

PRELIMINARY SEROLOGIC SURVEY OF SELECTED DISEASES AND MOVEMENTS OF FERAL SWINE IN TEXAS

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Abstract: Feral swine (*Sus scrofa*) populations occur throughout eastern, central, and southern Texas, and their populations appear to be increasing. Despite their abundance and wide distribution, little is known about their range and interaction with domestic animals. In the last decade the national pork production industry has enforced an eradication program for economically detrimental swine diseases such as pseudorabies and brucellosis. It is hypothesized that feral hogs can be reservoirs that could reintroduce diseases to disease-free domestic swine herds. The objectives of this on-going project are to determine the prevalence of selected swine diseases that exist within feral hog populations in eastern and southern Texas and to determine the potential for disease transmission between feral and domestic swine. To date, feral hogs were trapped and ear-tagged (N = 212), and blood was obtained (N = 163) for serology testing for pseudorabies, brucellosis, and classic swine fever (CSF). Selected adults (N = 57) were fitted with GPS telemetry collars and released at their capture site. Prevalence of brucellosis and pseudorabies was 23.5% and 22.5%, respectively, for feral hogs in Texas. Of the hogs exposed to disease, feral hogs from southern Texas were 3 times as likely to have been exposed to pseudorabies than brucellosis; whereas, the opposite occurred for feral hogs from eastern Texas, which were 3 times more likely to have been exposed to brucellosis than pseudorabies. Prevalence of CSF in feral hogs is pending. Movements of feral hogs in southern Texas indicate that the potential for disease transmission to domestic pigs exists. Data collection will continue for approximately 1.5 years.

Key words: brucellosis, disease, domestic pig, feral hog, pseudorabies, swine, Texas

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INTRODUCTION

Feral hogs (*Sus scrofa*) are an extremely successful invasive species that were first introduced into North America around the 1500s (Towne and Wentworth 1950). Their success is due, in part, to the

fact that they are reproductively prolific and opportunistic feeders that display high intelligence and evasive behavior, which makes their populations difficult to manage. Both valued and detested by land owners, feral hogs are a common topic of

conversation in Texas and there is a local saying that “in Texas there are two types of landowners: those with feral hogs and those who are about to get feral hogs.” Once a population is established, control or eradication is difficult, expensive, and generally considered impossible on a large scale.

Recent feral hog introductions in Texas most likely occurred from one of three scenarios. In the 1900s Texans practiced free-range livestock husbandry. The lack of well defined property lines and fencing allowed for uninhibited movement of hogs. In times of economic downturn, such as the Great Depression of the 1930s, or when the pork market prices decreased below production costs, it was not uncommon for farmers to abandon herds of pigs and move to the city for better economic opportunity. Over time domestic swine, that had evaded recapture or had been abandoned, became feral. Recently, the more common, and problematic origin is the intentional release of domestic or relocated feral hogs for sport hunting (Mayer and Brisbin 1991). Between these three modes of introduction, feral hogs have a distribution throughout Texas and are now reported in 32 of the 50 states (Romero et al. 2003). Some of these populations in the US, including Texas, also have introduced European boar genetics. The introduction of the European boar in Texas was first recorded in 1930 when some escaped (or were released) from the San Antonio Zoo into Aransas County (Taylor 1993). Since then, there are rumored to have been several other likely introductions. The motivation for introducing the European boar is for sport hunting aesthetics.

The national population of feral hogs has been estimated at 4 million, with the population in Texas constituting 1-1.5 million (Pimentel 2001). Reported to exist in 185 of Texas’ 254 counties (Rollins

1993), the heaviest densities occur in eastern and southern Texas, with the lightest densities in western Texas and the Panhandle region (Taylor 1993). In general, feral hog densities are difficult to ascertain because of the animal’s nocturnal and cautious behaviors, as well as the fact that they have an extremely high rate of reproduction (Taylor 1993, Engeman et al. 2001). However, estimates put them at the second most prolific species after white-tailed deer (*Odocoileus virginianus*) (Taylor 1993).

