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INFLUENZA EXPOSURE IN UNITED STATES FERAL SWINE POPULATIONS

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ABSTRACT: Swine play an important role in the disease ecology of influenza. Having cellular receptors in common with birds and humans, swine provide opportunities for mixed infections and potential for genetic reassortment between avian, human, and porcine influenza. Feral swine populations are rapidly expanding in both numbers and range and are increasingly coming into contact with waterfowl, humans, and agricultural operations. In this study, over 875 feral swine were sampled from six states across the United States for serologic evidence of exposure to influenza. In Oklahoma, Florida, and Missouri, USA, no seropositive feral swine were detected. Seropositive swine were detected in California, Mississippi, and Texas, USA. Antibody prevalences in these states were 1% in Mississippi, 5% in California, and 14.4% in Texas. All seropositive swine were exposed to H3N2 subtype, the predominant subtype currently circulating in domestic swine. The only exceptions were in San Saba County, Texas, where of the 15 seropositive samples, four were positive for H1N1 and seven for both H1N1 and H3N2. In Texas, there was large geographical and temporal variation in antibody prevalence and no obvious connection to domestic swine operations. No evidence of exposure to avian influenza in feral swine was uncovered. From these results it is apparent that influenza in feral swine poses a risk primarily to swine production operations. However, because feral swine share habitat with waterfowl, prey on and scavenge dead and dying birds, are highly mobile, and are increasingly coming into contact with humans, the potential for these animals to become infected with avian or human influenza in addition to swine influenza is a distinct possibility.

Key words: Avian influenza, feral swine, serosurvey, swine influenza.

INTRODUCTION

Swine (*Sus scrofa*) have important roles in the disease ecology of influenza because they can be infected by various influenza subtypes (H1-H13) (Kida et al., 1994). Since they share cellular receptors with birds and humans, swine have the potential to become coinfected with both avian and mammalian strains of influenza. Thus, swine are considered "mixing vessels," and novel strains of influenza may arise by genetic reassortment of existing strains (Ito et al., 1998; Zhou et al., 1999). This process is implicated in the generation of the Spanish flu pandemic of 1918 that killed an estimated 50 million people worldwide (Fanning et al., 2002).

Swine-adapted influenza (SI) also circulates in United States domestic swine populations. Historically the predominant influenza subtype in domestic swine has been H1N1. However, in the late 1990s a triple reassortant (H3N2) emerged and since has become the dominant SI subtype across the country (Webby et al., 2000). This subtype contains genes from human, avian, and swine lineages and illustrates the risks of coinfection and reassortment in swine. More recently a reassortant between H1N1 and H3N2 has emerged (H1N2), and these multiple lineages (H1N1, H3N2, H1N2) are cocirculating in United States swine populations. Other subtypes have been also been discovered in swine (H4N6, H3N1) but have not become widely established in the United States (Karasin et al., 2000a; Webby et al., 2004; Ma et al., 2006).

Feral swine pose serious threats to natural ecosystems and agriculture wherever they become established (Kotanen, 1995; Waithman et al., 1999). Their feeding and rooting habits can destroy native plant communities, damage watersheds and irrigation systems, and displace native species. Also, swine prey on other animals including birds, reptiles and even domestic livestock (Seward et al., 2004). Feral swine also carry diseases such as brucellosis, leptospirosis, and pseudorabies, with potential to spread to domestic swine operations and can have human health implications (Witmer et al., 2003). As populations and ranges rapidly expand across the United States, the economic and biologic impacts of feral swine will require greater scrutiny.

The natural reservoir of avian influenza (AI) is wild waterfowl, gulls, and shorebirds, and the prevalence of infected birds can be as high as 30% (Webster, 1992; Clark and Hall, 2006). Avian influenza is shed in feces by infected waterfowl and is relatively stable in water (Stallknecht et al., 1990; Ito et al., 1995). Feral swine often reside in the same habitats as waterfowl, feed in the same agricultural areas, wallow and swim in the same bodies of water, and often prey on, and scavenge, dead birds for food. Therefore, ample opportunities exist for feral swine to become exposed to AI by contact with waterfowl and their environment. These animals are also highly mobile, frequently coming into contact with domestic swine and increasingly with humans (Wyckoff et al., 2005).

To date, only a few studies have examined feral swine in the USA for exposure to influenza viruses (Saliki et al., 1998; Gipson et al., 1999). The objective of this study was to conduct a serosurvey on feral swine populations in various regions across the USA to determine the levels of influenza exposure in these populations and the possible sources of infection. As feral swine populations are rapidly growing and their range expanding, this knowledge is critical to accurately assess the risks posed to swine producers, the poultry industry, and human health.

