, A/Sw/NC/24277-2005. This procedure uses receptor-destroying enzyme treatment of swine sera for testing to assure specificity of antibody detection (Yoon et al., 2004).

**RESULTS**

**Feral swine associated with high-density commercial swine production in North Carolina**

Antibodies to PRV and *B. suis* were not detected in sera from any of the 120 samples collected in North Carolina (Table 1). However, one animal was antibody positive for PRRSV (1/120, 0.8%), and 86/120 (71.7%; 80 positives plus six suspects close to the positive range) were seropositive for PCV-2. Of the 119 feral swine tested for SIV, 108 (90.7%) tested seropositive for at least one of the four subtypes. Fifty-two (43.7%) feral swine were seropositive for only one subtype, 52 (43.7%) were seropositive for two subtypes, and four (3.4%) were seropositive for three subtypes (Table 1).

**Feral swine associated with transitional swine in South Carolina**

Sera from 10/50 (20.0%) feral swine were positive for antibodies against PRV and 7/50 (14.0%) were confirmed seropositive for *B. suis* by the State–Federal Brucellosis Laboratory in Atlanta, Georgia, USA. One additional feral swine was declared suspect for *B. suis* by the Athens Diagnostic Laboratory; however, there was insufficient serum for further testing. Sera from 29/49 (59.2%) feral swine were positive for antibodies against PCV-2 and sera from six additional animals were suspicious. Evidence of exposure to PRRS and SIV was not found in any of 49 samples tested from South Carolina (Table 1).

**DISCUSSION**

The feral swine populations included in our surveys in North Carolina and South Carolina differed in their associations with commercial swine and transitional swine

and in the evidence we documented of previous exposure to SIV, PRV, and *B. suis*. The presence of SIV (human-like H1N1, reassortant type H1N1, H1N2-like H1N1 and H3N2) in feral swine in the North Carolina populations versus their absence in the South Carolina population may simply be a result of these viruses being present in the North Carolina populations; however, it may also be related to the geographical association of these feral swine in North Carolina with areas of high-density commercial swine production. H1N2-like H1N1 and human-like H1N1 are the dominant H1N1 subtypes in domestic swine in North Carolina (Klimov, pers. comm.) and H3N2 viruses are found in domestic swine throughout the United States (Webby et al., 2000). Swine influenza viruses can be transmitted via direct contact and droplets (Easterday, 1986), and so may be spread from commercial swine to feral swine in the vicinity of the commercial swine barns via the ventilation systems of the barns. Subsequent spread among feral swine could occur where feral swine density allowed for a sufficient contact rate.

In contrast, the presence of PRV and *B. suis* in the South Carolina population may have been related to the association of these feral swine with transitional swine, and/or the length of time this population had been established in the area. Biosecurity at commercial swine farms precludes direct contact between feral and commercial swine, which would eliminate the potential for spread of sexually transmitted diseases. Biosecurity at transitional swine premises is limited or nonexistent and direct contact is possible. Furthermore, feral swine surveyed in North Carolina were from recently established populations reported in the 2004 survey of the distribution of feral swine in the United States (Corn et al., 1995; SCWDS, 2004), but not present in these counties in North Carolina in 1988 (SCWDS, 2004; Corn et al., 2005). Feral swine surveyed in South Carolina were from older popula-

tions that had been reported in the 1984 feral swine distribution survey (SCWDS, 2004; Corn et al., 2005).

The absence of evidence of PRV and *B. suis* in the feral swine populations in North Carolina probably is a result of these isolated and recently established feral swine populations having been established from feral or domestic swine not infected with either of these disease agents. Without an initial or subsequent introduction of either agent into the population, and with no recent domestic source of these agents, these populations have remained free of both PRV and *B. suis*. In contrast, the population in South Carolina was established prior to the eradication programs for PRV and *B. suis*; thus earlier generations of feral swine in this area could have become infected via an original introduction of infected feral swine, through subsequent introductions of infected feral swine, and/or through contact with infected transitional swine. These older populations of feral swine in South Carolina would have had more opportunities for exposure to PRV and *B. suis* as they were present before eradication procedures for these diseases reduced their incidence in domestic swine.