Hogs are susceptible to many different viruses, parasites, and bacteria. Within feral populations in the United States, up to 30 different diseases have been found (Davis et al. 1981). Two diseases in particular, brucellosis and pseudorabies (PRV) have been found in 10 (Miller 1993) and 11 states (Romero et al. 1997), respectively. Classical swine fever (CSF) is believed to have been eradicated in 1978 (Davidson and Nettles 1997), but is still considered a concern. These three diseases are economically significant to the domestic pork industry and are under government surveillance. The current option for outbreak control for all three diseases in domestic and feral populations is depopulation.

Brucellosis is caused by *Brucella suis*, a small gram-negative bacteria. Infections may be asymptomatic, or have chronic clinical signs including abortion, fetal reabsorption, infertility in sows, orchitis (inflammation of the testes) in boars, lameness, and a high mortality in piglets (Tessaro 1990, Davidson and Nettles 1997). Transmission occurs by oral and venereal routes and the bacteria localizes in lymph nodes with an incubation period between 2 weeks to several months (Davidson and Nettles 1997, Conger et al. 1999). A fully effective vaccine has not yet been developed and there is no known cure

for the disease. This disease also is zoonotic and poses a public health concern. Diagnosis can be confirmed by serological testing. In the United States brucellosis has been found in Alabama (Davidson and Nettles 1997), Arkansas (Zygmunt et al. 1982), California (Sweitzer et al. 1996, Davidson and Nettles 1997), Florida (Zygmunt et al. 1982, Belden 1993, van der Leek et al. 1993a, Davidson and Nettles 1997), Georgia (Hanson and Karstad 1950, Zygmunt et al. 1982, Davidson and Nettles 1997), Hawaii (Davidson and Nettles 1997), Louisiana (Zygmunt et al. 1982, Davidson and Nettles 1997), Oklahoma (Davidson and Nettles 1997), South Carolina (Wood et al. 1976, Davidson and Nettles 1997, Zygmunt et al. 1982, Gresham et al. 2002), and Texas (Randhawa et al. 1977, Corn et al. 1986, Davidson and Nettles 1997). Research has shown that prevalence in feral swine populations can range from 0 to 44% (Dees 1999).

Pseudorabies (PRV, Aujeszky's disease, Mad Itch) is an alphaherpes virus, suid herpesvirus 1 (SHV-1), which occurs naturally in swine species, but is lethal to non-swine species that contract the virus (Kocan 1990). When infection occurs, the virus travels along peripheral sensory nerves towards neurons in ganglia, where it maintains in latent status until reactivated in periods of stress (Romero et al. 2003). In swine the disease ranges from asymptomatic to fatal in young animals, and is dependant on strain and age of infected animal (Davidson and Nettles 1997). Clinical signs include fever, respiratory infection, loss of coordination, abortion, mummified fetuses, stunted growth, and high mortality in piglets less than 4 weeks old. (Kocan 1990, Davidson and Nettles 1997). A current theory is that modes of transmission are different in feral swine verses domestic pigs due to different ganglial sites of latency. Research by Romero et al. (2003) has

