Spillover of Swine Coronaviruses, United States

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Porcine epidemic diarrhea virus, a pathogen first detected in US domestic swine in 2013, has rapidly spilled over into feral swine populations. A better understanding of the factors associated with pathogen emergence is needed to better manage, and ultimately prevent, future spillover events from domestic to nondomestic animals.

Pathogen spillover mechanisms vary, but one route involves pathogens moving from heavily infected domestic animal hosts to nondomestic hosts (*1*). These spillover and emergence events create a dynamic landscape for pathogen transmission.

Porcine epidemic virus (PEDV) is an emergent pathogen in the United States. It can cause 90%–95% mortality in young, naive pigs and substantial weight loss and dehydration in adult swine. The virus was first documented in the United States in April 2013 and spread rapidly, leading to loss of 10% of the US commercial swine population in 31 states within 18 months (*2*), which cost the industry >US $400 million. Horizontal transmission of the virus on shared agricultural resources (*3*) most likely aided its rapid spread among facilities, demonstrating the difficulty of slowing the spread of robust pathogens.

During October 2012–September 2015, we collected serum from feral swine and analyzed it for PEDV exposure. The United States has ≈5–6 million feral swine, and their populations are expanding rapidly (*4*). Although opportunities for direct contact between feral swine and pigs in biosecure swine operations are limited, interactions have been documented with smaller backyard operations, and a recent multistate brucellosis outbreak was linked to backyard pigs infected by feral swine (*5*). Disease spillover into nondomestic hosts can serve as a continuous source for reintroduction into domestic animals, complicating international trade (*6*).

Of the 7,997 feral swine samples tested (Figure), 253 tested positive by PEDV ELISA (seroprevalence 3.2% [95% CI 2.8%–3.5%]). Those 253 samples underwent additional screening, and 8 (seroprevalence 0.1% [95% CI 0.03%–0.16%]) were confirmed to be PEDV antibody positive (online Technical Appendix Tables 1, 2, https://wwwnc.cdc.gov/EID/article/24/7/17-2077-Techapp1.pdf). Two additional samples were considered suspected positives. The remaining 245 positive samples (seroprevalence 3.1% [95% CI 2.7%–3.4%]) probably represent exposure to transmissible gastroenteritis virus (TGEV) rather than PEDV (online Technical Appendix Table 1).

The 8 PEDV-seropositive feral swine samples were from Hawaii and California (Figure). PEDV was first confirmed in California domestic swine in December 2013. The 4 positive feral swine samples from California were collected in September 2014 from adult animals in Santa Clara County. In Hawaii, seropositive feral swine were detected on Oahu and Kauai (Figure). Hawaii confirmed its first case of PEDV in domestic swine on Oahu in November 2014, but our findings identified a PEDV-positive feral swine sample collected in April 2014, before detection in domestic swine on the same island. This finding suggests initial PEDV introduction into domestic pigs in Hawaii might have gone undetected for 7 months before the first confirmed case. The 4 PEDV-positive feral swine samples from Hawaii were collected at 4 different times.

Results indicate that this newly introduced virus spilled over from domestic livestock to a nondomestic species during a relatively short period (<1 year). Prior research suggests directionality (*7*,*8*), with the virus moving from domestic swine to feral swine, rather than the reverse. Data presented here support this finding because positive feral swine were not detected until a year after detection in US domestic swine.

Biosecurity in the US commercial swine industry is comprehensive; however, the spread of PEDV demonstrates that a modern and precisely managed livestock industry is still susceptible to emergent pathogens. PEDV is relatively hardy, persisting on fomites for up to 20 days at low (4°C) temperatures (*9*). Biosecurity designed to prevent transmission of labile pathogens or to prevent introduction of a new pathogen through traditional routes may be insufficient for nonlabile pathogens introduced through new mechanisms.

