

HOG CHOLERA (Note: The preferred term for this disease is now classical swine fever.)

(Classical swine fever, peste du porc, colera porcina, Virusschweinepest)

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Definition [top](#)

Hog cholera (HC) is a highly contagious viral disease of swine that occurs in an acute, a subacute, a chronic, or a persistent form. In the acute form, the disease is characterized by high fever, severe depression, multiple superficial and internal hemorrhages, and high morbidity and mortality. In the chronic form, the signs of depression, anorexia, and fever are less severe than in the acute form, and recovery is occasionally seen in mature animals. Transplacental infection with viral strains of low virulence often results in persistently infected piglets, which constitute a major cause of virus dissemination to noninfected farms.

Etiology [top](#)

Although minor antigenic variants of hog cholera virus (HCV) have been reported, there is only one serotype. Hog cholera virus is a lipid-enveloped pathogen

belonging to the family Flaviviridae, genus *Pestivirus*. The organism has a close antigenic relationship with the bovine viral diarrhea virus (BVDV) and the border disease virus (BDV), as demonstrated in the immunodiffusion and immunofluorescence tests. The serum neutralization test can, however, differentiate between HCV and BVDV. In a protein-rich environment, HCV is very stable and can survive for months in refrigerated meat and for years in frozen meat. The virus is sensitive to drying (desiccation) and is rapidly inactivated by a pH of less than 3 and greater than 11.

Host Range [top](#)

The hosts of HCV are the pig and wild boar.

Geographic Distribution [top](#)

According to the FAO—WHO—OIE Animal Health Yearbook 1989, HC is recognized in 36 countries and is suspected of being present in another 2. The disease has been eradicated in Australia, Canada, and the United States. Constant progress toward eradication has been made in the countries of the European Economic Community since the guidelines for HC control in individual member states were accepted in 1980.

Transmission [top](#)

The pig is the only natural reservoir of HCV. Blood, tissues, secretions and excretions from an infected animal contain HCV. Transmission occurs mostly by the oral route, though infection can occur through the conjunctiva, mucous membrane, skin abrasion, insemination, and percutaneous blood transfer (e.g., common needle, contaminated instruments). Airborne transmission is not thought to be important in the epizootiology of HC, but such transmission could occur between mechanically ventilated units within close proximity to each other.

Introduction of infected pigs is the principal source of infection in HC-free herds. Farming activities such as auction sales, livestock shows, visits by feed dealers, and rendering trucks are also potential sources of contagion. Feeding of raw or insufficiently cooked garbage is a potent source of HCV. During the warm season, HCV may be carried mechanically by insect vectors that are common to the farm environment. There is no evidence, however, that HCV replicates in invertebrate vectors. Husbandry methods also play an important role in HC transmission. Large breeding units (100 sows) have a higher risk of recycling infection than small herds. In large breeding units where continuous farrowing is practiced, strains of low virulence may be perpetuated indefinitely until the cycle is interrupted by

stamping-out procedures and a thorough cleaning and disinfection are carried out.

Incubation Period [top](#)

The incubation period is usually 3 to 4 days but can range from 2 to 14 days.

Clinical Signs [top](#)

The clinical signs of HC are determined by the virulence of the strain and the susceptibility of the host pigs. Virulent strains cause the acute form of the disease, whereas strains of low virulence induce a relatively high proportion of chronic infections that may be inapparent or atypical. These strains are also responsible for the "carrier-sow" syndrome from which persistently infected piglets are produced.

Acute Hog Cholera

In acute HC, the pigs look and act sick. Their disease progresses to death within 10 to 15 days, and remissions are rare. In an affected herd, some pigs will become drowsy and inactive and will stand with arched backs. Other pigs will stand with drooping heads and straight tails. Some pigs may vomit a yellow fluid containing bile. The sick pigs will huddle and pile up on each other in the warmest corner of the enclosure and will rise only if prompted vigorously. Anorexia and constipation will accompany a high fever that may reach 108° F (42.2° C) with an average of 106° F (41.1° C). Pigs may continue to drink and may have diarrhea toward the end of the disease process. Conjunctivitis (Fig. [65](#)) is frequent and is manifested by encrustation of the eyelids and the presence of dirty streaks below the eyes caused by the accumulation of dust and feed particles. Sick pigs become gaunt and have a weak, staggering gait related to posterior weakness. In terminal stages, pigs will become recumbent, and convulsions may occur shortly before death. In the terminal stage, a purplish discoloration of the skin may be seen; if present, the lesions are most numerous on the abdomen and the inner aspects of the thighs.

