



FERAL SWINE CONTACT WITH DOMESTIC SWINE: A SEROLOGIC SURVEY AND ASSESSMENT OF POTENTIAL FOR DISEASE TRANSMISSION

Authors: Wyckoff, A. Christy, Henke, Scott E., Campbell, Tyler A., Hewitt, David G., and VerCauteren, Kurt C.

Source: Journal of Wildlife Diseases, 45(2) : 422-429

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-45.2.422>

BioOne Complete (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.

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.

FERAL SWINE CONTACT WITH DOMESTIC SWINE: A SEROLOGIC SURVEY AND ASSESSMENT OF POTENTIAL FOR DISEASE TRANSMISSION

A. Christy Wyckoff,¹ Scott E. Henke,^{1,4} Tyler A. Campbell,² David G. Hewitt,¹ and Kurt C. VerCauteren³

¹ Caesar Kleberg Wildlife Research Institute, Texas A&M University–Kingsville, Kingsville, Texas 78363, USA

² United States Department of Agriculture, Animal and Plant Health Inspection Service, Wildlife Services, National Wildlife Research Center, Texas A&M University–Kingsville, Kingsville, MSG 218, Texas 78363, USA

³ United States Department of Agriculture, Animal and Plant Health Inspection Service, Wildlife Services, National Wildlife Research Center, 4101 LaPort Ave, Fort Collins, Colorado 80521, USA

⁴ Corresponding author (email: scott.henke@tamuk.edu)

ABSTRACT: Feral swine (*Sus scrofa*) are present in 38 of the 50 United States, and their populations continue to expand. Domestic swine are widely regarded as vulnerable to diseases harbored by feral swine. Our objectives were to determine antibody prevalence for selected pathogens in Texas feral swine populations and identify contact events between feral and domestic swine. Overall prevalence of antibodies against brucellosis and pseudorabies virus was 11% and 30%, respectively. Antibodies to porcine reproductive and respiratory disease virus were detected in 3% of feral swine from southern Texas. All samples tested negative for antibodies to classical swine fever virus. To determine the frequency of contact events between feral swine and domestic swine in neighboring facilities, we analyzed movement data from 37 adult feral swine that were trapped ≤ 10 km from domestic swine facilities and equipped with geographic positioning system collars. Seven of the 37 feral swine had contact (relocated within 100 m) with domestic swine. We found that contact between feral swine and domestic swine occurred predominantly at night. Additionally, we analyzed 60 consecutive days of experimental track plots around pens that contained domestic swine and empty control pens, and found greater visitation by feral swine to the domestic swine pens. Our data demonstrate that feral swine have direct contact with domestic swine, which presents opportunity for disease transmission.

Key words: Contact, disease transmission, domestic swine, fence line interaction, feral swine, GPS collar, *Sus scrofa*.

INTRODUCTION

Feral swine (*Sus scrofa*) are considered resident in 38 of the 50 United States (Fogarty, 2007), and their populations appear to be expanding (SCWDS, 2008). As feral swine populations expand, conflicts between feral swine and human and livestock activities increase. In addition to causing damage to agricultural and natural resources, feral swine are reservoirs for pathogens important to the domestic swine industry such as pseudorabies virus (PRV), *Brucella suis*, and porcine reproductive and respiratory syndrome virus (PRRSV).

Swine brucellosis is a zoonotic bacterial infection and is transmitted by oral and venereal routes (Thorne, 2001). Pseudorabies virus is an alphaherpes virus and transmission usually occurs by oral or

venereal contact. The disease appears to be well established in feral populations throughout the USA and can persist in these populations for extended periods of time (Corn et al., 1986, 2004; Gresham et al., 2002). As of June 2007, domestic swine populations in all states in the USA, Puerto Rico, and the US Virgin Islands were reported as pseudorabies-free (USDA/APHIS, 2007a). As of June of 2007 all US domestic swine herds were designated brucellosis-free except in Texas (USDA/APHIS, 2007b). Unfortunately, the successful eradication of these two diseases is threatened by the presence of these same diseases in feral swine populations.

