Serologic Survey for Transmissible Gastroenteritis Virus Neutralizing Antibodies in Selected Feral and Domestic Swine Sera in the Southern United States

Authors: Woods, R. D., Pirtle, E. C., Sacks, J. M., and Gibbs, E. P. J.

Source: Journal of Wildlife Diseases, 26(3) : 420-422

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-26.3.420

Journal of Wildlife Diseases, 26(3), 1990, pp. 420–422

Serologic Survey for Transmissible Gastroenteritis Virus Neutralizing Antibodies in Selected Feral and Domestic Swine Sera in the Southern United States

R. D. Woods,[1] E. C. Pirtle,[1] J. M. Sacks,[1] and E. P. J. Gibbs,[2] [1] U.S. Department of Agriculture, Agricultural Research Service, National Animal Disease Center, P.O. Box 70, Ames, Iowa 50010, USA; [2] Department of Infectious Diseases, College of Veterinary Medicine, University of Florida, Gainesville, Florida 32612, USA

ABSTRACT: Serum samples collected from feral and domestic swine (*Sus scrofa*) in Florida and feral swine in Georgia and Texas were assayed by plaque reduction for their virus neutralizing (VN) antibodies against the porcine transmissible gastroenteritis virus (TGE). None of 560 samples collected from feral swine contained VN antibodies for TGE virus, but experimentally infected feral swine seroconverted. None of 665 samples from domestic swine contained TGE-VN antibodies. These results indicate feral swine are not a significant reservoir for TGE virus in southern states, but are capable of becoming infected and developing VN antibodies against TGE.

Key words: Reservoir, antibodies, transmissible gastroenteritis virus, feral swine, serosurvey, *Sus scrofa*.

Feral swine (*Sus scrofa*) can be found throughout the southern United States as free-ranging populations. Serologic surveys of feral swine sera from several areas demonstrate they may contain antibodies against various pathogens (Clark et al., 1983; Corn et al., 1986). In many areas, feral swine may come in contact with domestic swine that were vaccinated with a modified-live porcine transmissible gastroenteritis (TGE) virus, pigs that have recovered from TGE or perhaps even in contact with clinically affected pigs. However, no references were found on the prevalence of virus neutralizing (VN) antibodies to TGE virus in feral swine sera. While the complete host range of TGE virus in nature is unknown, it has been shown to survive in dogs and foxes (Haelterman, 1962), starlings (Pilchard, 1965) and house flies (Gough and Jorgenson, 1983). In addition, swine experimentally infected with TGE virus have been shown to harbor the virus in their respiratory tract for 104 days postexposure (Underdahl et al., 1975). The purpose of this report is to present the results of a serologic survey conducted on sera collected from feral swine in Texas, Florida, and Georgia and domestic swine in Florida.

Venous blood was taken from 262 feral swine from three locations in Florida, 184 feral swine on Ossabaw Island, Georgia, and 114 feral swine from 12 locations in Texas (USA). In addition, serum samples were taken from 665 domestic swine from the same general areas the feral swine were captured in Florida. These samples were collected from nine different locations and none of the swine were known to be exposed to TGE.

After collection, blood was allowed to clot, centrifuged at 1,500 *g* and the serum removed. Sera were heat inactivated (56 C for 30 min) and diluted from 1:2 through 1:1,024 in serum-free Eagle's minimum essential medium (MEM) with Earle's additives (GIBCO Laboratories, Grand Island, New York 14072, USA). The diluted samples were mixed with an equal volume of Eagle's MEM with 1% bovine fetal calf serum containing approximately 100 plaque forming units (pfu) of the Miller strain of TGE virus (Linda Saif, FAHRP, 1680 Madison Avenue, Wooster, Ohio 44691, USA) and held at 37 C for 60 min. Following incubation, the serum-virus mixtures were inoculated onto 5-day-old swine testes cell cultures in 60 × 15 mm tissue culture dishes and allowed to adsorb for 60 min at 37 C in a humidified CO_2 incubator. After incubation, the cell monolayer was overlaid with 0.5% agar in Eagle's serum free-MEM. The dishes were incubated at 37 C for 2 days, fixed with methanol and stained with 0.1% crystal

violet. The viral plaques were counted, and the 50% virus neutralization (VN) end point titer was calculated and recorded. Virus neutralizing titers of 1:4 or greater were considered positive.

