A FIELD STRAIN OF HOG CHOLERA VIRUS WITH VARIANT-LIKE CHARACTERISTICS

W. C. Stewart*, M. L. Snyder, and J. I. Kresse
NVSL, P. O. Box 844, Ames, Iowa 50010

Hog cholera (HC) viruses, which differed from other known HC viruses by their antigenic and immunological characteristics, were first reported in the United States in 1951 (Dale, et al.). These viruses were implicated in losses in farm herds when they were used in conjunction with certain hyperimmune serums in the simultaneous serum-virus treatment for immunization against HC. Identification of a variant virus was based on the observation that when pigs were inoculated with 2 ml of variant virus and a normally protective dose of HC antiserum, typical HC resulted; however, pigs inoculated with the same dose of this antiserum and 2 ml of regular virus were fully protected.

After this initial episode, the variant virus issue became an event of historical interest; however, approximately 20 years later during the latter stages of the HC eradication program, the problem recurred, but to a lesser degree. Hog cholera was confirmed on several Iowa farms in pigs newly arrived from Arkansas. Prior to interstate movement, the pigs had been assembled at an Arkansas sale barn, where they were administered HC antiserum. Since the pigs contracting HC had been given antiserum, the question naturally arose as to whether the virus implicated in this incident was a variant strain. Special testing by the Veterinary Biologics Division disclosed that the antiserum given to the Arkansas pigs had a HC neutralization (Nt) titer of 1:2,455.

Tests performed at the National Veterinary Services Laboratories disclosed that a special antiserum, Bureau of Animal Industry's (BAI) experimental serum #1, did not protect pigs against the isolate (designated Iowa strain) when it was given in a quantity that should have protected against a regular virus. A protracted infection occurred in two pigs inoculated with the Iowa strain and HC antiserum; the pigs died on postinoculation days (PID) 36 and 96, respectively. Two pigs given the Iowa strain without HC antiserum succumbed on PID 11 and 29. Hog cholera virus replicated to high titer, $\geq 10^4$ plaque-forming units (PFU), in the blood, spleen, and lymph nodes of all four pigs. High virus titers, $\geq$5,000 PFU, were also present in nonlymphoid tissues, i.e., epididymus, kidney, liver, lung, salivary gland, sheath, and testicle of the pig given serum and virus that lived 96 days.

Following this disclosure, comparative tests were performed in pigs with the standard virulent challenge virus (Ames strain) and the Iowa strain. Pigs of comparable weight (average 13.6 kg) in two groups of three each were administered 2 ml of blood virus (5.75×10^5 $TCID_{50}$/ml) prepared from either the Ames or Iowa strain along with either 4, 16, or 32 ml of the BAI experimental serum #1. The course of infection was followed in each pig and immunofluorescent studies were performed on blood and tissue specimens (Mengeling, et al., 1963; Stair, et al., 1963). Virus Nt tests were performed *in vitro* with immune serum prepared against each strain (Carbrey, et al., 1969).

Of the three pigs given 4 ml of BAI serum #1 and 2 ml of the Iowa strain, two died, one acutely (PID 16) and one chronically (PID 54). The course of infection in the latter pig was marked by frequent periods of remission of clinical signs. The third pig had an acute reaction, but recovered and appeared healthy when euthanatized on PID 69. Persistent viremia was a consistent finding in the latter two pigs. Hog cholera virus of low titer (<100 PFU/ml) was isolated from blood specimens as late as PID 42 and HC virus of high titer ($\geq$5,000 PFU/ml) was recovered from splenic tissues collected at necropsy.

Of the three pigs given the Ames virus and 4 ml of serum, one developed a debilitative, chronic infection and died PID 44. On PID 9 and thereafter, high virus titers were demonstrable in this pig. One of the two remaining pigs had a very severe reaction, but later recovered. Hog cholera virus of low titer was isolated from this pig on PID 9 and 12. The third pig had a very slight reaction and HC virus was never isolated from it. The latter two pigs developed moderately high antibody titers (1:256) and were euthanatized on PID 69.

When each virus was likewise administered with 16 ml of BAI serum #1, the 3 pigs receiving Ames virus remained healthy and by PID 48 had developed Nt titers of 1:64 (1) and 1:1,024 (2), respectively. However, the 3 pigs receiving the Iowa strain became acutely ill. One pig died on PID 9 following collection of a blood specimen, and a second pig was found dead on PID 14. Very low HC virus titers (<10 PFU) were isolated from blood specimens of these pigs on PID 9 as well as from splenic tissues at necropsy. The third pig returned to an apparently healthy state after a protracted illness of 23 days, but it remained persistently infected with HC virus until euthanatized on PID 57.

Subsequently, three pigs given the Iowa strain of HC virus and 32 ml of BAI serum #1 remained healthy and HC virus was not isolated from them.

A pig inoculated with inactivated virus of the Iowa strain developed a low antibody titer (1:16). When this antiserum was reacted with the Iowa strain in an indirect fluorescent antibody test, immunofluorescence of diminished intensity occurred compared to that observed with the Ames strain and homologous antiserum. Also, when reciprocal cross-Nt tests were performed with type specific antiserums, strains Ames and Iowa were neutralized to a greater degree by homologous than by heterologous antiserums (Table 1). These antigenic differences were consistent with results of the pig inoculation tests, and were similar to "Antigenic Differences In Two Hog Cholera Virus Strains" reported by Pirtle and Mengeling (1971).

In conclusion, 32 ml of BAI serum #1 was required to protect pigs against the Iowa strain, whereas 16 ml of serum protected pigs against the Ames strain. Pigs were not protected against either strain with 4 ml of serum. Whereas occasionally pigs died acutely from the Iowa strain, the salient clinical feature was one of chronic infection characterized by persistent viremia. Properties of poor immunogenicity and diverse antigenic composition were properties of the Iowa strain that contributed to the HC epizootic in pigs given HC antiserum and moved from Arkansas to Iowa.

Table 1. Hog Cholera Neutralizing Antibody Titers (log_{10}):

	Antiserum	
Virus	Iowa	Ames
Iowa	1.2	3.6
Ames	0.6	3.9

Selected references: Dale, CN, et al.: J Am Vet Med Assoc 1951, 118:279; Mengeling, WL, et al.: Can J Comp Med 1963, 27:249; Stair, EL, et al.: Proc Soc Exp Biol Med 1963, 113:656; Carbrey, EA, et al.: J Am Vet Med Assoc 1969, 155:2201; Pirtle, EC, and Mengeling, WL: Am J Vet Res, 1971, 32:1473.