Two additional viruses of concern are PRRSV, a newly emerging disease in domestic swine (Saliki et al., 1998; Neumann et al., 2005) and classical swine fever

virus (CSFV), which has been eradicated from the USA (Nettles et al., 1989). Unlike PRV and swine brucellosis, PRRSV does not appear to be well established in feral swine in the USA. However, feral swine have been found positive for antibodies against PRRSV in Oklahoma (Saliki et al., 1998) and Texas (Lawhorn, 1999). Classical swine fever was eradicated in the USA in 1978 (Davidson and Nettles, 1997) and is not believed to be present in US populations of feral swine (Nettles et al., 1989). However, an enhanced national surveillance program began in 2005 to provide early detection of this and other foreign disease introductions (National Veterinary Services Laboratories, 2005).

A growing concern in the USA is the role feral swine might have in the introduction, or reintroduction, of foreign and eradicated diseases to disease-free domestic swine (Witmer et al., 2003; Corn et al., 2005). Though many of the 100,000 US swine operators have some level of biosecurity (Witmer et al., 2003), a portion of the industry is still in “backyard” or transitional production with show pigs, breeders, and small-scale meat producers (SCWDS, 1992). Such backyard operations are the potential points for disease introduction and prevention. Disease transmission from feral to domestic swine can occur by direct contact between animals, either through fencing, by contaminated fomites, or possibly by aerosol dispersal (Christensen et al., 1993; Kristensen et al., 2004).

Farmers and ranchers report feral swine often come into contact with domestic swine, which causes concern because feral swine are a potential disease reservoir. Feral swine have been implicated in three recent outbreaks of swine brucellosis in domestic swine herds and in a herd of cattle (Feral Swine Subcommittee on Brucellosis and Pseudorabies, 2005). Our objectives were to 1) compare the prevalence of antibodies against PRV, *B. suis*, and PRRSV in feral swine found in proximity to domestic swine in eastern

and southern Texas; 2) test feral swine populations for antibodies to CSFV; 3) ascertain if contact occurs between feral swine and domestic swine; 4) determine the frequency of contact between feral and domestic swine; and 5) assess if male or female feral swine were more likely to visit domestic swine.

MATERIALS AND METHODS

Study sites

We selected study areas in southern and eastern Texas with recent feral swine activity. Trapping locations were ≤ 10 km from domestic swine facilities (4-H, Future Farmers of America, and breeder and feeder pigs) with the expectation that this distance was within feral swine movement capabilities (Wood and Brenneman, 1980; Caley, 1997). Sites in eastern Texas included Wildlife Management Areas in Anderson County (Texas Parks and Wildlife Department: Gus Engeling Wildlife Management Area [WMA; 4,434 ha; N31°56', W95°53'] and the Big Lake Bottom WMA [3,076 ha; N31°44', W95°48']) and industrial timberland in Angelina County (N31°10', W94°43'). Sites in southern Texas included La Copita Research Area (Texas A&M University; N27°46', W98°09') in Nueces County, private lands and the Texas A&M University-Kingsville (TAMUK) farm facility (N27°34', W97°50') in Kleberg and Kennedy counties.

Animal capture

We trapped, collected sera from, and applied global positioning system (GPS) collars to feral swine from May 2004 to June 2006. We trapped feral swine using box- and corral-style traps, according to the specifications of Wyckoff et al. (2005). We avoided trapping from June to September to prevent occurrences of heat stress and because of previous low trapping success during these months (Wyckoff et al., 2006).

We sedated trapped feral swine by using an air-pump projector (Pneu-Dart Inc., Williamsport, Pennsylvania, USA) to deliver a 3-ml dart containing a 2:1 mix of Telazol (3.23 mg/kg) and xylazine (1.63 mg/kg) (Sweitzer et al., 1996). If feral swine displayed symptoms of heat stress we administered cool-water enemas to lower body temperatures. We estimated age by tooth wear and eruption patterns (Matschke, 1967), and classified feral swine into two age categories: young (< 9 mo) and adult (≥ 9 mo). Feral swine with a neck

circumference >62.5 cm were fitted with very high frequency/GPS data-logging collars (Televilt Co., Lindsberg, Sweden). We collected blood samples for serology testing on all captured feral swine. If animals were later recaptured, we collected another blood sample before release. All captive and handling procedures were approved by the Texas A&M University–Kingsville Animal Care and Use Committee (IACUC permit 1-04-36) and the National Wildlife Research Center Institutional Animal Care and Use Committee (QA 1240).

