

CLASSICAL SWINE FEVER

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AETIOLOGY

Classification of the causative agent

Classical swine fever (CSF) virus is a member of the family *Flaviviridae*, genus *Pestivirus*, containing one serotype divided into three major genotypes, including numerous subgenotypes. CSF virus (CSFV) is closely related to ruminant pestiviruses causing bovine viral diarrhoea and border disease.

Resistance to physical and chemical action

Temperature:	Readily inactivated by cooking: heating meat to 65.5°C for 30 minutes or 90–100°C for one minute. Survives months in refrigerated meat and years in frozen meat. Some strains are partially resistant to moderate heat (56°C).
pH:	Stable at pH 5–10. Rapidly inactivated at pH <3.0 or pH >10.
Chemicals/Disinfectants:	Susceptible to ether, chloroform, β -propiolactone (0.4%). Inactivated by sodium hypochlorite, phenolic compounds, quaternary ammonium compounds and aldehydes, chlorine-based disinfectants, cresol (5%), sodium hydroxide (2%), formalin (1%), sodium carbonate (4% anhydrous or 10% crystalline, with 0.1% detergent), ionic and non-ionic detergents, and strong iodophors (1%) in phosphoric acid.
Survival:	Moderately fragile and does not persist in the environment. Sensitive to drying and ultraviolet light. Survives well in pens during cold conditions (up to 4 weeks in winter). Survives 3 days at 50°C and 7–15 days at 37°C. Survives in meat during salt curing and smoking for 17 to >180 days depending on the process used. Virus persists 3–4 days in decomposing organs and 15 days in decomposing blood and bone marrow.

EPIDEMIOLOGY

CSFV is highly contagious. Disease severity is related to viral and host factors, such as the virulence of the viral strain, age and immune system of the pig, as well as the immune status of the herd. Accordingly, the disease can range from peracute and acute to subacute and chronic, and can last for several weeks or even months. The virus can pass from the sows to their offspring, generating congenitally infected pigs that may go undetected for months favouring viral prevalence in the field.

Highly virulent strains of CSFV, which were prevalent in the past, caused outbreaks with morbidity and mortality rates that can approach 100%. However, most outbreaks are now caused by moderately virulent strains, and less virulent strains also circulate. Some strains of low virulence have caused only 20% mortality in experimentally infected pigs.

Case fatality rates also differ with the form of the disease, and are very high in the acute form, but lower in subacute cases. Mortality tends to be lower in adult pigs compared with young animals, especially with less virulent strains.

Hosts

- Pigs and wild boar are the only natural reservoir of CSFV. All feral and wild pigs, including European wild boar, are susceptible
- CSFV has been detected in a white-lipped peccary (*Tayassu pecari*), and experimental infections have been established in common warthogs (*Phacochoerus africanus*), bush pigs (*Potamochoerus larvatus*) and collared peccaries (*Tayassu tajacu*)
- Experimental infections without clinical signs have been reported in cattle, sheep, goats and deer, but there is no evidence that these species become infected in nature. Some CSFV strains can also be adapted to passage in rabbits
- There is no evidence that CSFV infects humans

Transmission

- Mainly by the oral and oronasal routes, via direct or indirect contact
- Direct contact between animals (secretions, excretions, semen, blood)
- Spread by farm visitors, veterinarians, pig traders
- Indirect contact through premises, implements, vehicles, clothes, instruments, needles and insects
- “Neighbourhood effect” during outbreaks in areas of high pig farm density: airborne transmission over short distances (up to 1 km in one study)
- Insufficiently cooked waste food fed to pigs: most common means of entry into free countries
- Transplacental infection: may create inapparent carrier piglets or congenital abnormalities
- Wild boar populations may harbour virus; domestic pigs in the affected area are at a high risk; and biosecurity is crucial

Sources of virus

- Blood, secretions and excretions (oronasal and lachrymal discharges, urine, faeces and semen) and tissues of sick or dead animals, including meat
- Congenitally infected piglets are persistently viraemic and may shed the virus for 6–12 months before dying
- Infection routes: ingestion (most common), contact with the conjunctiva or mucous membranes, skin abrasions, genital transmission, artificial insemination, percutaneous blood transfer

Occurrence

The disease is endemic in much of Asia, Central and South America and some Caribbean countries. CSFV has been eradicated from a number of countries, including European Union countries, United States of America, Canada, New Zealand and Australia.