MATERIALS AND METHODS

Sample collection

Blood was collected from feral swine during control operations in various US counties in California, Texas, Missouri, Oklahoma, Florida, and Mississippi. Samples were collected from California, Mississippi, Missouri, and Texas in 2005 and 2006 and from Oklahoma in 2005 and Florida in 2006. Blood was collected via cardiac puncture, allowed to clot, and subsequently centrifuged in Microtainer® vials (Fisher Scientific) to separate sera from cellular blood components. Sera were transferred to fresh vials and stored frozen until transported to the USDA–APHIS–Wildlife Services–National Wildlife Research Center by overnight delivery. Sera were stored at −80 C until analyses. All capture and handling procedures were approved by an Institutional Animal Care and Use and Institutional Biosafety Committees.

Screening for the presence of influenza antibodies by agar gel immunodiffusion

Agar gel immunodiffusion (AGID) is a serologic assay used to detect antibodies to influenza viruses. The antigen used in this assay was derived from the matrix and nucleoproteins of AI virus and is used to detect antibodies to all subtypes of AI virus. The procedure is described by Beard (1970) and was performed using reagents and the protocol provided by the Center for Veterinary Biologics and National Veterinary Services Laboratories (NVSL).

Determination of influenza antibody subtypes

Hemagglutination (HA) is a presumptive assay used to detect hemagglutinating viruses, including influenza virus, and hemagglutination inhibition (HI) is used to determine subtype identity of an isolate and to determine subtypes of antibodies in sera. Prior to analyses the sera were treated with receptor-destroying enzyme to eliminate nonspecific receptors that would bind to red blood cells. The reference antigens used to determine HA subtypes were the following: H1 - A/NJ/8/76/EQ/1 - H1N7; H2 - A/PINTAIL/ALBERTA/293/77 - H2N9; H3 - A/DK/UKRANE/1/63 - H3N8; H3 - A/SWINE/NC/35922/98 - H3N2 (The "swine" type of H3N2 that circulates in turkeys is not detected with the A/DK/UKRAINE reagent, therefore NVSL uses

TABLE 1. Influenza seroprevalence in feral swine from Oklahoma, Missouri, and Florida.

State	Year	No. sampled	No. positive	Subtypes
Oklahoma	2005	50	0	—
Missouri	2005	50	0	—
—	2006	55	0	—
Florida	2006	60	0	—

two H3 reagents for the HI test); H4 - A/MYNAH/MASS/71 - H4N8; H5 - A/TY/WISCONSIN/68 - H5N9; H6 - A/TY/ONTARIO/63 - H6N8; H7 - A/TY/OREGON/71 - H7N3; H8 - A/TY/ONTARIO/6118/67 - H8N4; H9 - A/GULL/MD/4435/80 - H9N5; H10 - A/CK/GERMANY"N"/49 - H10N7; H11 - A/DK/MEMPHIS/456/74 - H11N9 (for 462775 and 474274) A/DK/ENGLAND/56 - H11N6; H12 - A/DK/ALBERTA/60/76 - H12N5; H13 - A/GULL/MD/704/77 - H13N6; H14 - A/MALLARD/GURJEV/263/82 - H14N5; and H15 - A/SHEARWATER/WESTERN AUSTRALIA/2576/79 - H15N9.

Neuraminidase inhibition determines the antibody subtypes present in the sera that are directed against the neuraminidase protein on the surface of the virus. These procedures are described by Beard (1970), and all procedures were performed at the Center for Veterinary Biologics and NVSL using their protocols.

RESULTS

No seropositive feral swine were detected in Missouri (2005, 2006), Oklahoma (2005), and Florida (2006; Table 1). Of 91 sampled feral swine form Mississippi, one (1%) animal from Coahoma County that was sampled in 2005 tested positive (1.1%); this animal had antibodies to H3N2 (Table 2). In California antibodies to influenza virus were detected in three swine from Santa Clara County in 2005 and in individual swine from San Benito and Sutter Counties in 2006 (Table 3). Subtype analyses revealed that all five had been exposed to H3N2 influenza subtypes.