indicated that the virus settles in the sacral (most common in feral swine) and trigeminal ganglia (most common in domestic swine) of the nervous system tissues, and also can be isolated from the tonsil. In feral hogs, due to virus' location in sacral ganglia, venereal transmission has the highest frequency (Romero et al. 1997, 2001, 2003), unlike in domestics where the virus is predominantly transmitted through exchange of oral and nasal fluids. However, PRV has occurred by aerosol transmission (Schoenbaum et al. 1990, Christensen et al. 1993), infected meat, and contaminated food and water (Kocan 1990, Hahn et al. 1997, Kluge et al. 1999). Diagnosis can be confirmed by serological tests, which will show antibody titers indicating that the animal was exposed to the virus at some point, though the virus may be currently latent (Kocan 1990). Evidence also suggests that seronegative animals can convert to seropositive under stressful conditions such as transport (Hahn et al. 1999). The wild-type of PRV, found in feral swine, appears to be attenuated, having a lower pathogenicity than those found in domestic herds, and therefore may not manifest symptoms making it difficult to recognize the virus in domestic herds (Romero et al. 1999). Pseudorabies appears to be well established in feral populations throughout the US, and persists in populations through time (Gresham et al. 2002, Corn et al. 2004). Infected populations have been found in Florida (van der Leek et al. 1993a, b), Georgia (Pirtle et al. 1989), Oklahoma (Davidson and Nettles 1997), South Carolina (Wood et al. 1992, Gresham et al. 2002), Texas (Corn et al. 1986) and have been confirmed in 12 unlisted states (Miller et al. 1993). Rates of infection have varied from not present to 70% (Pirtle et al. 1989, van der Leek et al. 1993a, Sweitzer et al. 1996, Hahn et al. 1999, Gresham et al. 2002, Corn et al. 2004), and seem to be dependant

on location, season of sampling, and age structure of sampled population (Romero et al. 1999).

Classical Swine Fever, a viral disease, was eradicated in 1978 (Davidson and Nettles 1997) and is not believed to currently be present in the United States. Symptoms of infection include lethargy, fever, inappetence, pneumonia, and gastroenteritis (Davidson and Nettles 1997). It is generally fatal, though mild cases can be overcome and animals can become carriers (Davidson and Nettles 1997). All suid species are susceptible, though to varying degrees. Transmission is through direct contact with infected animals, or contaminated food, water, and fomites (Davidson and Nettles 1997).

Feral hogs, as a disease reservoir, can be economically significant. The US pork industry is valued at \$30 billion annually, employs over 600,000 jobs, and produces 10% of the world's pork supply (www.aphis.usda.gov/vs/nahps/pseudorabies/q-a.html, Witmer et al. 2003) giving the industry valid concern when it comes to disease management. Since 1989, the domestic pork industry has participated in a USDA coordinated, national campaign to eradicate brucellosis and PRV. It has been estimated that PRV alone costs the national pork industry \$40 million annually, not including loss of market opportunity internationally (NIAA: www.animalagriculture.com). The PRV program has five stages, stage I is preparation, stage II is control, stage III is mandatory cleanup of all pseudorabies-infected herds, stage IV is surveillance to make sure no infection remains, and stage V status is when all herds are pseudorabies-free for one year or more. As of November 2003, 46 states were in stage V, with Texas, Iowa, Pennsylvania and Florida at stage IV (NIAA: www.animalagriculture.com). The threat of reintroduction of these diseases to

uninfected domestic herds by diseased feral populations has been considered in scientific literature, but has not been actively researched. Therefore, the objectives of this on-going project are to determine the prevalence of brucellosis, PRV, and CSF that exist within feral hog populations in eastern and southern Texas and to determine the potential for disease transmission between feral and domestic swine.

METHODS

Southern and eastern Texas were selected as collection sites because feral hogs are more abundant in these regions of Texas. Specific locations in eastern Texas included Gus Engeling Wildlife Management Area, Big Lake Bottom Wildlife Management Area, and Temple Inland forestry land. Collection locations in southern Texas included private lands, La Copita Research Area, and the Texas A&M University-Kingsville farm facility. Each trapping area had neighboring domestic swine facilities that ranged from large scale pork production (>100 pigs) to "ma and pa" show and feeder pig operations. Locations of domestic pig facilities were recorded using a hand-held Garmin GPS unit.

Feral hog trap sites were chosen in areas with habitat that appeared suitable for hogs, or in areas where sign of recent use by hogs (i.e., rooting, scat and tracks) was present. Traps consisted of corral (3 to 5 m in diameter and constructed of hog panel fencing with fence posts) and box trap (2.5 x 1 x 1 m) styles. Traps were placed in shaded areas to prevent trapped animals from over-heating. Trap sites were baited with soured corn. Traps were checked at least once per day just after sunrise to reduce heat exposure and traps were re-baited each morning.