Serology

Seerologic testing of antibodies against *Brucella* spp. and PRV was done by the Texas Animal Health Diagnostic Laboratory (Austin, Texas, USA). For *Brucella*, a rapid card test and particle concentration fluorescence immunoassays were used. Tests are not specific for *B. suis* and antibodies to *Brucella abortus* and *Brucella menitensis* (but not *Brucella ovis*) will be detected. An enzyme-linked immunosorbent assay (ELISA) and a serum neutralization test were performed for PRV antibody testing. Testing for antibodies to PRRSV was done at the Texas Veterinary Medical Diagnostic Laboratory (College Station, Texas, USA) using ELISA. Testing for antibodies against CSFV was conducted at Plum Island, New York, through the Foreign Animal Disease Diagnostic Laboratory where ELISA and immunoperoxidase tests were performed.

We tested for antibody prevalence differences between geographic regions (eastern and southern Texas) and sexes using chi-squared analysis with Yates correction (Steel and Torrie, 1980). Duplicate blood samples from recaptured animals were assessed for seroconversion. If recaptured animals tested positive at any time, we considered them as a single positive for these analyses. We considered tests significant at $\alpha=0.05$.

Feral-domestic swine contact

We programmed GPS collars to collect and store one data point every 4 hr, 4 days/wk, for 2.1 yr (Wyckoff et al., 2007). Collars from hunter-harvested feral swine and dead feral swine, or collars that slipped off live animals, were retrieved and the stored location data were imported as shape files into ArcView 9.1 (ESRI, Redlands, California, USA). Selected collars were placed in known locations under various vegetation canopy types within our study area for 1 wk to verify location accuracy of collars. We did not include animals that died within 48 hr of trapping in our analysis.

Domestic swine facilities ≤ 10 km from our

trapping sites were identified with help of local 4-H chapters and domestic swine breeders, located, and mapped. We identified 33 and 11 domestic swine facilities ≤ 10 km of our traps in eastern and southern Texas, respectively. Locations of feral swine and domestic swine facilities were overlaid onto Landsat images (digital orthophoto quarter quadrangle). Because the collar location data points represented only a small fraction of the actual animal movements, we used ArcView to delineate two contact zones. The first was a 100-m buffer around all domestic swine facilities designated as contact zone A. Feral swine relocated in contact zone A were considered to have had a high likelihood of interacting with domestic swine.

Contact zone B was a 500-m buffer overlaid around domestic swine facilities to identify feral swine in proximity to domestic swine facilities. Locations that fell >100 m and <500 m were considered contact events within contact zone B. Animals relocated within contact zone B were assumed to have access to domestic swine facilities and were identified as potential contacts.

Location points were categorized into day and night data points using sunrise and sunset as day-to-night delineators (United States Naval Observatory, 2006). Day and night data point shape files were overlaid on 100-m and 500-m buffer shape files to identify overlap. We used chi-squared analysis to test for differences in the number of contact events between males and females, and between night use and day use within contact zones A and B. We considered tests significant at $\alpha=0.05$.

Contact stimuli

To determine stimuli for attracting feral swine to domestic swine facilities (presence of domestic swine or presence of food) and rate of contact we selected three sites in southern Texas ≥ 5 km from each other. At each site we placed two 3.5×3.5 -m pens; one was a treatment pen that contained a mature domestic female pig provided with food and water daily, and the other a control pen that received equivalent amounts of food and water daily. Pens were paired at each site and were 250 m apart. A 1-m wide track plot was disked around the perimeter of each pen and raked daily. Each morning track plots were checked for feral swine tracks. If evidence of feral swine contact existed, it was counted as one contact event. Two motion-sensing digital cameras (Non Typical, Inc., Park Falls, Wisconsin, USA) were placed at each pen to verify species contact. The experiment was

TABLE 1. Prevalence of antibodies against swine pathogens in Texas feral swine populations in proximity to transitional domestic swine herds.