Chronic Hog Cholera

Chronic HC is characterized by prolonged and intermittent disease periods with anorexia, fever, alternating diarrhea and constipation, and alopecia. A chronically infected pig may have a disproportionately large head relative to the small trunk. These runt pigs may stand with arched backs and their hind legs placed under the body. Eventually, all chronically infected pigs will die.

Congenital Hog Cholera

Congenital HCV infection by virulent strains will likely result in abortions or in the birth of diseased pigs that will die shortly after birth. Transplacental transmission with low-virulence strains may result in mummification, stillbirth, or the birth of weak and "shaker" pigs. Malformation of the visceral organs and of the central nervous system occurs frequently. Some pigs may be born virtually healthy but persistently infected with HCV. Such infection usually follows exposure of fetuses to HCV of low virulence in the first trimester of fetal life. Pigs thus infected do not produce neutralizing antibodies to HVC and have a lifelong viremia. The pigs may be virtually free of disease for several months before developing mild anorexia, depression, conjunctivitis, dermatitis, diarrhea, runting, and locomotive disturbance leading to paresis and death. In breeding herds affected with lowvirulence strains of HCV, poor reproductive performance may be the only sign of disease.

Gross Lesions [top](#)

Acute Hog Cholera

The most common lesion observed in pigs dying of acute HC is hemorrhage. Externally, a purplish discoloration of the skin is the first observation. There may be necrotic foci in the tonsils (Fig. 66). Internally, the submandibular and pharyngeal lymph nodes are the first to be affected and become swollen owing to edema and hemorrhage. Because of the structure of the pig lymph node, hemorrhages are located at the periphery of the node (Fig. 67). As the disease progresses, the hemorrhage and edema will spread to other lymph nodes. The surface of the spleen, and particularly the edge of the organ, may have raised, dark wedge-shaped areas. These are called splenic infarcts. Infarcts are frequently observed in pigs infected experimentally with older strains of HCV but are less commonly seen with the contemporary strains (Fig. 68).

Pinpoint to ecchymotic hemorrhages on the surface of the kidney are very common in HC (Fig. 69). Such lesions are easier to see in the decapsulated kidney. Hemorrhages are also found on the surface of the small and large intestine (Fig. 70), the larynx, the heart, the epiglottis, and the fascia lata of the back muscles. All serous and mucosal surfaces may have petechial or ecchymotic hemorrhages.

Accumulation of straw-colored fluids in the peritoneal and thoracic cavities and in the pericardial sac may be present.

The lungs are congested and hemorrhagic and have zones of bronchopneumonia.

Chronic Hog Cholera

In chronic HC, the lesions are less severe and are often complicated by secondary bacterial infections. In the large intestine, button ulcers are an expression of such a secondary bacterial infection. In growing pigs surviving for more than 30 days, lesions may be seen at the costochondral junction of the ribs and at the growth plates of long bones.

Congenital Hog Cholera

In pigs infected transplacentally with HCV strains of low virulence, the most commonly seen lesions are hypoplasia of the cerebellum, thymus atrophy, ascites, and deformities of the head and of the limbs. Edema and petechial hemorrhages of the skin and of the internal organs are seen at the terminal stage of the disease.

Morbidity and Mortality [top](#)

In acute HC, the morbidity and mortality are high.

Diagnosis [top](#)

Field Diagnosis [top](#)

Septicemic conditions in which pigs have high fever should be investigated carefully. A thorough history from the herd owner should be obtained to determine if raw garbage was fed, if unusual biological products were used, or if recent additions were made to the herd. Careful observation of the clinical signs and of the necropsy lesions should be recorded. In acute HC, it is helpful to necropsy four or five pigs to increase the probability of observing the representative lesions.