Trapped hogs were anesthetized via a dart gun equipped with a Telazol and xylazine combination according to the

methods and dosages of Sweitzer et al. (1996). In brief, threshold dosages for 16-170 kg pigs are 2.8 – 3.23 mg/kg for Telazol, and 1.44 – 1.63 mg/kg for xylazine. Weight of captured hogs initially was estimated to administer the Telazol:xylazine combination. Heart rate, respiration and temperature of captured hogs were monitored during immobilization as a determinant of stress.

Blood was obtained from each captured hog. Samples were centrifuged and serum collected, and then froze at 0 C. Serum was sent to the Texas Animal Health Diagnostic laboratory (Austin, Texas) for serological testing of brucellosis, PRV, and CSF. Body measurements (cm) of length (tip of snout to base of tail), shoulder height (tip of right front hoof to upper shoulder blade), chest circumference (just behind front legs), neck circumference (taken at mid-neck region), and length of longest tusk were taken with a measuring tape. Actual weight of each captured hog was taken with a sling scale to the nearest 2 kg. Sex was recorded. Age was estimated by tooth wear and eruption patterns and hogs were classified into one of three age categories: young (less than 9 months), juvenile (9-22 months), and adult (older than 22 months) (Matschke 1967). All captured hogs were ear-tagged with an individual identification number, while selected adults (i.e., hogs with neck circumference > 62.5 cm) were fitted with a GPS-telemetry collar (Televilt Co., Sweden). Hogs were placed in a shaded area and allowed to fully recover from the anesthesia before being released.

GPS collars were programmed to acquire and store their location from satellites 24 times per week. In addition, GPS collars emitted a VHF signal for a 4-hour period twice per week. During these 4-hour periods, the study areas were flown once per month to gain additional locations of collared feral hogs and to determine that

the GPS collars were continuing to function properly. Collars from hunter-harvested hogs, dead hogs, or collars that slipped off live animals were retrieved and stored location data within these collar were downloaded into a computer.

Radio-tracking data were analyzed and mapped using ArcView. Locations of feral hogs and domestic pig facilities were overlaid onto DOQQ photos. Relocations of collared feral hogs that were within 100 m of a domestic swine facility were considered an interaction between the feral and domestic populations. Prevalence of diseases were analyzed between geographic regions (i.e., eastern and southern Texas) with Chi-squared analysis using the Yates correction factor. Values for the mean serologic titer of disease were rank-transformed (PROC RANK; SAS Institute Inc., 1990). Rank-transformed values for serologic titers of disease were examined for main effects of geographic region (i.e., eastern and southern Texas), hog age (i.e., juvenile, adult), and interactive effects with analysis of variance (ANOVA; SAS Institute., 1990). If significant interactions were detected, single variates of the interaction were analyzed separately within each grouping of the other main effect. Multiple comparisons were made using Tukey's studentized range (HSD) test when significant effects were found (Cochran and Cox, 1957). All tests were considered significant at $P \leq 0.05$. Descriptive statistics are presented as the mean \pm 1 SE.

RESULTS

To date, 212 feral hogs have been captured, of which 18 were recaptures. Sex ratio (N = 194) did not deviate (93:101 [M:F]; $\chi^2=0.25$, $df = 1$, $P > 0.65$) from a 1:1 relationship. Age structure of captured hogs was 80, 48, and 34 for young, juvenile, and adult hogs, respectively. Age structure of feral hogs differed ($\chi^2= 20.5$,

df = 2, $P > 0.001$) between age categories. Young animals were more numerous and constituted a greater percentage of the calculated chi-square value (61%) than adults (36% of chi-square value) and juveniles (3% of chi-square value).