Two 5-mo-old feral swine were inoculated with 3×10^3 pfu of virulent Miller TGE virus via stomach tube. Twenty-four hr later two additional 5-mo-old feral pigs were added as contact controls.

None of 560 serum samples collected from feral swine contained detectable levels of VN antibodies for TGE virus. Clinical signs of TGE were not reported among captured and released feral swine at any location.

Serum samples collected from 665 domestic swine in Florida were also seronegative for TGE-VN antibodies. Clinical signs of TGE were never reported in any of the herds.

All pigs in the TGE susceptibility study were seronegative for TGE antibody on post-exposure (PE) day 0; by 14 days PE three of the four had seroconverted and by 28 days PE all four pigs had a VN titer of $\geq$1:8 (Table 1). None of the pigs had clinical signs of TGE during the test.

Although none of the serum samples from 560 feral swine in Florida, Texas and Georgia had VN antibodies for the TGE virus, there is no evidence to suggest they may be resistant to a TGE infection. These swine probably never received an infective dose of TGE virus adequate to stimulate antibody production. This idea is supported by the fact that when they were either given an infective dose of virulent virus or mixed with pigs that were clinically affected with the disease, they seroconverted with the production of VN antibodies for TGE virus. Thus, not only are they capable of being infected, they are also capable of spreading the virus to susceptible pigs.

In addition to the feral pigs, serum samples collected from domestic swine in Florida also were seronegative for TGE antibodies. Lack of VN antibodies for TGE in both feral and domestic swine indicates the virus is not common in this part of the United States. This may not be unusual because most coronaviruses are very heat labile, and thus, they may not survive well in the environment under the climatic conditions in this region of the United States. Therefore, feral swine are unlikely to be a significant reservoir of the disease for dissemination to uninfected domestic swine in nearby herds or being transported to other swine populations.

TABLE 1. Virus neutralizing antibody titers in feral swine given virulent transmissible gastroenteritis virus and contact controls.

Days post exposure	VN titer			
	Pig 32	Pig 33	Pig 34	Pig 35
0	<1:4	<1:4	<1:4	<1:4
14	1:128	1:128	<1:4	1:8
28	1:128	1:128	1:8	1:32

[a] Pig given 3×10^3 pfu of virulent Miller strain of TGE virus.

Serological evidence indicates feral pigs are probably a reservoir for maintaining vesicular stomatitis, pseudorabies, brucellosis and leptospirosis in the southern states (Clark et al., 1983; Corn et al., 1986). Antibodies against the following viral infections also have been reported: porcine enterovirus, Venezuelan equine encephalitis, reovirus, parvovirus, influenza and hemagglutinating encephalomyelitis (Beran, 1990). Serological evidence has demonstrated that feral pigs have antibodies for other rickettsial and bacterial infections and at least 25 endoparsites and seven ectoparasites have been observed on these pigs (Beran, 1990). While none of the feral pigs in this limited survey were identified as having antibody for TGE virus, they were fully susceptible to the virus and were capable of transmitting it to other feral pigs. For these reasons, it is important to minimize exposure of domestic swine to feral pigs. Thus, relocation and movement of infected but apparently normal feral swine should be limited to reduce the chances for dissemination of various disease agents to man and commercial swine

herds that may cause health and economic problems.

LITERATURE CITED

BERAN, G. W. 1990. Feral swine and disease. Proceedings of the Annual Meeting of the U.S. Animal Health Association 93: In press.

