Classical Swine Fever

Hog Cholera,
Swine Fever,
European Swine Fever,
Peste du Porc,
Colera Porcina,
Virusschweinepest

Last Updated: May 2025

Importance

Classical swine fever (CSF) is a highly contagious and economically significant viral disease of pigs. The severity of the illness varies with the strain of the virus, the age of the pig, and the immune status of the herd. Acute infections, which are caused by highly virulent isolates and have a high mortality rate in naive herds, are likely to be diagnosed rapidly. However, less virulent isolates, which are currently prevalent, cause illnesses that resemble other swine diseases and can be more difficult to recognize, particularly in older pigs.

Although classical swine fever was once found throughout much of the world, a number of countries have eradicated this disease from domesticated swine, and a few regions, such as North America, are free of CSF in all suids. Reintroduction of the virus to these areas can be devastating. In 1997-1998, an outbreak in the Netherlands spread to involve more than 400 herds and cost $2.3 billion to eradicate. Approximately 12 million pigs were killed, some in eradication efforts but most for welfare reasons associated with the epidemic. In addition, the virus sometimes becomes established in wild suids after an outbreak, complicating eradication efforts and resulting in an ongoing risk of reintroduction to domesticated pigs.

Etiology

Classical swine fever results from infection by classical swine fever virus (CSFV; official species name *Pestivirus C*), a member of the genus *Pestivirus* and family Flaviviridae. There is only one serotype but several genotypes and subgenotypes. Some closely related pestiviruses of ruminants or swine (e.g., bovine virus diarrhea virus, border disease virus, Linda virus) can cause serological reactions in pigs that may be mistaken for CSF.

Species Affected

CSFV seems to be capable of infecting most or all members of the pig family (Suidae). Naturally acquired infections are known to occur in domesticated pigs (*Sus scrofa domestica*), Eurasian wild boar (*Sus scrofa scrofa*), white-lipped peccaries (*Tayassu pecari*) and collared peccaries (*Pecari tajacu*), and experimental infections have been established in common warthogs (*Phacochoerus africanus*) and bush pigs (*Potamochoerus larvatus*).

There are also reports of experimental infections in cattle, sheep, goats and deer, without clinical signs, and some recent descriptions of viruses that appear to be CSFV in naturally infected cattle. Some strains of CSFV can be adapted to passage in rabbits.

Zoonotic potential

There is no evidence that CSFV infects humans.

Geographic Distribution

Classical swine fever is endemic in parts of Asia, Africa, and South and Central America, including some islands in the Caribbean. While many countries have eradicated this virus from domesticated pigs, only some locations, such as the U.S., Canada, New Zealand, Australia, Iceland and some European nations, are also CSF-free in wild and feral suids.

Transmission

Pigs are mainly thought to become infected with CSFV by inhalation or ingestion; however, it may also enter the body via other mucus membranes (including genital transmission in semen), and skin abrasions. Pigs can shed this virus in oronasal and ocular secretions, urine, feces and semen. Shedding can begin before the onset of clinical signs. Because CSFV can persist in blood and tissues after death, it is readily transmitted by feeding uncooked swill that contains tissues from infected pigs. Aerosol transmission has been demonstrated experimentally with some viral strains, but is most likely to occur between nearby pens or when there are large concentrations of infected animals and mechanically ventilated buildings in close proximity.

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Piglets infected before birth or shortly thereafter (at least up to 3 weeks of age) sometimes become persistently infected, and can excrete the virus continuously or intermittently as long as they survive, which may be several months or more. These animals are immunotolerant to CSFV and do not produce antibodies to this virus. Preliminary evidence suggests that certain live attenuated vaccine viruses that have been shown to cause reproductive losses in pregnant sows might also be capable of establishing persistent infections in piglets.

Virus transmission on fomites can be important in introducing CSFV to uninfected regions or farms. This virus can survive for particularly long periods in porcine tissues. It has been reported to remain infectious for nearly 3 months (and possibly longer) in refrigerated meat and for more than 4 years in frozen meat. Reported virus survival times in various cured and smoked meats vary with the technique, and range from 17 days to more than 6 months. In tissues from infected pigs kept between 25ºC (77°F) and 68ºC (154°F), CSFV remained viable longer in serum than muscle, fat or lymph nodes. It survived longer in meat than lymph nodes at room temperature (25ºC), although the lymph nodes initially contained more virus.