Of the 163 serum samples collected thus far, we have brucellosis and PRV results for 102 animals. Results for CSF are pending. Prevalence of brucellosis and PRV was 23.5% and 22.5%, respectively, for feral hogs in Texas (Table 1). Feral

hogs from eastern Texas were 3 times more likely ($\chi^2 = 5.1$, df = 1, $P < 0.02$) to be exposed to brucellosis than pseudorabies; whereas, feral hogs from southern Texas were 3 times more likely ($\chi^2 = 6.5$, df = 1, $P < 0.01$) to be exposed to pseudorabies than brucellosis. However, titers from positive animals for both diseases were low (<53 PCFIA, brucellosis; <1/64 S/N, PRV; Table 1) and did not differ ($F < 1.97$, $P > 0.34$) by geographic region, hog age category, or interactive effects.

Table 1. Serologic prevalence of brucellosis and pseudorabies in 102 feral hogs from eastern and southern Texas during 2004-2005.

Disease	Eastern Texas				Southern Texas			
	N ^a	n ^b	\bar{x} ^c	Range ^d	N ^a	n ^b	\bar{x} ^c	Range ^d
Brucellosis	60	20	31.1	15 - 53	42	4	22.2	18 - 31
Pseudorabies	60	7	1:9	1:8 - 16	42	16	1:27	1:4 - 64

^aNumber of feral hogs sampled.

^bNumber of feral hogs with positive antibody titers.

^cMean antibody titer.

^dRange of antibody titers.

Fifty-seven adult hogs have been fitted with GPS telemetry collars, of which 27 collars were retrieved from hunted and dead hogs or were collars that slipped off the hogs. Currently 30 collars remain active on live feral hogs (25 hogs in eastern Texas and 5 hogs in southern Texas). Of the 27 retrieved collars, 6 collars were on live animals long enough (> 1 month) to gain information concerning hog movements. Three of them were frequently located near the domestic pig facility of the Texas A&M University-Kingsville farm. The remaining hogs from which we have location information did not interact with domestic pigs.

DISCUSSION

Serum antibody titers suggested that feral hogs captured in our study had been exposed to brucellosis and pseudorabies. Corn et al. (1986) reported similar results for feral hogs in Texas. However, our results may be conservative because serologic testing for antibodies may not be sensitive enough to give a complete picture of disease prevalence. The viral DNA recovery through Polymerase Chain Reaction (PCR) is twice as sensitive, and allows positive identification of infected individuals with extremely low titers (Hahn et al. 1999). However, this method requires tissue sampling from the lymphatic or the nervous systems. PCR technology also is

allowing for distinction between feral swine virus and domestic swine virus, giving the potential to identify the original disease source (Hahn et al. 1999). In addition, our prevalences may be conservative because both diseases potentially have a high mortality rate in piglets (Davidson and Nettles 1997), and animals seronegative for pseudorabies can convert to seropositive under stressful conditions such as transport (Hahn et al. 1999).

Interestingly, the prevalences of brucellosis and pseudorabies were reversed between eastern and southern Texas. Such results suggest that each disease may be regionally important rather than of statewide concern. However, it must be kept in mind that serological tests indicate that an animal was exposed to a disease at some point, though the disease may be currently latent (Kocan 1990).

Preliminary hog movement data indicate that feral hogs do interact with domestic pigs. Our sample size was small (N = 3 of 6); however, the potential for interaction between the two groups of swine exists and interactions do occur, even if it only occurs minimally. Our interaction data between the two groups of swine most likely is conservative because our known interactions only occur when the GPS collars are activated (i.e., 24 15-minute windows per week). It is possible that feral hogs and domestic swine interacted more frequently, but that these interactions occurred outside our window of data acquisition. Therefore, we arbitrarily designated a 100-m zone around a domestic pig facility as an interaction in an attempt to offset this potential bias. In addition, although anecdotal, we have been told by more than one land owner that he witnessed a feral hog boar breed one of his domestic pig sows through a fence barrier.

In conclusion, even though our results are preliminary, feral hogs are exposed to economically significant diseases and movements of feral hogs indicate that the potential for disease transmission to domestic pigs exists. Data collection will continue for approximately 1.5 years.