The status of classical swine fever in some areas of Africa may be uncertain due to limited or no surveillance.

CSF is one of the diseases for which the OIE has a procedure for the official recognition of disease status. For more information, visit the status portal on the OIE website [<http://www.oie.int/en/animal-health-in-the-world/official-disease-status/>]

For more recent, detailed information on the occurrence of this disease worldwide, see the OIE World Animal Health Information Database (WAHID) Interface [<http://www.oie.int/wahis/public.php?page=home>].

DIAGNOSIS

Swine exposed to CSFV prenatally may be persistently infected throughout life and may have an incubation period of several months before showing signs of disease. Swine exposed postnatally have an incubation period of 2–14 days, and often 3–7 days in acute cases. Swine are usually infective between post-infection days 5 and 14, but up to 3 months in cases of chronic infections.

The variability of the clinical signs and post-mortem lesions does not provide firm evidence for unequivocal diagnosis.

Clinical signs

Acute form (more virulent virus strains and/or younger pigs)

- Fever (41°C)
- Anorexia, lethargy
- Multifocal hyperaemia and/or haemorrhagic lesions of the skin
- Conjunctivitis
- Enlarged, swollen lymph nodes
- Cyanosis of the skin especially of extremities (ears, limbs, tail, snout)

- Transient constipation followed by diarrhoea
- Vomiting (occasional)
- Dyspnoea, coughing
- Ataxia, paresis and convulsion
- Pigs huddle together
- Death occurs 5–25 days after onset of illness
- Mortality in young pigs can approach 100%

Chronic form (less virulent virus strains or partially immune herds)

- Dullness, capricious appetite, pyrexia, diarrhoea for up to 1 month
- Ruffled appearance of pigs
- Growth retardation
- Poor reproductive performance may be the only sign in some breeding herds infected with less virulent strains
- Apparent recovery with eventual relapse and death within about 3 months

Congenital form (outcome depends on virulence of virus strain and stage of gestation)

- Fetal death, resorption, mummification, stillbirth
- Abortion
- Congenital tremor, weakness
- Runting and poor growth over a period of weeks or months leading to death
- Born clinically normal but persistently viraemic with no antibody response: important intermittent shedders of virus until dying in 6–12 months (late onset form)

Mild form (usually older animals; outcome depends on virulence of virus strain)

- Transient pyrexia and inappetence
- Recovery and (lifelong) immunity

Lesions

Acute form: Lesions are usually complicated by secondary infections

- Leucopenia and thrombocytopenia
- Enlarged hemorrhagic lymph nodes are common
- Widespread petechiae and ecchymoses, especially in the skin, lymph nodes, epiglottis, bladder, kidney and rectum
- Severe tonsillitis with necrotic foci sometimes occurs
- Multifocal infarction of the margin of the spleen is characteristic: nearly pathognomonic but occurs infrequently with currently circulating strains
- Lungs may be congested and hemorrhagic
- Encephalomyelitis with perivascular cuffing is common

Chronic form: Lesions are usually complicated by secondary infections

- 'Button' ulcers in the cecum and large intestine mucosa
- Generalised depletion of lymphoid tissue
- Transverse striations of unmodelled growth cartilage at costochondral junctions in growing pigs
- Hemorrhagic and inflammatory lesions are often absent

Congenital form

- Central dysmyelination, cerebellar hypoplasia, microencephaly, pulmonary hypoplasia, hydrops and other malformations