Estimates of CSFV persistence in other environments vary, and can be influenced by factors such as the initial concentration of the virus, the presence of organic matter and the temperature, with longer virus survival in the cold. Some studies suggest that, at room temperature (e.g., 20ºC/ 68°F), this virus is inactivated within a few days to 2 weeks in feces (or slurry) and urine, as well as on certain fomites; however, in one report, it remained viable in pig slurry for at least 70 days at 17ºC (63°F). Other experiments describe virus survival for 1-3 months at 4-5ºC (39-41°F) when protected by material such as pig slurry. A study that used an environmental chamber, programmed to simulate conditions during a winter shipment of 37 days, found that residual CSF viruses in sterilized, virus-spiked soybean meal and pork sausage casings were able to infect pigs at the end of this time, though the virus was inactivated much sooner in some items such as dry dog food. The presence of other microorganisms in the environment can sometimes greatly reduce pathogen survival, and the viability of CSFV in unsterilized feed ingredients under transport conditions remains to be determined.

Disinfection

CSFV is susceptible to a number of disinfectants including sodium hypochlorite, phenolic compounds, detergents, organic solvents, quaternary ammonium compounds and aldehydes (formaldehyde, glutaraldehyde). It is also sensitive to drying, heat and ultraviolet light. This virus is reported to be destroyed by heating for a minute or less at 90-100ºC or 5 minutes at 70°C (158°F). In meat, it can be inactivated by a temperature of 65.5ºC (150°F) or higher, maintained for 30 minutes. CSFV is relatively stable at pH 5-10, but inactivated by pH $\leq$ 3 or pH > 10. Inactivation by acidic conditions occurs more rapidly at room temperature than in the cold, with its mean half-life reported to be 70 hours at 4°C but 5 hours at 21°C (70°F) at pH 3.

Incubation Period

The incubation period can range from 2 to 15 days, but it is most often 3-7 days in acute cases. Under field conditions, the disease may not become evident in a herd for 2 to 4 weeks or more.

Clinical Signs

The clinical signs vary with the strain of CSFV and the age and susceptibility of the pigs. While highly virulent strains were prevalent in the past, most outbreaks are now caused by moderately virulent strains, and the signs are often less severe and distinctive. Secondary infections can complicate the course of the illnesses and affect the clinical presentation, particularly with less virulent strains.

Highly virulent strains of CSFV tend to cause acute, severe illnesses in naive herds. Common clinical signs in these animals are a high fever, nonspecific signs such as huddling, weakness, drowsiness and anorexia, and conjunctivitis, which may cause severe crusting of the eyelids. Constipation, with the passage of hard fecal pellets, is typically followed by, or intermittent with, watery diarrhea. Some animals may be incoordinated or exhibit an unsteady, weaving or staggering gait, which often progresses to posterior paresis. Pigs may also vomit yellow, bile-containing fluid or develop respiratory signs. The skin often becomes hyperemic and may develop hemorrhages (especially on the abdomen, inner thighs and ears) or a purple cyanotic discoloration, which tends to be seen on the snout, ears and tail. Severe leukopenia is a common laboratory abnormality. Pigs with acute classical swine fever often die within 1-3 weeks, and convulsions may occur in the terminal stages.

The subacute form of CSF, which can be seen in herds with partial immunity or naive herds infected with less virulent strains, has a more prolonged course, with similar but less severe clinical signs and a lower mortality rate. Older animals often have milder illnesses than young pigs in this form, and many animals may have only nonspecific signs of illness. Neurological signs may be limited to piglets, which are usually the most severely affected group. Reproductive losses and sick piglets can be the only sign of the disease in some breeding herds.

Pregnant animals infected with CSFV often abort or give birth to stillborn, mummified or weak piglets. Some offspring may have a congenital tremor and/or congenital malformations of the visceral organs and central nervous system. Some piglets exposed during gestation or the first few weeks of life can also become persistently infected with this virus. These animals sometimes have clinical signs at birth; however, many are asymptomatic and only become ill after several months, with signs of “late onset” disease such as inappetence, depression, stunted growth, dermatitis, diarrhea, conjunctivitis, ataxia or posterior paresis. Although persistently infected pigs can survive for 2 months or more, all typically die within a year.