Pathogen ^a	Sex	Region					
		Eastern			Southern		
		Sera tested	Positive		Sera tested	Positive	
No.	%		No.	%			
PRV	M	68	12	18%	123	39	31.7%
	F	57	10	18%	121	48	39.7%
<i>Brucella</i> spp	M	68	17	25.0%	122	9	7.4%
	F	57	13	23%	121	2	1.7%
PRRSV	M	27	0	0.0%	40	1	3%
	F	33	0	0.0%	37	1	3%
CSFV	M	68	0	0.0%	123	0	0.0%
	F	57	0	0.0%	121	0	0.0%

^a PRV = pseudorabies virus; PRRSV = porcine reproductive and respiratory disease virus; CSFV = classical swine fever virus.

conducted for 60 days from April to May 2006. We used a paired *t*-test to compare visitation to the treatment pen to the control food pen. We considered the test significant at $\alpha=0.05$.

RESULTS

Serology

We collected serum samples from 373 feral swine from eastern ($n=127$) and southern ($n=246$) Texas during 2004–06 (Table 1). We found overall antibody prevalence rates for *Brucella* spp. and PRV of 11% and 30%, respectively. *Brucella* antibody prevalence rates for southern Texas (5%) were lower ($\chi^2_1 = 30.88, P<0.001$) than in eastern Texas (24%). Pseudorabies virus antibody prevalence rates were lower in eastern Texas (18%) than in southern Texas (36%) ($\chi^2_1 = 12.32, P<0.001$). Feral swine in

southern Texas were 7.2 times more likely to have been exposed to PRV than to *Brucella*, whereas feral swine from eastern Texas were 1.3 times more likely to have been exposed to *Brucella* than to PRV.

We found no differences between sexes ($\chi^2_1 = 0.083, P>0.25$) in *Brucella* antibody prevalence in eastern Texas. However, *Brucella* antibody prevalence was greater in males than in females ($\chi^2_1 = 4.6, P<0.05$) in southern Texas (Table 1). We found no differences between sexes (both $\chi^2_1 \leq 1.69, P>0.2$) in PRV antibody prevalence in eastern or southern Texas.

We collected multiple serum samples ($n=30$) from 19 recaptured feral swine (Table 2). Time between recapture events ranged from 1 wk to 10 mo. Of the 19 feral swine sampled on multiple occasions, we found the status of disease antibody

TABLE 2. Serum samples ($n=30$) collected from previously sampled feral swine ($n=19$) from southern and eastern Texas during 2004–05. Seven of the nine seroconversions were juveniles ($n=10$) when they were first tested.

Region	<i>Brucella</i>				Pseudorabies virus			
	Remained		Changed		Remained		Changed	
	+	-	+	-	+	-	+	-
Southern Texas	1	13	1	0	6	2	5	2
Eastern Texas	2	1	0	1	0	2	2	0
All regions	3	14	1	1	6	4	7	2

TABLE 3. Contact of individual collared feral swine within contact zones A and B (100-m and 500-m buffer) of domestic swine at the Texas A&M University–Kingsville farm during 2004–06.

Sex	Days recorded ^a	Events		% of locations ^c	Total no. data points ^d
		100 m (D/N) ^b	500 m (D/N)		
F	7	1 (0/1)	6 (1/5)	31.6	19
F	21	7 (0/7)	13 (2/11)	35.1	37
M	89	15 (0/15)	53 (0/53)	40.2	132
F	120	26 (0/26)	71 (1/70)	23.1	307
M	180	4 (0/4)	20 (0/20)	6.9	289
F	370	42 (0/42)	169 (12/157)	20.0	846
F	488	33 (1/32)	163 (7/156)	13.5	1209

^a Number of days over which collars logged locations.

^b D/N = logged data points from day versus night.

^c Percentage of total locations within 500 meters of domestic swine.