The highest antibody prevalences to influenza virus was detected in Texas (Table 4). Although the overall prevalence was relatively high (14.4% in 2005 and 2006 combined), there was large geographical variation in local prevalence (Fig. 1). For instance, adjacent counties (San Saba and Mills) showed markedly different antibody prevalence, 88% versus 0%, respectively. Interestingly, all of the antibody positive feral swine detected in Texas had been exposed to the H3N2 subtype except in San Saba County, where analyses revealed antibodies to H3N2 in four individuals, H1N1 in four, and antibodies to both H1N1 and H3N2 in seven swine. There also was a temporal difference in prevalence. In 2005, there was an overall prevalence in Texas of 3.5% versus 16.8% in 2006. Although much of this difference could be a function of geographic variation as sampling occurred

TABLE 2. Influenza seropositive feral swine in Mississippi.

Year	County	No. sampled	No. positive (%)	Subtypes
2005	Noxubee	9	0	—
	Hancock	19	0	—
	Coahoma	19	1 (5)	H3N2
	Clarke	7	0	—
	Sunflower	12	0	—
	Wilkinson	19	0	—
	Attala	6	0	—
	2005 totals	91	1 (1)	—
2006	Hancock	5	0	—
	Noxubee	3	0	—
	2006 totals	8	0	—

TABLE 3. Influenza seropositive feral swine in California.

Year	County	No. sampled	No. positive (%)	Subtypes (*n*)
2005	Santa Clara	21	3 (14)	H3N2 (3)
	Mariposa	8	0	—
	Kern	5	0	—
	2005 totals	34	3 (9)	—
2006	Santa Clara	11	0	—
	San Benito	28	1 (3.6)	H3N2 (1)
	Madera	5	0	—
	Sutter	13	1 (7.7)	H3N2 (1)
	Monterey	1	0	—
	Merced	1	0	—
	Shasta	1	0	—
	2006 totals	60	2 (3)	—

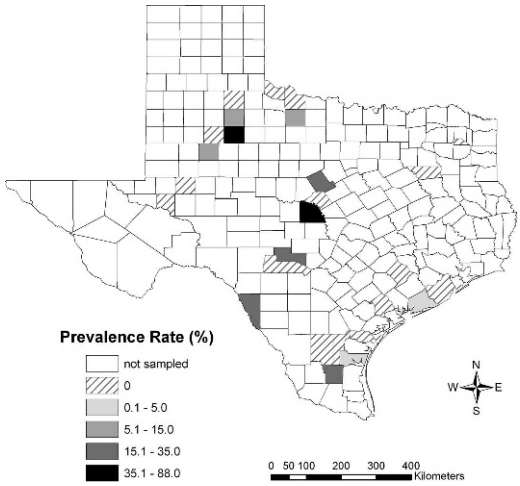

FIGURE 1. Geographic distribution of influenza seroprevalence in Texas feral swine (2005, 2006 combined).

largely in different counties, there does appear to be year-to-year variation in antibody levels as indicated by the Kerr County, Texas (Table 4), and Santa Clara County, California (Table 3), data.

DISCUSSION

The population of feral swine in Texas is estimated to be greater than two million, and therefore they outnumber domestic swine in the state by a ratio of approximately 2:1 (Mapston, 2004). In the Texas counties where high influenza antibody prevalence in feral swine was detected, there were relatively few domestic pork operations. For example, in Maverick County there were only 20 total reported domestic swine in the 2002 National Agriculture Census, yet we found that 23% (11/46) of feral swine were seropositive to influenza. At the county level there was no correlation between domestic swine population size and seroprevalence in feral swine (Pearson product-moment correlation; $P=0.46$, $r=-0.158$). Thus, it appears unlikely that the influenza exposures in feral swine documented in this study originated from domestic sources. The sheer difference in numbers of wild compared to domestic animals suggests that the virus is circulating within the feral population, although the potential spread to agricultural operations is always a distinct possibility. In particular, producers who raise swine for their own consumption, often in their backyards, have little or no biosecurity measures in place, and thus are particularly vulnerable for feral swine to come into contact with and transmit influenza virus to their animals. Unfortunately, there are no data available indicating if sympatric domestic swine have similar exposure histories as feral swine.

The reasons for the difference in prevalence between Texas and the other states are unclear. Certainly the large populations of feral swine in California and Texas may facilitate waves of influenza infection in these populations. Higher local densities of swine may also explain the seroprevalence differences observed in adjacent counties.

The predominant influenza virus exposure in feral swine was to subtype H3N2. The predominant SI subtype currently circulating in domestic swine (Webby et al., 2000) is H3N2, and, although we cannot be certain that the H3N2 that these feral swine were exposed to is SI, it is very likely the case, especially given the fact that one of the H3 antigens used for subtyping was the swine type of influenza. No evidence of exposure to avian influenza was uncovered even though many of the areas where feral swine were sampled have large seasonal concentrations of waterfowl. For example, the Gulf Coast of Texas and the central valley of California are major wintering grounds for waterfowl and other migratory birds, and AI is fairly prevalent in these areas (Hanson et al., 2005). Thus, based on these data, the potential for coinfection in feral swine with avian, human, and/or swine influenza appears to be low.