Chronic CSF, which may affect only a few animals in the herd, can be seen with less virulent strains of the virus or in partially immune herds. Chronically infected individuals, most of which are juveniles, initially have typical CSF signs such as anorexia, depression, elevated temperature, leukopenia, and periods of constipation and/or diarrhea, and usually improve after several weeks, but subsequently develop recurrent signs. These pigs may also be stunted or thin, and sometimes develop alopecia and skin lesions, while immunosuppression from the infection may lead to concurrent illnesses from other agents. The clinical signs in these animals can wax and wane for weeks to months, and the outcome is often fatal.

Clinical signs in wild boar appear to be similar to those in domesticated pigs. Experimentally infected bushpigs also became ill and some cases were severe, with fever, anemia, diarrhea and conjunctivitis, as well as a prolonged clotting times upon blood collection. Most experimentally infected warthogs in the same study did not develop clinical signs, despite virological and serological evidence of infection, though one animal had moderate to severe diarrhea. A short period of fever and nonspecific signs were reported in experimentally infected collared peccaries.

Post Mortem Lesions

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The lesions of classical swine fever are highly variable and may be absent or inconspicuous in animals that die peracutely. During outbreaks, the likelihood of observing the typical necropsy lesions is better if four or five pigs are examined.

Many carcasses have evidence of severe tonsillitis, sometimes with necrotic foci, and swollen and hemorrhagic lymph nodes, while cyanotic and/or hemorrhagic discoloration of the skin may be seen in some animals with acute, severe CSF. Petechial or ecchymotic hemorrhages are often apparent in the subcutaneous tissues and on the serosal and mucosal surfaces of various internal organs, including the kidney, urinary bladder, epicardium, epiglottis, larynx, trachea, intestines and spleen. The stomach sometimes contains hemorrhagic lesions, while mild to moderate catarrhal enteritis may be noted in the small intestine and button ulcers are sometimes found in the colon. Straw–colored fluid can be present in the peritoneal and thoracic cavities and the pericardial sac, and the lungs may be congested and hemorrhagic. Splenic infarcts (raised, dark, wedge-shaped lesions) are a characteristic lesion in severe CSF, but are seen only occasionally with the currently circulating strains. Some congenitally infected piglets may have cerebellar hypoplasia, thymic atrophy and deformities of the head and legs, as well as hemorrhagic lesions and ascites.

The lesions in chronic cases are less severe and may be complicated by secondary infections. Necrotic foci or "button" ulcers are common in the intestinal mucosa, epiglottis and larynx of these animals. Button ulcers in the intestine may be followed by diffuse, diphteroid-necrotizing enteritis. In growing pigs that have survived for more than a month, there may also be bone lesions at the costochondral junction of the ribs and the growth plates of the long bones.

Diagnostic Tests

CSFV, its nucleic acids and antigens can be detected in blood or tonsil swabs collected from live animals, or in tissue samples (tonsils, pharyngeal and mesenteric lymph nodes, spleen, kidneys, distal ileum) taken at necropsy. Samples from live animals should be collected when they are febrile. Viremia may be brief in animals infected with less virulent strains. Sampling of oral fluids via ropes hung in pigpens is under investigation as a potential method of surveillance.

Reverse transcriptase polymerase chain reaction (RT–PCR) tests are often used to diagnose clinical cases. Loop-mediated isothermal amplification (RT-LAMP) assays to detect nucleic acids have also been published. Viral antigens are often identified with direct immunofluorescence or ELISAs, but other tests, including rapid immunochromatographic assays, are also available in some locations. Cross-reactivity with some other pestiviruses can be an issue in antigen-detection tests, particularly ELISAs, which have low sensitivity and are only considered suitable as herd tests. CSFV can be isolated in several cell lines including PK–15 and SK-6 cells, with virus identification by methods such as immunostaining or RT-PCR. Identification of the viral genotype or subgenotype, using various genetic methods, can be useful in epidemiology.

Serology is also used in diagnosis and surveillance; however, antibodies do not usually become detectable until 2-3 weeks after exposure. Persistently infected piglets are seronegative, and titers may be low or absent in some chronically infected animals, probably due to the sequestration of antibodies in immune complexes. The most commonly used serological tests are virus neutralization assays and various ELISAs, but other tests, including rapid immunochromatographic (lateral flow) assays, are also used in some countries. Cross-reactivity to certain bovine or porcine pestiviruses (e.g.., bovine virus diarrhea virus, border disease virus, Linda virus), can be an issue in some serological tests; however, there reactions can be distinguished by comparative serum neutralization.