Porcine circovirus 2 and PRRSV are common in commercial swine in North Carolina (Erickson, unpubl. data), but although animals seropositive for PCV-2 were prevalent in feral swine in all of our survey areas, only one animal was seropositive for PRRSV. Close contact may be the primary route of transmission for PRRSV (Albina, 1997) but various routes for transmission have been suggested, including vaccination, semen, airborne transmission, contaminated equipment, contaminated environment, feces, urine, visitors, and vectors (Albina, 1997; Mortensen et al., 2002). Studies on the subject have been contradictory; Wills et al. (1997) found that transmission by direct contact was more efficient than transmission across a space of up to 102 cm (Wills et al. 1997), and Hermann et al. (2005)

reported a median infectious dose ( $ID_{50}$ ) of  $10^{5.3}$  median tissue culture infective doses ( $TCID_{50}$ ) for oral exposure but of  $10^{4.3}$   $TCID_{50}$  for intranasal exposure. In contrast, others have suggested airborne spread (Mortensen et al., 2002; Kristensen et al., 2004) and Cho et al. (2007) demonstrated a difference in rates of airborne transmission with the use of different PRRSV isolates. Data on transmission of PCV-2 are lacking, but PCV-2 probably is spread via the oronasal route, and horizontal transmission is efficient (Segalés et al., 2005). The low prevalence of feral swine with antibodies against PRRS may be due to a difficulty in transmission of the virus from commercial to feral swine. However, the high prevalence of feral swine testing seropositive for PCV-2 may indicate efficient transmission, either from commercial and transitional swine to feral swine or efficient transmission within feral populations. Additional data on transmission risks are needed to understand these associations.

Feral swine are abundant in the southeastern United States, Texas, and California, have become more widely distributed in the United States in recent years, and these increases in distribution have resulted in increased risks for transmission of disease agents between feral swine and commercial and transitional swine (Corn et al., 2005). Furthermore, the association of feral swine with commercial and transitional swine also presents a risk for transmission of foreign animal diseases. If SIV is spread from commercial swine to feral swine via airborne droplets, and airborne spread of SIV is used as a model for airborne spread of foot-and-mouth (FMD) disease, our surveys suggest a substantial risk for airborne spread of FMD from commercial swine to feral swine. Additional epidemiologic studies are needed to understand the risk of transmission of disease agents among commercial, transitional, and feral swine; the role of feral swine as reservoirs of these disease agents; and the mechanisms

by which disease agents are transmitted among feral, commercial, and transitional swine.

#### ACKNOWLEDGMENTS

We gratefully acknowledge the assistance of B. Hamrick, G. Martin, and B. Chandler of SCWDS and C. Betsill and S. Owen of Wildlife Services, Animal and Plant Health Inspection Service, U.S. Department of Agriculture in the collection of feral swine for this survey. We also thank the numerous private landowners and Congaree National Park for allowing access to their properties. Funding for this project was provided through Cooperative Agreements 0591130863CA, 0691130863CA, and 0791130863CA, Veterinary Services, Animal and Plant Health Inspection Service, U.S. Department of Agriculture. Additional funds were provided through sponsorship from the fish and wildlife agencies of Alabama, Arkansas, Florida, Georgia, Kansas, Kentucky, Louisiana, Maryland, Mississippi, Missouri, North Carolina, Puerto Rico, South Carolina, Tennessee, Virginia, and West Virginia; through the Federal Aid to Wildlife Restoration Act (50 Stat. 917) and Grant Agreement 06ERAG0005, Biological Resources Division, U.S. Geological Survey, U.S. Department of the Interior; and through Cooperative Agreements 0596130032CA, 0696130032CA, and 0796130032CA, Veterinary Services, Animal and Plant Health Inspection Service, U.S. Department of Agriculture.

