



AFRICAN SWINE FEVER

ANIMAL GROUP AFFECTED	TRANSMISSION	CLINICAL SIGNS	FATAL DISEASE ?	TREATMENT	PREVENTION & CONTROL
Domestic pigs, wild boars, bush pigs, warthogs, American wild pigs. African wild swine (warthogs and bush pigs) are usually inapparently infected	Direct contact oronasal, lachrymal secretions, urine, faeces, insemination, blood transfer, feeding of raw or insufficiently cooked meat, transplacental infection	Severe depression, anorexia, diarrhoea, high fever, superficial and internal haemorrhages, prolonged and intermittent in chronic form, intrauterine infection: abortion	In acute form morbidity and mortality can be high	Strict eradication	<i>In houses</i> <i>in zoos</i> Serological monitoring system, strict importation restrictions, no garbage/meat feeding

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Susceptible animal groups Domestic pigs and wild boars, bush pigs, warthogs, American wild pigs. African wild swine (warthogs and bush pigs) are usually inapparently infected.	
Causative organism African swine fever virus (ASFV) is a large (about 200 nm) lipoprotein-enveloped, icosahedral, double-stranded DNA virus. Originally classified as an iridovirus, it was found to have many characteristics of poxvirus; thus a new virus family was established for ASFV.	
Zoonotic potential Humans are not susceptible to ASFV infection.	
Distribution African swine fever (ASF) is enzootic in most countries of Sub-Saharan Africa. In Europe it has been reported in the Iberian Peninsula and in Sardinia. It was present in four South American and Caribbean countries, but has been eradicated. It has not been reported in Australia, Canada, New Zealand, and the USA.	
Transmission Primarily faecal-oral route, though infection can occur through the conjunctiva, mucous membranes, skin abrasions, insemination, and percutaneous blood transfer (e.g. iatrogenic). Blood, tissues, semen, secretions and excretions from an infected animal contain ASFV. Biological vectors transmit the virus: soft ticks of the genus <i>Ornithodoros</i> . ASFV replicates in the tick and there is transstadial, transovarial, and sexual transmission in <i>Ornithodoros</i> ticks. A carrier state exists, especially in African wild swine, and in domestic pigs in enzootic areas. ASFV in wild pigs in Africa is now believed to cycle between soft ticks living in warthog burrows and newborn warthogs. Contact with infected animals is the principal source of infection with ASFV. Feeding of raw or insufficiently cooked meat or contaminated food (access of infected wild boars to food source) is a potent source of ASFV. Transplacental infection with viral strains of low virulence may result in persistently infected piglets. In a protein-rich environment, ASFV 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. In serum-free medium, ASFV is inactivated at pH 3.9 or lower and at pH 11.5 or higher. In the presence of 25 percent serum, ASFV will remain viable for 7 days at pH 13.4. The virus will survive for 15 weeks in putrefied blood, 3 hours at 50 C, 70 days in blood on wooden boards, 11 days in faeces held at room temperature, 18 months in pig blood held at 4 C, 150 days in boned meat held at 4°C and 140 days in salted dried hams.	

**Incubation period**

The incubation period is 5-15 days.

Clinical symptoms

ASFV isolates with lower virulence have emerged during the last decade, especially in the Iberian peninsula. In peracute forms caused by highly virulent strains in 7-10 days after exposure, mortality can be up to 10% and some pigs are suddenly found dead, or close to death.

Moderately virulent strains cause the acute form of disease in which a high percentage of the pigs survive. There is fever (40.5-41.7°C), loss of appetite and inactivity. Areas of red or blue skin discoloration may appear on the ventral chest or abdomen, tips of ears or tail, or distal limbs. Diarrhoea, vomiting, coughing, breathing difficulty and abortion may also occur. Marked leukopenia is common at the onset of high fever and persists throughout the course of the disease. Almost 100% of pigs with these symptoms will die within 7 days. Pigs that recover can be lifelong carriers of virus.

Subacute forms are mainly caused by strains of moderate virulence. Affected pigs are only mildly ill, but sows may abort. There can be intermittent fever for up to one month, followed in most cases by recovery. Mortality ranges from 30-70%. Recovered pigs can still be excreting the virus up to six weeks after infection. Very young pigs may have a high mortality and have lesions similar to infection by highly virulent virus.

In chronic forms caused by low virulent strains often only seroconversion occurs. Although having occasional episodes of fever, these pigs show little illness apart from reduced growth, stunting or emaciation. There may be necrotic patches of skin or chronic skin ulcers. They are vulnerable to secondary infections, pneumonia and lameness (arthritis). Infection lasts for two to five months but mortality is less than 30%. In contrast to classical swine fever (CSF), ASFV-infected pigs do not develop a conjunctivitis or encephalitis, and, despite the high fever, stay in good condition, whereas CSF-infected pigs quickly lose much weight.

Post mortem findings

Pigs that die peracutely may have poorly developed lesions. The early cause of death is frequently haemorrhages into the stomach as a sequel to thrombocytopenia that results in a prolonged bleeding time and haemorrhage from a pre-existing gastric ulcer.