Differential diagnosis Varies with form of the disease

- African swine fever
- Septicemias: erysipelas, *Mycoplasma suis*, salmonellosis, streptococcosis, pasteurellosis, actinobacillosis, and *Hemophilus parasuis*
- Haemorrhage: porcine dermatitis and nephropathy syndrome, haemolytic disease of the newborn, coumarin poisoning, thrombocytopenic purpura
- Runtling: post weaning multisystemic wasting syndrome, enterotoxigenic, swine dysentery, campylobacteriosis
- Abortions: Aujeszky's disease (pseudorabies virus), encephalomyocarditis virus infection, porcine reproductive and respiratory syndrome, parvovirus
- Nervous signs: viral encephalomyelitis, salt poisoning
- Congenital infection with ruminant pestiviruses: bovine viral diarrhoea and border disease

Laboratory diagnosis

Samples

Method of choice for detecting herds early in infection is to collect whole blood and tissues from multiple febrile or recently dead animals. Refrigerate and ship to laboratory as quickly as possible.

- Live cases (samples collected during febrile periods)
 - Blood in EDTA or Heparin
 - Sera
 - Tonsil swabs
- Necropsy of recently dead animals
 - Tonsils (the most suitable organ for virus isolation)
 - Pharyngeal and mesenteric lymph nodes
 - Spleen
 - Kidneys
 - Distal ileum

Procedures

Identification of the agent

- Virus isolation
 - Isolation in PK-15 cell line culture
 - Demonstration by an immunostaining method (FAT or immunoperoxidase)
- Reverse transcription polymerase chain reaction (RT-PCR) techniques
 - Detects infected animals early during the incubation period and for a longer period of time in cases where the pigs recover.
 - Detects viral nucleic acid only and positive results may be obtained in cases where virus isolation or other techniques yield negative results.
 - It is a suitable approach for screening and confirmation of suspected cases of disease and is now accepted by a number of countries and including the European Union.
- Molecular epidemiology and genetic typing
 - Molecular epidemiology of CSF is based on the comparison of genetic differences between virus isolates,
 - Two regions have been extensively studied and provide large sets of sequence data with which new isolates can be compared. One of these regions lies within the 5'-nontranslated region (5'NTR) of the genome (150 nucleotides) and the other within the E2 major glycoprotein gene (190 nucleotides).
 - the method used involves extracting virus RNA from clinical samples or cell cultures infected with low passage CSFV, performing RT-PCR to amplify one or both targets

- within the 5'NTR or the E2 gene, and then determining the nucleotide sequence of the products and comparing with stored sequence information
 - CSFV isolates from primary outbreaks should be sent to an OIE Reference Laboratory for investigation of molecular epidemiology.
- Antigen-capture enzyme-linked immunosorbent assays (ELISAs)
 - The test is not suitable for the diagnosis of CSF in a single animal, but should only be used at the herd level
 - In any primary case, positive results must be confirmed using another test (i.e. virus isolation, RT-PCR or FAT).
- Fluorescent antibody test (FAT)
 - It is a rapid test that can be used to detect CSFV antigen in cryostat sections of samples
 - Tissues should be collected from several (febrile and/or diseased) animals and transported without preservatives under cool conditions, but not frozen.
 - In subacute and chronic cases, the ileum is frequently positive and occasionally may be the only tissue to display fluorescence.
 - A negative FAT result does not completely rule out CSF infection.

Serological tests

Due to the immunosuppressive effect of CSFV, antibodies cannot be detected with certainty until at least 21 days post-infection. Serological investigations aimed at detecting residual foci of infection, especially in breeding herds, may be useful in a terminal phase of CSF eradication. Antibody titres provide valuable epidemiological information and may be of help in determining the entry route of the virus.