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LITERATURE CITED

- APHIS. Pseudorabies. www.aphis.usda.gov/vs/nahps/pseudorabies/q-a.html [Accessed 19 December 2003.]
- BELDEN, R.C. 1993. Feral hogs: The Florida experience. Pages 101-106 *in* D. Rollins, editor. Proceedings of feral swine: A compendium for resource managers. Texas Animal Health Commission, Austin, TX, USA.
- CHRISTENSEN, L.S., S. MORTENSEN, A. BOTNER, B.S. STRANDBYGAARD, L. RONSHOLT, C.A. HENRIKSEN, AND J.B. ANDERSEN. 1993. Further evidence of long distance airborne transmission of Aujeszky's disease (pseudorabies) virus. *The Veterinary Record* 132:317-321.

- COCHRAN, W.G., AND G.M. COX. 1957. Experimental designs, Second Edition. John Wiley & Sons, Inc., New York, NY, USA.
- CONGER, T.H., E. YOUNG, AND R.A. HECKMANN. 1999. *Brucella suis* in feral swine. Proceedings of the National Feral Swine Conference 1:98-107.
- CORN, J.L., P.K. SWIDEREN, 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.
- _____, 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.
- DAVIDSON, W.R., AND V.F. NETTLES. 1997. Field manual of wildlife diseases in the southeastern United States, Second edition. Southeastern Cooperative Wildlife Disease Study, University of Georgia, Athens, GA, USA.
- DAVIS, J.W., L.H. KARSTAD, AND D.O. TRAINER. 1981. Infectious diseases of wild mammals, Second edition. The Iowa State University Press, Ames, IA, USA.
- DEES, T. 1999. Feral/wild swine surveillance for foreign animal diseases and some field study projects on brucellosis and pseudorabies in the southeastern USA. Proceedings of the National Feral Swine Conference 1:121-123.
- ENGEMAN, R.M., B. CONSTANTIN, M. NELSON, J. WOOLARD, AND J. BOURASSA. 2001. Monitoring changes in feral swine abundance and spatial distribution. Environmental Conservation 28:235-240.
- 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 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.
- HAHN, N., C. HSU, AND B. PASZKIET. 1999. Research on PRV in feral swine: Past, present and future direction. Proceedings of the National Feral Swine Conference 1:75-85.
- HANSON, R.P., AND L. KARSTAD. 1950. Feral swine in the southeastern United States. Journal of Wildlife Management 23:64-74.
- KLUGE, J.P., G.W. BERAN, H.T. HILL, AND K.B. PLATT. 1999. Pseudorabies (Aujeszky's Disease). Pages 233-246 in B.E. Straw, S. D'Allaire, W.L. Mengelng, and D.L. Taylor, editors. Diseases of swine. Iowa State University Press, Ames, IA, USA.
- KOCAN, A. 1990. Pseudorabies. Pages 43-44 in R. Lind, editor. Review of wildlife disease status in game animals in North America, ADF - Saskatchewan Agriculture Development Fund, Regina, Saskatchewan, Canada.
- MATSCHKE, G.H. 1967. Aging European wild hogs by dentition. Journal of Wildlife Management 31:109-113.
- MAYER, J.J., AND I.L. BRISBIN, JR. 1991. The wild pigs of the United States: Their history, morphology and current status. First edition. University of Georgia Press, Athens, GA, USA.
- MILLER, J.E. 1993. A national perspective on feral swine. Pages 9-16 in D. Rollins, editor. Proceedings of feral swine: A compendium for resource managers. Texas Animal Health Commission, Austin, TX, USA.
- PIMENTEL, D. 2001. Economic and environmental threats of alien plant, animal, and microbe invasions. Agriculture Ecosystems and Environment 84:1-20.
- PIRTLE, E.C., J.C. SACKS, V.F. NETTLES, AND E.A. ROLLOR, III. 1989. Prevalence