^d Total number of locations collected on individual animals.

prevalence changed for seven animals from southern Texas and two animals from eastern Texas (Table 2). Seven of the nine seroconversions were in feral swine that were <9 mo old when they were first captured. One feral swine animal that was initially negative for *Brucella* antibodies later seroconverted and then on its fourth sampling was found to be negative.

We collected 142 serum samples from 137 feral swine for PRRSV antibody determination; two animals (3%) from southern Texas ($n=77$) tested positive, albeit titers were low (sample-to-positive [S/P]=0.411 and 0.466, positive at S/P \geq 0.4). No animals ($n=60$) were found positive for PRRSV antibodies in eastern Texas. We submitted 373 serum samples for CSFV antibody determination. All samples tested negative.

Feral-domestic swine contact

Seventy-nine individual feral swine were collared between May 2004 and December 2005. Of the 79 individuals collared, usable location data (i.e., \geq 1 wk of data) were recovered from 35 animals (Wyckoff et al., 2007). Locational data were collected from 16 individuals in southern Texas and 19 individuals in eastern Texas. Vegetation canopy cover did not affect location accuracy of collars.

Contact events in zone A occurred only at the TAMUK farm facility in southern Texas. The sex ratio of collared feral swine in proximity to this facility was four males to five females. Seven animals, five females and two males, were recorded within contact zone A 128 times (5% of their total locations; Table 3). We pooled the male and female interaction events and found a preference for nighttime activity ($\chi^2_1 = 73.68$, $P < 0.001$) with 99% of contact events within contact zone A occurring at night.

We found activity in contact zone B only at the TAMUK farm facility, involving the seven aforementioned feral swine (Table 3). Most locations in contact zone B were collected at night (63%) versus day (37%) for these seven animals. Overall, the seven feral swine that entered contact zone B utilized the area at night ($\chi^2_1 = 42.01$, $P < 0.001$). Distances between the trapping locations and domestic swine facilities for the seven animals relocated in contact zones A and B ranged from 50 m to 886 m. For feral swine at this study site that were collared for more than 80 days, the home range major axis for an individual averaged 7.9 km, with a range of 5.9 km to 11.3 km.

Contact stimuli

We found all three sites had contact events by feral swine at both control pens

and treatment pens. Of the 180 pen-days, pens containing domestic female swine had contact events with feral swine on 74 (41%) of the pen-days while the control pens had contact events with feral swine on 10 (6%) of the pen-days. We found that pens containing domestic female swine tended toward having more contact events ($t_1=3.46$, $P<0.07$) than control pens. Remotely captured digital images suggest that feral swine made physical contact through the fence and attempted to enter pens containing domestic female swine.

DISCUSSION

Prevalence rates for PRV antibodies in eastern Texas (18%) and southern Texas (36%) are comparable with other reports from Texas (Nettles and Erickson, 1984; Corn et al., 1986; Lawhorn, 1999). Our prevalence rates of antibodies against *Brucella* in eastern (24%) and southern (5%) Texas are also comparable to previous reports (Conger et al., 1999; Lawhorn 1999). Prevalence rates can vary by region, sample size, age category, and possibly the natural history of the population. Hahn et al. (1999) compared polymerase chain reaction analysis of viral PRV DNA in feral swine tissues to antibody detection results using ELISA and found that ELISA testing may underestimate antibody prevalence by 50%. This suggests that our estimates of disease exposure may be an underestimate of actual levels within populations.

Recaptures of individual swine provided a unique opportunity to determine if disease status changed within individuals over time. The seroconversion events for PRV and *Brucella* could be the result of a formerly latent infection (PRV, Hahn et al., 1999) or a new exposure to the pathogen. Our data demonstrate that the risk of exposure to these diseases increases with age.

We recorded the third occurrence of exposure to PRRSV within feral swine in the USA. Previously, low antibody preva-

lence rates were found in Oklahoma (Saliki et al., 1998) and Texas (Lawhorn, 1999). The possible emergence of PRRSV in feral swine populations could eventually pose a similar threat to disease-free domestic swine as PRV and brucellosis. Our failure to detect antibodies to CSFV within feral swine populations was expected, and supports the findings of Nettles et al. (1989).