A marked leukopenia is detectable at the time of initial rise in body temperature and persists throughout the course of the acute and chronic disease. This feature was once widely used in the field diagnosis of HC. Nowadays, with the development of more specific laboratory diagnostic methods, which are aimed at demonstrating the virus or its structural antigens in tissues or at detecting specific antibodies in the serum, the white blood count is not as widely used. In endemic areas it could be helpful.

Specimens for the Laboratory [top](#)

For virus isolation and antigen detection, the tonsils are considered essential. In addition, submandibular and mesenteric lymph nodes, spleen, kidneys, and the distal part of the ileum should be collected. In live pigs, tonsil biopsies and whole blood collected with anticoagulants are useful to diagnose HC. Sample collection should be targeted to pigs having fever or showing other signs of the disease. Each sample of tissue should be placed in a separate plastic bag and identified. The samples should not be frozen (interference with fluorescent antibody tissue section test) but kept at refrigeration temperature. The material should be transported and stored in leak-proof containers in accordance with national regulations for transportation of diagnostic biologic samples.

Serum samples for antibody detection should be collected from animals that have recovered from suspected infection or from sows known to have been in contact with infected or suspected cases. A sufficient number of samples should be collected to ensure a high probability of detecting infection.

A complete set of tissues, including the whole brain, should be submitted in 10 percent buffered formalin.

Laboratory Diagnosis [top](#)

Any clinical diagnosis of HC must be confirmed by the submission of specimens to a specialized diagnostic laboratory that should also have the capability to distinguish between HC and African swine fever.

The laboratory diagnostic procedures for HC have evolved in parallel with the emergence of new technologies. Until the 1960's, laboratory diagnosis was restricted to recognition of gross lesions and confirmation by histopathology. Inoculation of susceptible pigs was often used as final confirmatory test and to determine the virulence of the viruses. Numerous laboratory techniques have been described to diagnose HC, but only a few have gained international acceptance and have been integrated into national HC control programs. Only these will be discussed in this presentation.

In the fluorescent antibody tissue section test (FATST), direct fluorescent antibody technique is applied to detect HC viral antigens in frozen tissues of organs from dead pigs, in biopsy material, or in impression smears. Theoretically, a diagnosis can be confirmed within hours from the reception of the specimen. In countries where the disease has been eradicated, the diagnosis of the "index case" by the FATST alone may be difficult, and confirmation in cell culture may be needed. The

FATST may not differentiate HC from BVDV infection; an accurate distinction between the two viruses has to be made before releasing a final diagnosis. Differentiation between HCV and BVDV can readily be made with the immunoperoxidase test using monoclonal antibodies or the serum neutralization test.

The isolation of HCV in cell culture and the identification using fluorescein-labeled hog cholera antibody (fluorescent antibody cell culture test) can provide confirmation in cases where the results of investigation of frozen tissue sections are inconclusive.

As control measures for HC are implemented in a country, virulent strains of HCV will be reduced, and there will be a relative increase of low-virulence strains. As the proportion of subclinical cases in a national herd increases, it will become increasingly difficult to recognize the disease. The antigen detection systems previously described become less effective; thus, serological tests are essential for a successful control and eventual eradication program.

Approximately 75 percent of pigs infected with acute HC have microscopic lesions of an encephalitis characterized by perivascular cuffing, endothelial proliferation, and microgliosis. This feature is easily recognized in a nonspecialized diagnostic laboratory and may constitute the most important single factor that will cause the pathologist to suspect HC.

Differential Diagnosis [top](#)

Differential diagnosis of HC should include African swine fever, erysipelas, salmonellosis, eperythrozoonosis, and salt poisoning.

Vaccination [top](#)

Over the years, numerous regimens of vaccination have been advocated with a variable degree of success. In the past two decades, modified live vaccines (MLV) with no residual virulence for pigs have become available. The lapinized Chinese (C) strain, the Japanese guinea pig cell culture-adapted strain, and the French Thiverval strain have been widely used. All three strains are considered innocuous for pregnant sows and piglets over 2 weeks old.