The transmission pathway from infected facilities to feral swine is unknown. Previous research has detected PEDV in the environment (*3*,*7*) but did not differentiate viral RNA from infectious virus. Swine facilities often move waste to holding ponds, and these ponds could be a source of infectious virus. Infected swine in backyard operations also could facilitate spillover.

Our data also demonstrate that 3.1% of feral swine had been exposed to another coronavirus, probably TGEV. TGEV, like PEDV, is found only in swine, can survive on fomites, and can cause high mortality rates in pigs <2

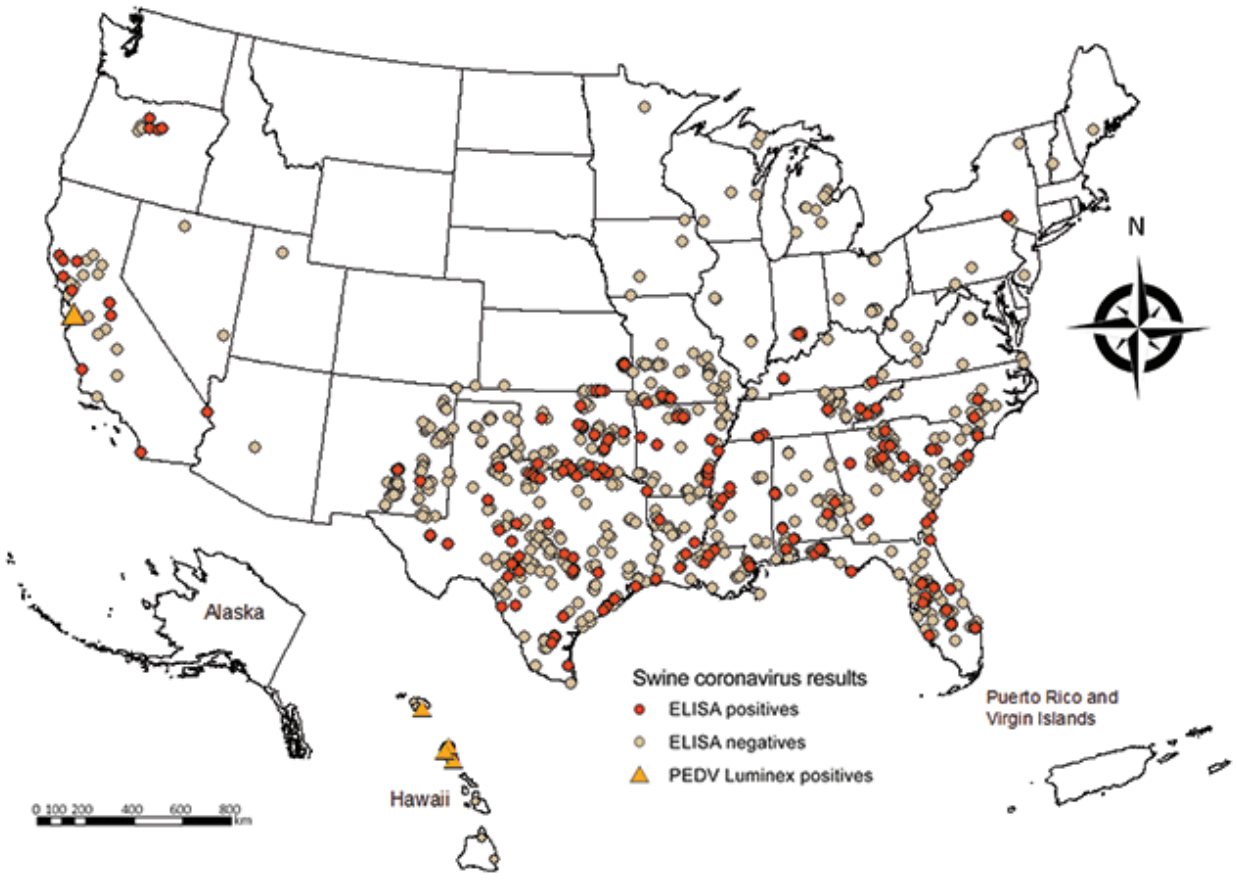

Figure. Collection locations of feral swine samples tested for exposure to swine coronaviruses, United States. In California, 4 PEDV-positive samples were detected at the same location. Samples that were ELISA-positive but PEDV-negative probably indicate exposure to transmissible gastroenteritis virus.