Swine play important roles in influenza transmission cycles. Swine influenza is a

TABLE 4. Influenza seropositive feral swine in Texas.

Year	County	No. sampled	No. positive (%)	Subtypes (*n*)
2005	Kerr	18	0	—
	Matagorda	15	1 (7)	H3N2 (1)
	Motley	7	0	—
	Dickens	10	1 (10)	H3N2 (1)
	Kleberg	35	1 (3)	H3N2 (1)
	2005 totals	85	3 (3.50)	—
2006	Bandera	15	0	—
	Baylor	9	1 (11)	H3N2 (1)
	Borden	9	1 (11)	H3N2 (1)
	Brazoria	10	0	—
	Brooks	9	3 (33)	H3N2 (3)
	Camp	1	0	—
	Colorado	18	0	—
	Comanche	9	2 (22)	H3N2 (2)
	Crane	4	0	—
	Duval	16	0	—
	Garza	4	0	—
	Hardeman	3	0	—
	Henderson	1	0	—
	Jim Wells	2	0	—
	Kent	35	28 (80)	H3N2 (28)
	Kerr	5	4 (80)	H3N2 (4)
	Matagorda	25	0	—
	Maverick	46	11 (24)	H3N2 (11)
	Midland	11	0	—
	Mills	11	0	—
	Real	4	0	—
	San Patricio	105	0	—
	San Saba	17	15 (88)	H3N2 (4), H1N1 (4), H3N2+H1N1 (7)
	Victoria	3	0	—
	Wilbarger	15	0	—
	2006 totals	387	65 (16.8%)	—

zoonotic disease and poses risks to human health (Myers et al., 2006; Sencer and Millar, 2006). Human influenza also can cause disease in swine (Karasin et al., 2000b; Landolt et al., 2003). Even though we found no evidence in feral populations, it is known that swine are capable of being infected with a variety of avian influenza subtypes (Kida et al., 1994). Additionally, Wright et al. (1992) found that 73% of influenza virus isolates from turkeys contained genes of swine influenza origin, swine influenza virus H3N2 has also been isolated from domestic turkeys (Choi et al., 2004), and SI reassortant H1N2 has been isolated from a wild duck (Olsen et al., 2003). These examples illustrate the reciprocal risks of human, swine, and poultry populations to cross-species transmission of influenza where they come into contact.

Once an animal becomes infected, influenza can easily and rapidly spread throughout a swine herd by aerosol and contact transmission, potentially causing severe respiratory disease (Olsen et al., 2000). Considering that pork production is a $15 billion industry in the United States, any increase in time to market weight, or loss of production due to morbidity or mortality, could have significant economic impacts.

By virtue of having both avian and human cellular receptors for influenza, swine can become coinfected by more than one influenza virus strain. This scenario creates the potential for genetic recombination of AI RNA segments with the generation of new combinations of genetic elements, new subtypes, and potential for new pandemic variants. An example of reassortment is the H3N2 subtype currently widespread in US swine. This subtype has genes from human lineages (HA, NA, PB1), swine lineages (NS, M, NP), and avian lineages (PB2, PA) (Webby et al., 2000).

As in domestic swine, the ecology of feral swine and influenza encompasses all of these factors. Whether intentionally or accidentally released into the wild, feral swine are essentially the same behaviorally, genetically, and biologically as domestic swine. However, without biosecurity and physical constraints, free-ranging feral swine often reside in habitat shared with wild waterfowl. They frequent the same bodies of water and feed in the same areas, and feral swine often prey on and scavenge dead waterfowl. Feral swine are also highly mobile and travel among suburbia, wetlands, and agricultural operations. As feral swine populations have rapidly expanded throughout the country, they increasingly contact humans, domestic swine, and poultry operations, as well as wild waterfowl. The potential for feral swine to become infected with avian, swine, and/or human influenza, to act as recombination vessels, and to transport and transmit virus to agricultural and human sites is significant. Additional research is necessary to accurately determine contact rates between domestic and feral swine and transmission from feral swine to peridomestic and other mammalian species, and to measure the effects of influenza on morbidity and mortality in feral swine. These data are required to meaningfully assess risks posed to humans and agriculture of influenza infection in feral swine populations.

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