Control and Eradication [top](#)

In countries where HC is enzootic, a systematic vaccination program is effective in preventing losses. Experience in the United States and in some countries of the

European Union has proven that a strict regimen of vaccination will reduce the number of outbreaks to a level at which complete eradication by sanitary measure alone will be feasible. At that point, vaccination must be stopped. A successful eradication program requires a massive input of funds from a central government and cooperation from the government, the swine industry, and the veterinary profession. Eradication measures will be assisted by strictly enforcing the garbage cooking laws, having an effective swine identification system, and using serological surveys targeted primarily to breeding sows to detect subclinical infections.

In countries where HC has been eradicated and in which the threat of reintroduction is significant, it is essential to initiate an effective serological monitoring system. Sampling may be limited to strategic locations such as the border of an infected neighbor country or be intensified to target populations such as the garbage-fed herds. Such a system has been in effect in the United States since successful eradication in 1976; several thousand samples have been accessed annually.

Public Health [top](#)

Human beings are not susceptible to HCV infection.

GUIDE TO THE LITERATURE [top](#)

1. ANONYMOUS. 1989. FAO-WHO-OIE Animal Health Yearbook.
2. BALER, J. A., and SHEFFY, B. E. 1960 A persistent hog cholera viremia in young pigs. *Proc. Soc. Exp. Biol. Med.*, 105: 675-678.
3. CARBERY, E. A., ERICKSON, G.A., and METZ, C. A. 1984. Diagnosis of hog cholera. *Preventive Vet. Med.*, 2: 103-108.
4. CARBERY, E. A., STEWART, W. C., YOUNG, S. H., and RICHARDSON, G. C. 1966. Transmission of hog cholera by pregnant sows. *J. Am. Vet. Med. Assoc.*, 149: 23-30.
5. CHEVILLE N. F., and MENGLING, W. L. 1969. The pathogenesis of chronic hog cholera (swine fever). Histologic, immunofluorescent, and electron microscopic studies. *Lab. Invest.*, 20: 261-274.
6. EMERSON, J. L., and DELEZ, A. L. 1965. Cerebellar hypoplasia,

- hypomyeliogenesis, and congenital tremors of pigs associated with prenatal vaccination of sows. *J. Am. Vet. Med. Assoc.*, 147: 47-54.
7. EDWARDS, S., MOENNIG, V., and WENSWOORT, G. 1991. The development of an international reference panel of monoclonal antibodies for the differentiation of hog cholera virus from other pestiviruses. *Vet. Micro.*, 29: 101-108.
8. HANSON, R. P. 1957. Origin of hog cholera. *J. Am. Vet. Med. Assoc.*, 131; 211-218.
9. HOLM JENSEN, M. 1981. Detection of antibodies against hog cholera virus and bovine viral diarrhea virus in porcine serum. A comparative examination using CF, PLA, and NPLA assays. *Acta Vet. Scand.*, 22: 85-98.
10. JUBB, K. V. F., KENNEDY, P.C, and PALMER, N. 1985. Pathology of Domestic Animals. Vol. 3. San Diego:Academic Press, Inc. pp 66-67.
11. LIESS, B. 1981. Hog Cholera. In Virus Diseases of Food Animals, Vol. II: Disease Monographs, E. P. J. Gibbs, ed. New York:Academic Press. pp 627-650.
12. TERPSTRA, C., BLOEMRAAD and GIELKINS, A. L. J. 1987. The neutralizing peroxidase-linked assay for the detection of antibody against swine fever virus. *Vet. Micro.*, 9: 113-120.
13. TERPSTRA, C. 1990. Manual of Recommended Diagnostic Techniques and Requirements for Biological Products for List A & B Diseases. Office International des Epizooties Manual: Vol II, pp. 1/15-15/15.
14. VAN BEKKUM, J. G. 1977. Experience in the Netherlands with the Lapinized, So-called Chinese (C) Strain of Vaccine. Agri. Res. Semin. on Hog Cholera/classical Swine Fever and African Swine Fever. Hannover, Eur. 5904, pp 379-391.
15. VAN OIRSCHOT, J. T. and TERPSTRA, C. 1989. Hog Cholera Virus. In *Virus Infections of Porcines*. M. B. Pensaert, ed.; New York:Elsevier Science Publishers, pp113-130
16. VAN OIRSCHOT, J. T. 1986. Hog Cholera. In Diseases of Swine, 6th ed. Ames, IA:The Iowa State University Press, pp. 289-300.

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