#### LITERATURE CITED

- ALBINA, E. 1997. Epidemiology of porcine reproductive and respiratory syndrome (PRRS): An overview. *Veterinary Microbiology* 55: 309–316.
- , A. MESPLÈDE, G. CHENUT, M. F. LE POTIER, B. BOURBAO, S. LE GAL, AND Y. LEFORBAN. 2000. A serological survey on classical swine fever (CSF) Aujeszky's disease (AD) and porcine reproductive and respiratory syndrome (PRRS) virus infections in French wild boars from 1991 to 1998. *Veterinary Microbiology* 77: 43–57.
- BONILAUDI, P., G. MERIALDI, M. DOTTORI, AND I. BARBIERI. 2006. Presence of PRRSV in wild boar in Italy. *Veterinary Record* 158: 107–108.
- CHO, J. G., J. DEEN, AND S. A. DEE. 2007. Influence of isolate pathogenicity on the aerosol transmission of porcine reproduction and respiratory syndrome virus. *Canadian Journal of Veterinary Research* 71: 23–27.
- CORN, J. L., P. K. SWIDEREK, B. O. BLACKBURN, G. E. 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.
- , D. E. STALLKNECHT, N. M. MECHLIN, M. P. LUTTRELL, AND J. R. FISCHER. 2004. Persistence of pseudorabies virus in feral swine populations. *Journal of Wildlife Diseases* 40: 307–310.
- , J. C. CUMBEE, B. A. CHANDLER, D. E. STALLKNECHT, AND J. R. FISCHER. 2005. Implications of feral swine expansion: Expansion of feral swine in the United States and potential implications for domestic swine. *In Proceedings of the 109th Annual Meeting of the United States Animal Health Association*, 295–297.
- EASTERDAY, B. C. 1986. Swine influenza. *In Diseases of swine*. 6th Edition. A. D. LEMAN, B. STRAW, R. D. GLOCK, W. L. MENGELING, R. H. C. PENNY, AND E. SCHOLL. 1986. Iowa State University Press, Ames, Iowa, pp. 244–255.
- ELLIS, J., M. SPINATO, C. YONG, K. WEST, F. MCNEILLY, B. MEEHAN, S. KENNEDY, E. CLARK, S. KRAKOWKA, AND G. ALLAN. 2003. Porcine circovirus 2-associated disease in Eurasian wild boar. *Journal of Veterinary Diagnostic Investigation* 15: 364–368.
- GEORGE, R. C., J. L. CORN, J. R. FISCHER, AND D. E. STALLKNECHT. 2003. A GIS-based approach to pseudorabies virus surveillance in feral swine. *In Proceedings of the 107th Annual Meeting of the United States Animal Health Association*, pp. 170–177.
- GILLESPIE, R. R., AND M. A. HILL. 1996. Infection of pigs by aerosols of Aujeszky's disease virus and their shedding of the virus. *Research in Veterinary Science* 60: 228–233.
- GIPSON, P. S., J. K. VEATCH, R. S. MATLACK, AND D. P. JONES. 1999. Health status of a recently discovered population of feral swine in Kansas. *Journal of Wildlife Diseases* 35: 624–627.
- HAHN, E. C., G. R. PAGE, P. S. HAHN, K. D. GILLIS, C. ROMERO, J. A. ANNELLI, AND E. P. J. GIBBS. 1997. Mechanisms of transmission of Aujeszky's disease virus originating from feral swine in the USA. *Veterinary Microbiology* 55: 123–130.
- HALL, J. S., R. B. MINNIS, T. A. CAMPBELL, S. BARRAS, R. W. DEYOUNG, K. PABILONIA, M. L. AVERY, H. SULLIVAN, L. CLARK, AND R. G. MCLEAN. 2008. Influenza exposure in United States feral swine populations. *Journal of Wildlife Diseases* 44: 362–368.
- HERMANN, J. R., C. A. MUÑOZ-ZANZI, M. B. ROOF, K. BURKHART, AND J. J. ZIMMERMAN. 2005. Probability of porcine reproductive and respiratory syndrome (PRRS) virus infection as a function of exposure route and dose. *Veterinary Microbiology* 110: 7–16.