CLARK, R. K., D. A. JESSUP, D. W. HIRD, R. RUPPANNER, AND M. E. MEYERS. 1983. Serological survey of California wild hogs for antibodies against selected zoonotic disease agents. Journal of the American Veterinary Medical Association 183: 1248–1251.

CORN, J. L., P. K. SWIDEREK, B. O. BLACKBURN, G. O. ERICKSON, A. B. THIERMANN, AND V. F. NETTLES. 1986. Survey of selected diseases in wild swine in Texas. Journal of the American Veterinary Medical Association 189: 1029–1032.

GOUGH, P. M., AND R. D. JORGENSON. 1983. Identification of porcine transmissible gastroenteritis virus in house flies (*Musca domestica* Linneaus). American Journal of Veterinary Research 44: 2076–2082.

HAELTERMAN, E. O. 1962. Epidemiological studies of transmissible gastroenteritis of swine. Proceedings of the U.S. Livestock Sanitation Association 66: 305–315.

PILCHARD, E. L. 1965. Experimental transmission of transmissible gastroenteritis virus by starlings. American Journal of Veterinary Research 26: 1177–1179.

UNDERDAHL, N. R., C. A. MEBUS, AND A. TORRES-MEDINA. 1975. Recovery of transmissible gastroenteritis virus from chronically infected experimental pigs. American Journal of Veterinary Research 36: 1473–1476.

Received for publication 6 October 1989.

Journal of Wildlife Diseases, 26(3), 1990, p. 422

BOOK REVIEW . . .

Mechanisms of Viral Toxicity in Animal Cells, L. Carrasco. CRC Press, Boca Raton, Florida 33431, USA. 1987. 196 pp. $124.95 U.S.

Viral pathogenesis, the mechanisms that underlie the production of disease by viral agents, was an area of research for many years where most answers were elusive. This was due in part to the lack of "clean cut" experiments, capable of showing what specific consequences viral infection had in the complexity of the cellular environment. Many attempts to determine the mechanisms of disease production by viral agents were hampered by the limitations of the techniques available. However, during the last decade, technological advances in the fields of biochemistry, biophysics, molecular biology and immunology have allowed a much better understanding of the pathogenesis of viral diseases. The current proliferation of scientific papers in this area, and the variety of journals in which they are published, makes it difficult to keep up with the latest developments in this fast moving field.

Mechanisms of Viral Toxicity in Animal Cells by L. Carrasco presents a series of excellent reviews on the specific subject of viral pathogenesis in animal cells. The "general overview" in chapter one states very well the scope of the book. The remaining six chapters deal with the mechanisms of penetration of cells, inhibition of host transcription and protein synthesis, mechanisms of cell toxicity by different families of DNA and RNA viruses and interferon effects on specific functions of infected cells.

Chapter two illustrates the technical problems underlying studies of virus attachment and penetration of cells. It also reviews recent data on the events that lead to viral penetration and uncoating. In chapter three, a series of works on suppression of host transcription by different viruses is presented. Chapters four through six present specific examples of inhibition of host protein synthesis by DNA viruses, cell killing by RNA viruses and regulation of translation by picornaviruses. Finally, chapter seven presents a review of interferon action on cells infected with several DNA and RNA viruses. All chapters contain data to support the proposed mechanisms of pathogenesis, but also present different points of view and interpretations of the data. This particular presentation makes this book an ideal reference for graduate-level viral pathogenesis courses or seminars. The book also should appeal to virologists studying viral pathogenesis, since it discusses areas not present in other texts.

The amount of specific information and reference material summarized in this book makes it an excellent source of information for scientists studying viral diseases. The book provides a good, current view of the mechanisms of viral toxicity in animal cells.

Luis L. Rodriguez, Tropical Disease Research Program, Universidad Nacional, P.O. Box 304-3000, Heredia, Costa Rica.