The contact events of the seven feral swine within contact zone A demonstrates the threat feral swine present to domestic livestock. Of the seven feral swine relocated in contact zone A, four were positive for antibodies against PRV or *Brucella*. The known presence of PRV and *Brucella* antibody-positive animals in proximity to domestic swine is cause for concern. The possibility of aerosol transmission further complicates disease prevention for backyard and transitional domestic swine (Christensen et al., 1993; Kristensen et al., 2004). The higher ratio of contact events by females to males is interesting and contrary to expectation. This may have behavioral basis or could be an artifact of sample size.

The contact stimuli experiment, though not statistically significant, may be biologically significant. It suggests that food alone may not be enough of a stimulus for feral swine to approach domestic swine facilities. The slight preference of feral swine for the domestic female pig pen may be related to male feral swine seeking a mate (Barrett, 1971; Dexter, 1999) or to the social nature of the species.

Feral swine, particularly males, have been recorded traveling several kilometers during a few days (Caley, 1997; Dexter, 1999), which would present them the opportunity to visit domestic swine within 10 km of their area of use. Our study suggests that Texas feral swine do not travel far in general, with 8 km being a normal length of utilized area for animals in southern Texas. Despite the fact that many of the neighboring domestic swine facilities were ≤ 10 km of trapping sites, only seven of 35 collared feral swine appeared to be attracted to those areas.

Management implications

The findings from our study demonstrate that feral swine do have contact with domestic swine, presenting a disease transmission threat. However, the limited number of contact events also indicates that the problem could be mitigated with relatively simple management options. A double fence construction around domestic swine paddocks would likely reduce the chance of direct contact-based disease transmission. The efficacy of this option should be further investigated, and farmers and ranchers should consider the economic cost of materials and construction. In addition, our data suggest that reduced human activity and the cover of night create opportunity for feral swine to increase their use of areas around domestic swine facilities. Future research should investigate disruptive technologies that farmers and ranchers could use to prevent these opportunities for feral swine to come into contact with their livestock.

Finally, in Texas, feral swine did not appear to travel large distances to visit the neighboring farm facilities. Instead, the animals that were relocated inside contact zones A and B also were trapped and remained in the general surrounding area. This suggests that reduction of feral swine populations could be applied to the immediate area around a facility to reduce the disease threat. Research is needed to identify an effective buffer measurement and to assess the probability of feral swine immigration.

ACKNOWLEDGMENTS

We thank the US Department of Agriculture, Animal Plant Health Inspection Services, Wildlife Services, National Wildlife Research Center; the US Department of Agriculture, Animal Plant Health Inspection Services, Veterinary Services; and the Caesar Kleberg Wildlife Research Institute for financial support. Land access and logistical support were generously provided by Texas Parks and Wildlife Department, Texas A&M University-Kingsville Farm facilities, Texas A&M University System, D. Baldree, F. Flournoys, J. Maxy, and additional private landowners. We also thank S. Edwards,

J. Fischer, M. Hall, B. Hopkins, D. Krapes, M. Lavelle, D. Long, J. Moczygemba, K. Porter, M. Reidy, L. Roberson, M. Schenk, H. Smith, J. Stevenson, J. Treadway, S. Ynostrosa, E. Wehland, A. Windham, and S. Wyckoff for assistance with field work and associated project aspects. This is publication number 08-124 of the Caesar Kleberg Wildlife Research Institute.