weeks of age. TGEV has been found in the US domestic swine industry since the 1940s. We found TGEV-positive feral swine throughout the entire sampling period and throughout the United States (Figure; online Technical Appendix Tables 1, 2), suggesting that TGEV is probably being persistently transmitted among feral swine, although continual spillover from domestic swine cannot be ruled out. Whether PEDV will display a similar pattern of endemicity over time is unknown; however, our data did not suggest continual transmission or high seroprevalence. For example, the most recent PEDV-seropositive feral swine in Hawaii was detected in January 2015. Seventy-six feral swine sampled from the same island after that date were seronegative, suggesting that either seroprevalence was low enough to evade detection or that viral transmission burned out, most likely after initial deaths of susceptible piglets. Research in Asia, however, has found higher PEDV exposure in wild boar, reinforcing that animals can survive infection and raising the possibility of continual transmission in nondomestic swine populations (*6*).

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LETTERS

Adenovirus Type 4 Respiratory Infections among Civilian Adults, Northeastern United States, 2011–2015

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To the Editor: We read with interest the article by Kajon et al. (*1*), which highlighted that human adenovirus type 4 might be an underrecognized cause of acute respiratory disease (ARD) outside military settings. We report that human adenovirus B7 (HAdV-B7) might also be a cause of this disease.

HAdV-B7 is well recognized as a causative agent of neonatal disease and infections in immunocompromised patients. However, we identified an unusual cluster of 4 cases of severe ARD caused by this pathogen in immunocompetent adults in Dublin, Ireland. These patients had acute respiratory illness when they came to the emergency department of Mater Misericordiae University Hospital in Dublin. The patients came to the hospital over a 4-week period during the summer of 2017, and each patient required intensive care support for single-organ failure. Three patients required intubation and ventilation; all 4 patients recovered.

Three patients reported gastrointestinal and respiratory symptoms, as seen in Oregon, USA (*2*). Although coinfection with other viruses or bacteria has been described (*3*), only 1 patient in our cluster had a possible concomitant pathogen. None of the 4 patients were given antiviral therapy but all received antimicrobial drugs.

All 4 case-patients were either homeless or in temporary accommodations for homeless adults, but we did not identify any epidemiologic link. The Department of Public Health and temporary accommodation sites were notified to raise awareness and offer early testing of symptomatic persons. However, no additional cases were identified.

HAdV-B7 was identified by BLAST analysis (https://blast.ncbi.nlm.nih.gov/Blast.cgi) of viral hexon genes (*4*). Each virus had 100% identity within the region sequenced to a strain previously associated with respiratory illness in a military training camp in China (GenBank accession no. KP896481).

This cluster of HAdV-B7 causing severe ARD in immunocompetent adults appears to have no clear epidemiologic link. We agree that HAdV might be an underrecognized pathogen in severe communityonset ARD. Testing for viral respiratory pathogens should be considered in all patients and not just the immunocompromised.

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Technical Appendix

Methods

Serum samples from 7,997 feral swine removed as part of permitted wildlife damage management activities were collected across the United States (Technical Appendix Table 1) during October 2012-September 2015. This covers both the time period before porcine epidemic virus (PEDV) was first detected in US domestic swine and the initial rapid spread of the virus in domestic swine facilities. Samples were collected throughout the entirety of the range of breeding feral swine populations and in both domestic swine PEDV-positive and PEDV-negative states.