Treatment

There is no treatment for classical swine fever, other than supportive care.

Control

Disease reporting

Veterinarians who encounter or suspect classical swine fever should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

In countries where classical swine fever is endemic, this disease may be excluded from a herd by buying animals from CSFV-free herds, quarantining the new stock for 4 months

and testing them before adding them to the herd. Vaccines can protect animals from clinical signs, and may also be employed to reduce the prevalence of infections during an eradication program. Modified live, chimeric pestivirus and subunit vaccines may be available, depending on the location. Some of these vaccines have companion serological tests that can distinguish vaccinated from infected animals. Persistently infected piglets do not respond to vaccination and remain seronegative.

CSF-free regions are protected by import restrictions, quarantines and other border controls. Because outbreaks caused by some of the currently circulating viruses might readily be missed until the virus is widespread, some countries conduct routine surveillance for this virus, such as periodic sampling of tonsils from dead pigs. Outbreaks in CSF-free nations are eradicated with standard stamping out methods (i.e., slaughter of confirmed cases and contact animals, cleaning and disinfection of infected premises, movement controls and quarantines), sometimes combined with vaccination.

In areas where CSFV occurs in wild or feral suids, domesticated pigs should be protected from contact with these animals with measures such as fencing. Oral vaccines have been used in wild boar to decrease the prevalence of CSFV and potentially eradicate the virus. Reductions in wild boar populations may also be helpful, and fencing has sometimes been employed to control the movements of these animals; however, these methods are not considered as effective as vaccination, and may be difficult to implement well.

Morbidity and Mortality

The severity of classical swine fever is influenced by the viral strain, the age and immune status of the pigs, and other factors such as the animals' general health and viral dose. Highly virulent strains of CSFV, which were prevalent at one time, have morbidity and mortality rates that can approach 100% in a naive herd. However, most outbreaks are now caused by moderately virulent strains, and less virulent strains also circulate. The illnesses caused by these viruses are typically more severe in young animals, especially piglets, with lower mortality rates in adults. Some moderately virulent strains have caused only 20% mortality in experimentally infected pigs.

Regardless of the virulence of the isolate, chronically infected animals and persistently infected piglets usually die. Persistently infected piglets, which are immunotolerant to CSFV, do not respond to CSF vaccines. However, even when unvaccinated, these animals were resistant to inoculation with a highly virulent strain of this virus, with little or no virus replication and no clinical signs. Persistently-infected piglets might also have altered responses to other pathogens. Wild boar piglets persistently infected with CSFV and inoculated with African swine fever virus had high mortality rates that were similar to those in CSFV-free piglets, but the clinical signs were different, with the persistently infected piglets developing a very severe, hemorrhagic illness. Limited evidence suggests that the percentage of piglets that becomes persistently infected is lower when the animals are exposed to CSFV at 3 weeks than one day after birth.

Understanding of the course of a CSF epidemic among wild suids is still limited. CSFV does not seem to have been sustained in wild boar populations in the past, possibly because their numbers were smaller and the circulating viruses usually killed naive animals rapidly. However, wild boar seem to be capable of maintaining the currently circulating strains for prolonged periods, or indefinitely, under some conditions. Although these viruses have been reported to disappear spontaneously from some small wild boar populations, large numbers of animals and high population densities favor their persistence.

Internet Resources

The Merck Veterinary Manual

United States Animal Health Association. Foreign Animal Diseases

World Organization for Animal Health (WOAH)

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals; WOAH Terrestrial Animal Health Code

Acknowledgements

This factsheet was written by Anna Rovid Spickler, DVM, PhD, Veterinary Specialist from the Center for Food Security and Public Health. The U.S. Department of Agriculture Animal and Plant Health Inspection Service (USDA APHIS) provided funding for this factsheet through a series of cooperative agreements related to the development of resources for initial accreditation training.

The following format can be used to cite this factsheet. Spickler, Anna Rovid. 2025. *Classical Swine Fever*. Retrieved from http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php.

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