LITERATURE CITED

- BARRETT, R. H. 1971. Ecology of the feral pig in Tehama County, California. PhD Thesis, University of California, Berkeley, California, 368 pp.
- CALEY, P. 1997. Movements, activity patterns and habitat use of feral pigs (*Sus scrofa*) in a tropical habitat. *Wildlife Research* 24: 77–87.
- CHRISTENSEN, L. S., S. MORTENSEN, A. BOTNER, B. S. STRANDBYGAARD, L. RONSHOLD, C. A. HENRIKSEN, AND J. B. ANDERSEN. 1993. Further evidence of long distance airborne transmission of Aujeszky's disease (pseudorabies) virus. *Veterinary Record* 132: 317–321.
- CONGER, T. H., E. YOUNG, AND R. A. HECKMANN. 1999. *Brucella suis* in feral swine. In Proceedings of the National Feral Swine Conference 1, Fort Worth, Texas, 2–3 June 1999, pp. 98–107.
- CORN, J. L., J. C. CUMBEE, B. A. CHANDLER, D. E. STALKNECHT, AND J. R. FISHER. 2005. Implications of feral swine expansion: Expansion of feral swine in the United States and potential implications for domestic swine. In Feral swine subcommittee on brucellosis and pseudorabies report. Southeastern Cooperative Wildlife Disease Study, United States Animal Health Association, St. Joseph, Missouri, pp. 5–7.
- , D. E. STALKNECHT, 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.
- , P. K. SWIDEREK, B. O. BLACKBURN, G. A. ERICKSON, A. B. THIERMAN, AND V. F. NETTLES. 1986. Survey of selected disease in wild swine in Texas. *Journal of American Veterinary Medicine Association* 189: 1029–1032.
- DAVIDSON, W. R., AND V. F. NETTLES. 1997. Field manual of wildlife diseases in the southeastern United States. 2nd Edition. Southeastern Cooperative Wildlife Disease Study, University of Georgia, Athens, Georgia, 417 pp.
- DEXTER, N. 1999. The influence of pasture distribution, temperature and sex on home-range size of feral pigs in a semi-arid environment. *Wildlife Research* 26: 755–762.
- FERAL SWINE SUBCOMMITTEE ON BRUCELLOSIS AND PSEUDORABIES. 2005. Feral swine subcommittee on brucellosis and pseudorabies report. United States Animal Health Association, St. Joseph, Missouri.