Feral swine samples were screened using a whole-virus PEDV ELISA (*1*,*2*). The ELISA is based on a US PEDV isolate (USA/NC35140/2013) which detects IgA and IgG to PEDV strains (protoype and S INDEL) circulating in the United States. Gimenez-Lirola et al. demonstrated that cross-reactivity can occur with the PEDV ELISA against transmissible gastroenteritis virus (TGEV), but not against other swine coronaviruses, such as porcine respiratory cornonavirus, and porcine deltacoronavirus. Because of this potential cross-reactivity, all positive ELISA samples (sensitivity 88.8%, specificity 100%) were confirmed using a PEDV specific S1 multiplex fluorescent microbead-based immunoassay (*3*). Confidence intervals for seroprevalence values were calculated using the Copper-Pearson exact method. Results are broken down by both state and calendar of sample collection (Technical Appendix Tables 1, 2).

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Technical Appendix Table 1. Feral swine sample sizes by state, United States*

State/ Territory	No.	No. coronavirus-positive	Coronavirus seroprevalence (95% CI)	No. PEDV-positive	PEDV seroprevalence (95% CL)
Alabama	283	10	0.04 (0.02–0.06)	0	0
Arkansas	476	14	0.03 (0.02–0.05)	0	0
Arizona	67	1	0.01 (0–0.08)	0	0
California	641	18	0.03 (0.02–0.04)	4	0.006 (0–0.02)
Colorado	5	0	0 (0–0.52)	0	0
Florida	843	0	0 (0–0.01)	0	0
Georgia	440	12	0.03 (0.01–0.05)	0	0
Guam	12	0	0 (0–0.26)	0	0
Hawaii	443	18	0.04 (0.02–0.06)	4	0.009 (0–0.02)
Iowa	4	0	0 (0–0.60)	0	0
Illinois	36	0	0 (0–0.10)	0	0
Indiana	83	3	0.04 (0.01–0.10)	0	0
Kansas	251	11	0.04 (0.02–0.08)	0	0
Kentucky	25	1	0.04 (0–0.20)	0	0
Louisiana	398	14	0.04 (0.02–0.06)	0	0
Maine	2	0	0 (0–0.84)	0	0
Michigan	31	0	0 (0–0.11)	0	0
Minnesota	1	0	0 (0–0.98)	0	0
Missouri	171	4	0.02 (0.01–0.06)	0	0
Mississippi	389	15	0.04 (0.02–0.06)	0	0
North Carolina	320	5	0.02 (0.01–0.04)	0	0
New Hampshire	5	0	0 (0–0.52)	0	0
New Jersey	5	0	0 (0–0.52)	0	0
New Mexico	176	2	0.01 (0–0.04)	0	0
Nevada	9	0	0 (0–0.34)	0	0
New York	28	1	0.04 (0–0.18)	0	0
Ohio	80	0	0 (0–0.05)	0	0
Oklahoma	677	30	0.04 (0.03–0.06)	0	0
Oregon	83	4	0.05 (0.01–0.12)	0	0
Pennsylvania	6	0	0 (0–0.46)	0	0
South Carolina	402	10	0.02 (0.01–0.05)	0	0
Tennessee	170	5	0.03 (0.01–0.07)	0	0
Texas	1285	53	0.04 (0.03–0.05)	0	0
Utah	5	0	0 (0–0.52)	0	0
Virginia	94	1	0.01 (0–0.06)	0	0
Wisconsin	4	0	0 (0–0.60)	0	0
West Virginia	47	0	0 (0–0.08)	0	0

*PEDV, porcine epidemic virus.

Technical Appendix Table 2. Feral swine samples sizes by yea, United States*

Year	No.	No. coronavirus-positive	Coronavirus seroprevalence (95% CL)	No. PEDV-positive	PEDV seroprevalence (95% CL)
2012	491	11	0.022 (0–0.037)	0	0
2013	2079	49	0.024 (0–0.011)	0	0
2014	2931	106	0.036 (0–0.002)	8	0.003 (0–0.005)
2015	2496	87	0.035 (0–0.011)	1	0 (0–0.002)

*PEDV, porcine epidemic virus.