- KRISTENSEN, C. S., A. BØTNER, H. TAKAI, J. P. NIELSEN, AND S. E. JORSAL. 2004. Experimental airborne transmission of PRRS virus. *Veterinary Microbiology* 99: 197–202.
- MORTENSEN, S., H. STRYHN, R. SØGAARD, A. BOKLUND, K. D. C. STÅRK, J. CHRISTENSEN, AND P. WILLEBERG. 2002. Risk factors for infection of sow herds with porcine reproductive and respiratory syndrome (PRRS) virus. *Preventive Veterinary Medicine* 53: 83–101.
- NETTLES, V. F., AND G. A. ERICKSON. 1984. Pseudorabies in wild swine. *In Proceedings of the 88th Annual Meeting of the United States Animal Health Association*, pp. 505–506.
- OSLAGE, U., J. DAHLE, T. MÜLLER, M. KRAMER, D. BEIER, AND B. LIESS. 1994. Prävalenz von Antikörpern gegen die viren der Europäischen Schweinepest, der Aujeszky'schen Krankheit und des "porcine reproductive and respiratory syndrome" (PRRS) bei wildschweinen in den Bundesländern Sachsen-Anhalt und Brandenburg. *Deutsche Tierärztliche Wochenschrift* 101: 33–38.
- PRITCHARD, G., I. DENNIS, AND J. WADDILOVE. 2005. Biosecurity: Reducing disease risks to pig breeding herds. *In Practice* 27: 230–237.
- SALIKI, J. T., S. J. RODGERS, AND G. ESKEW. 1998. Serosurvey of selected viral and bacterial diseases in wild swine from Oklahoma. *Journal of Wildlife Diseases* 34: 834–838.
- SEGALÉS, J., G. M. ALLAN, AND M. DOMINGO. 2005. Porcine circovirus diseases. *Animal Health Research Reviews* 6: 119–142.
- SOUTHEASTERN COOPERATIVE WILDLIFE DISEASE STUDY (SCWDS). 2004. Wildlife Distribution Maps. [http://www.uga.edu/scwds/dist\\_maps.htm](http://www.uga.edu/scwds/dist_maps.htm). Accessed 26 March 2009.
- US DEPARTMENT OF AGRICULTURE (USDA). 2003. Pseudorabies eradication state–federal–industry program standards. USDA, APHIS 91–55–071.
- . 2002. Census of Agriculture. USDA, National Agricultural Statistics Service. [http://www.nass.usda.gov/Census\\_of\\_Agriculture/](http://www.nass.usda.gov/Census_of_Agriculture/). Accessed 26 March 2009.
- VAN DER LEEK, M. L., H. N. BECKER, E. C. PIRTLE, P. HUMPHREY, C. L. ADAMS, B. P. ALL, G. A. ERICKSON, R. C. BELDEN, W. B. FRANKENBERGER, AND E. P. J. GIBBS. 1993. Prevalence of pseudorabies (Aujeszky's disease) virus antibodies in feral swine in Florida. *Journal of Wildlife Diseases* 29: 403–409.
- VICENTE, J., L. LEÓN-VIZCAÍNO, C. GORTÁZAR, M. J. CUBERO, M. GONZÁLEZ, AND P. MARTÍN-ATANCE. 2002. Antibodies to selected viral and bacterial pathogens in European wild boars from south-central Spain. *Journal of Wildlife Diseases* 38: 649–654.
- WEBBY, R. J., S. L. SWENSEN, S. L. KRAUSS, P. J. GERRISH, S. M. GOYAL, AND R. G. WEBSTER. 2000. Evolution of swine H3N2 influenza viruses in the United States. *Journal of Virology* 74: 8243–8251.
- WILLS, R. W., J. J. ZIMMERMAN, S. L. SWENSON, K. J. YOON, H. T. HILL, D. S. BUNDY, AND M. J. MCGINLEY. 1997. Transmission of PRRSV by

direct, close, or indirect contact. *Swine Health and Product* 5: 213–218.

YOON, K. J., B. H. JANKE, R. W. SWALLA, AND G. ERICKSON. 2004. Comparison of a commercial H1N1 enzyme-linked immunosorbent assay and hemagglutination inhibition test in detecting

serum antibody against swine influenza viruses. *Journal of Veterinary Diagnostic Investigation* 16: 197–201.

*Received for publication 10 March 2008.*