- FOGARTY, E. P. 2007. National distribution of and stakeholder attitudes toward feral hogs. MS Thesis, Department of Wildlife and Fisheries, Mississippi State University, Starkville, Mississippi, 99 pp.
- GRESHAM, C. S., C. A. GRESHAM, M. J. DUFFY, C. T. FAULKNER, AND S. PATTON. 2002. Increased prevalence of *Brucella suis* and pseudorabies virus antibodies in adults of an isolated feral swine population in coastal South Carolina. *Journal of Wildlife Diseases* 38: 653–656.
- HAHN, N., C. HSU, AND B. PASKIET. 1999. Research on PRV in feral swine: Past, present and future direction. *In Proceedings of the National Feral Swine Conference 1*, Fort Worth, Texas, 2–3 June 1999, pp. 75–85.
- KRISTENSEN, C. S., A. BOTNER, H. TAKAI, J. P. NIELSEN, AND S. E. JORSAL. 2004. Experimental airborne transmission of PRRS virus. *Veterinary Microbiology* 99: 197–202.
- LAWHORN, B. 1999. Texas rolling plains feral swine disease survey. *In Proceedings of the National Feral Swine Conference 1*, Fort Worth, Texas, 2–3 June, pp. 124–127.
- MATSCHKE, G. H. 1967. Aging European wild hogs by dentition. *Journal of Wildlife Management* 31: 109–113.
- NATIONAL VETERINARY SERVICES LABORATORIES. 2005. *Classical swine fever (CSF) surveillance procedure manual*, http://www.aphis.usda.gov/vs/nahss/swine/csf/CSF_Procedure_Manual.pdf. Accessed August 2006.
- NETTLES, V. F., J. L. CORN, G. A. ERICKSON, AND D. A. JESSUP. 1989. A survey of wild swine in the United States for evidence of hog cholera. *Journal of Wildlife Diseases* 25: 61–65.
- , AND G. A. ERICKSON. 1984. Pseudorabies in wild swine. *Proceedings of the United States Animal Health Association* 88: 505–506.
- NEUMANN, E. J., J. B. KLIEBENSTEIN, C. D. JOHNSON, J. W. MABRY, E. J. BUSH, A. H. SEITZINGER, A. L. GREEN, AND J. J. ZIMMERMAN. 2005. Assessment of the economic impact of porcine reproductive and respiratory syndrome on swine production in the United States. *Journal of the American Veterinary Medical Association* 227: 385–392.
- SALIKI, J. T., S. J. RODGERS, AND G. ESKWE. 1998. Serosurvey of selected viral and bacterial diseases in wild swine from Oklahoma. *Journal of Wildlife Diseases* 34: 834–838.
- [SCWDS] SOUTHEASTERN COOPERATIVE WILDLIFE DISEASE STUDY. 1992. Wild swine disease awareness: 8.3.
- [SCWDS] SOUTHEASTERN COOPERATIVE WILDLIFE DISEASE STUDY. 2004. Southwestern Cooperative Wildlife Disease Study. Feral/wild swine populations 2004 map, Southeastern Cooperative Wildlife Disease Study, Athens, Georgia.
- STEEL, R. G. D., AND J. H. TORRIE. 1980. Principles and procedures of statistics: A biometrical approach. 2nd Edition. McGraw-Hill Book Company, New York, New York, 633 pp.
- SWEITZER, R. A., I. A. GARDENER, B. J. GONZALES, D. VAN VUREN, AND W. M. BOYCE. 1996. Population densities and disease surveys of wild pigs in the coast ranges of central and northern California. *In Proceedings of the Vertebrate Pest Conference 17*, C. Crabb (ed.), Rohnert Park, California, 5–8 March, pp. 75–82.
- THORNE, E. T. 2001. Brucellosis. *In Infectious diseases of wild mammals*, E. S. Williams and J. K. Barker (eds.). Iowa State University Press, Ames, Iowa, pp. 372–395.
- [USDA/APHIS] UNITED STATES DEPARTMENT OF AGRICULTURE/ANIMAL PLANT HEALTH INSPECTION SERVICE. 2007a. *Pseudorabies state status map*, www.aphis.usda.gov/animal_health/animal_dis_spec/swine/images/pseu_st_status_map.jpg. Accessed June 2008.
- [USDA/APHIS] UNITED STATES DEPARTMENT OF AGRICULTURE/ANIMAL PLANT HEALTH INSPECTION SERVICE. (USDA/APHIS). 2007b. *Brucellosis state status map*, www.aphis.usda.gov/animal_health/animal_dis_spec/swine/images/br_status_map.jpg. Accessed June 13, 2008.
- UNITED STATES NAVAL OBSERVATORY, ASTRONOMICAL APPLICATIONS DEPARTMENT. http://aa.usno.navy.mil/data/docs/RS_Oneyear.php. Accessed September 2006.
- WITMER, G. W., R. B. SANDERS, AND A. C. TAFT. 2003. Feral swine—are they a disease threat to livestock in the United States? *Proceedings of the Wildlife Damage Management Conference 10*: 316–325.
- WOOD, G. W., AND R. E. BRENNEMAN. 1980. Feral hog movements and habitat use in coastal South Carolina. *Journal of Wildlife Management* 44: 420–427.
- WYCKOFF, A. C., S. E. HENKE, T. A. CAMPBELL, D. G. HEWITT, AND K. C. VERCAUTEREN. 2005. Preliminary serologic survey of selected disease and movements of feral swine in Texas. *In Proceedings of the Wildlife Damage Management Conference*, D. L. Nolte and K. A. Fagerstone (eds.), Traverse City, Michigan, 16–19 May, 11: pp. 23–32.
- , ———, ———, ———, AND ———. 2007. GPS collars: Considerations before you open your wallet. *In Proceedings of the Wildlife Damage Management Conference*, D. L. Nolte and K. A. Fagerstone (eds.), Corpus Christi, Texas, 9–12 April 2007, pp. 571–576.
- , ———, ———, AND ———. 2006. Is trapping success of feral hogs dependent upon weather conditions? *In Proceedings of the Vertebrate Pest Conference 22*, R. Timm (ed.), Berkeley, California, 6–9 March, pp. 370–372.

Received for publication 28 February 2008.