- and transmission of pseudorabies virus in an isolated population of feral swine. *Journal of Wildlife Diseases* 25:605-607.
- RANDHAWA, A.S., V.P. KELLY, AND E.F. BAKER JR. 1977. Agglutinins to *Coxiella burnetii* and *Brucella spp*, with particular reference to *Brucella canis* in wild animals of southern Texas. *Journal of American Veterinary Medical Association* 171:939-942.
- ROMERO, C.H., P. MEADE, J. SANTAGATA, K. GILLIS, G. LOLLIS, E.C. HAHN, AND E.P.J. GIBBS. 1997. Genital infection and transmission of pseudorabies virus in feral swine in Florida, USA. *Veterinary Microbiology* 55:131-134.
- _____, P.N. MEADE, B.L. HONER, J.E. SHULTZ, AND G. LOLLIS. 2003. Potential sites of virus latency associated with indigenous pseudorabies viruses in feral swine. *Journal of Wildlife Diseases* 39:567-575.
- _____, P.N. Meade, J.E. Shultz, H.Y. Chung, E.P. Gibbs, E.C. Hahn, AND G. Lollis. 2001. Venereal transmission of pseudorabies viruses indigenous feral swine. *Journal of Wildlife Diseases* 37:289-296.
- SAS INSTITUTE, INC. 1990. SAS/STAT User's Guide, Sixth edition, Vols. 1 and 2. SAS Institute, Cary, NC, USA.
- SCHOENBAUM, M. A., J. J. ZIMMERMAN, G. W. BERAN, AND D. P. MURPHY. 1990. Survival of pseudorabies virus in aerosol. *American Journal of Veterinary Research* 51:331-333.
- Sweitzer, S., I.A. Gardener, B.J. Gonzales , D. Van Uren, AND W. M. Boyce. 1996. Population densities and disease surveys of wild pigs in the coast ranges of central and northern California. *Proceedings of the Vertebrate Pest Conference* 7:75-82.
- TAYLOR, R.B. 1993. History and distribution of feral hogs in Texas. Pages 17-27 in D. Rollins, editor. *Proceedings of feral swine: A compendium for resource managers*. Texas Animal Health Commission, Austin, TX, USA.
- TESSARO, S.V. 1990. Brucellosis caused by *Brucella suis*. Pages 105-107 in R. Lind, editor. *Review of wildlife disease status in game animals in North America*. ADF - Saskatchewan Agriculture Development Fund, Regina, Saskatchewan, Canada.
- TOWNE, C.W., AND E.N. WENTWORTH. 1950. *Pigs from cave to corn-belt*. University of Oklahoma Press, Norman, OK, USA.
- VAN DER LEEK, M.L., H.N. BECKER, E.C. PIRTEL, P. HUMPHREY, C.L. ADAMS, B.P. ALL, G.A. ERICSON, R.C. BELDEN, W.B. FRANKENBERGER, AND E.P.J. GIBBS. 1993a. Prevalence of pseudorabies (Aujeszky's Disease) virus antibodies in feral swine in Florida. *Journal of Wildlife Diseases* 29:403-409.
- _____, H.N. BECKER, P. HUMPHREY, C.L. ADAMS, R.C. BELDEN, W.B. FRANKENBERGER, AND P.L. NICOLETTI. 1993b. Prevalence of *Brucella sp.* antibodies in feral swine in Florida. *Journal of Wildlife Diseases* 29:410-415.
- 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., J.B. HENDRICKS, AND D.E. GOODMAN. 1976. Brucellosis in feral swine. *Journal of Wildlife Diseases* 12:579-582.
- _____, L.A. WOODWARD, D.C. MATHEWS, AND J.R. SWEENEY. 1992. Feral hog control efforts on a coastal South Carolina plantation. *Annual Conference of Southeastern Association of Fish and Wildlife Agencies* 45:167-178.

ZYGMONT, S.M., V.F. NETTLES, E.B. SHOTTS
JR., W.A. CARMENT, AND B.O.
BLACKBURN. 1982. Brucellosis in
wild swine: A serologic and
bacteriologic survey in the southeastern
United States and Hawaii. Journal of
American Veterinary Medical
Association 181:1285-1287.