Sera from pigs infected with BVDV or BDV may show cross-neutralising antibody titres that react in the FAVN or NPLA as if the pigs were infected with CSFV. The extent of cross-reactivity depends on the strain of ruminant pestivirus involved and the interval between infection and time of sampling. In case of continued doubt, comparative tests using a strain of CSFV, a strain of BVDV and a strain of BDV, that are representative for the country or region, have proven useful.

As the incidence of infection with ruminant pestiviruses may be high, particularly in breeding stock, only tests that will discriminate between CSF and BVD/BD antibodies are useful. Virus neutralisation (VN) and the ELISA using MAbs satisfy the requirements for sensitivity, but positive results should be confirmed by comparative VN testing.

The following may be used for serological diagnosis or surveillance, and are also tests identified by the OIE as fit for purpose for screening for international trade:

- Neutralising peroxidase linked assay (NPLA)
- Fluorescent antibody virus neutralisation (FAVN)
- Enzyme linked immunosorbent assay (ELISA)

For more detailed information regarding laboratory diagnostic methodologies, please refer to Chapter 3.8.3 Classical swine fever in the latest edition of the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading "Diagnostic Techniques"

PREVENTION AND CONTROL

No treatment is possible. Affected pigs must be slaughtered and the carcasses buried or incinerated.

Sanitary prophylaxis

- Effective communication between veterinary authorities, veterinary practitioners and pig farmers
- Effective disease reporting system
- Strict import policy for live pigs, pig semen, and fresh and cured pig meat
- Quarantine of pigs before admission into herd
- Efficient sterilisation (or prohibition) of waste food fed to pigs
- Efficient control of rendering plants
- Structured serological surveillance targeted to breeding sows and boars

- Effective pig identification and recording system
- Effective hygiene measures protecting domestic pigs from contact with wild boar
- Response to outbreak:
 - Slaughter of all pigs on affected farms
 - Safe disposal of carcasses, bedding, etc.
 - Thorough disinfection
 - Designation of infected zone, with control of pig movements
 - Detailed epidemiological investigation, with tracing of possible sources (up-stream) and possible spread (down-stream) of infection
 - Surveillance of infected zone and surrounding area

Medical prophylaxis

Vaccination

- Modified live vaccines (MLVs) based on several attenuated virus strains are most widely used, and many of them have proven to be both safe and efficacious.
- New generations of marker vaccines are also being developed, including a new chimeric pestivirus vaccine that has been licensed by the European Medicines Agency (EMA).
- Different strategies are available to differentiate infected from vaccinated animals (DIVA) by serological methods (e.g. ELISA) or genome detection methods (e.g. RT-PCR).
- An opinion published by the European Food Safety Authority (EFSA, 2008) demonstrated that the combination of a vaccine that uses the C-strain with RT-PCR to detect viral genome in slaughtered animals can be successfully used in a vaccination-to-live strategy.
- For the newly licensed chimeric pestivirus vaccine, the use of a ruminant pestivirus backbone provides serological differentiation by use of CSFV Erns-ELISAs. However, evaluation of two such assays for DIVA capability revealed that test specificity may be compromised by infection with ruminant pestiviruses, resulting in induction of cross-reactive antibodies.

Disease free countries

- The majority has adopted a control strategy without prophylactic vaccination but established legal provisions for emergency vaccination scenarios.
- During epidemic incidents in otherwise free areas, emergency vaccination can be an additional tool to control and eradicate the disease.

Endemic countries

- In endemic situations, vaccination is mainly used to lower the impact of the disease or as a first step in an eradication programme.
- Moreover, oral vaccination of affected wild boar populations may be considered

For more detailed information regarding vaccines, please refer to Chapter 3.8.3 Classical swine fever in the latest edition of the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading “Requirements for Vaccines and Diagnostic Biologicals”.

For more detailed information regarding safe international trade in terrestrial animals and their products, please refer to the latest edition of the OIE *Terrestrial Animal Health Code*.

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The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (scientific.dept@oie.int). Last updated January 2020