Pigs with acute and subacute disease often have splenomegaly, and lymphadenopathy of the gastrohepatic and renal lymph nodes. Affected spleens are greatly enlarged, dark red to black and friable. The affected lymph nodes are diffusely haemorrhagic or have a marbled appearance. Other lesions are more variable and include dark red to purple areas of skin on ears, feet, and tail, petechial to echymotic haemorrhages on serosal surfaces and in the renal cortex, perirenal oedema, oedema of the gall bladder, hepatomegaly and pulmonary oedema. Moderately virulent ASFV may cause splenomegaly, but the spleen although enlarged, has a more normal colour and is not friable.

In chronic ASF the most common lesions are cutaneous necrosis, lobular pneumonia and pleuritis, generalized lymphadenopathy, arthritis and pericarditis. Aborted fetuses may be anasarca, and there may be petechial haemorrhages in the placenta, skin, and myocardium, and a mottled liver.

The warthog and bush pig develop viraemia, but have very mild or subclinical disease.

Microscopic lesions:

- Acute form: endothelial damage causing microthrombosis and haemorrhages. Diffuse necrosis of the spleen and lymph nodes is common.
- Chronic forms: microscopic lesions in the respiratory tract incl. fibrinous pleuritis and caseous pneumonia, cutaneous necrosis and pericarditis.

Diagnosis

With highly virulent strains of ASF essentially 100% of the pigs will die. Lesser virulent strains of ASFV are more difficult to diagnose but an infection should be excluded when there are febrile pigs and necropsy findings include splenomegaly, and lymphadenopathy of the gastro-hepatic and renal lymph nodes.

The initial diagnosis can be based on demonstration of infectious virus, viral antigen, viral DNA or specific antibodies. ASFV is present in the blood starting about 2 days post inoculation. Less virulent isolates of ASFV can usually be isolated from the blood for 25 or more days post inoculation.

Pieces of tissues (see next section), the brain, and any other gross lesion should be submitted in 10% buffered formalin.

Neither complement fixation, immunoperoxidase staining, ELISA, electron microscopy nor DNA-hybridization are useful tests for the routine diagnosis of ASFV. The direct immunofluorescence (DIF) and haemagglutination (HA) are recommended in suspect cases. However, for chronic forms the DIF has only a sensitivity of 40%. HA is more sensitive, but a few field strains have been isolated that do not induce HA. PCR has been employed as a more rapid, highly specific and sensitive tool for the diagnosis of ASFV. A diagnosis can be confirmed by demonstrating ASF antigen in tissue using immunohistochemistry or in-situ hybridization. Indirect immunofluorescence and ELISA are used to detect antibodies against ASFV. Early appearance and persistence of antibodies make these test a useful tool to detect subacute and chronic disease.

The main differential diagnose for ASF is CSF. Septicemic conditions in which pigs have high fever should be carefully investigated. Septicemic bacterial diseases, including *Salmonella choleraesuis*, *Actinobacillus suis*,



Hemophilus parasuis, *Erysipelothrix rhusiopathiae* and *Eperythrozoon* may all cause similar clinical symptoms and lesions. ASF has frequently been misdiagnosed as CSF. In contrast to CSF, ASFV-infected pigs do not develop a conjunctivitis or encephalitis, and despite the high fever, the ASFV-infected pigs stay in good condition.

Material required for laboratory analysis

Heparinized blood, clotted blood or serum, submandibular lymph node, inguinal lymph node, tonsil, spleen, gastro-hepatic lymph node, lung, liver, kidney. Bone marrow should be submitted if there are considerable postmortem changes. The specimens should be shipped refrigerated or frozen.

Samples should be collected from pigs that have fever or show other signs of the disease. Serum samples for antibody detection should be collected from animals that have recovered from suspected infection or from sows that have been in contact with infected or suspected cases. Aborted foetuses are usually free of virus; therefore, it is necessary to submit a blood sample from the sow.

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Treatment

There is no treatment for ASFV infected pigs.

Prevention and control in zoos

Control 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. The threat of reintroduction is significant and it is essential to initiate an effective serological monitoring system. Strict importation restrictions must be applied to pigs and pork products originating from countries where ASFV is present to prevent outbreaks. Garbage feeding must be stopped or carefully regulated to insure proper cooking. The virus is heat inactivated by 56°C/70 min; 60°C/20 min.

Suggested disinfectant for housing facilities

ASFV can be inactivated by cresol, sodium hydroxide (2%), formalin (1%), sodium carbonate (4% anhydrous or 10% crystalline, with 0.1% detergent), ionic and non-ionic detergents, strong iodophors (1%) in phosphoric acid, lipid solvents such as chloroform. Examples of effective disinfectants: potassium peroxydisulfate (Antec Virkon S at a dilution rate of 1:100); hypochlorites (bleach, Chlorox (The Chlorox Company) at a dilution rate of 1:32 (only in the absence of organic material, disinfectant properties of sodium hypochlorite are inactivated by organic material and diminished by alkaline materials (lime) and moisture, contact with skin is irritating), phenols and related compounds, e.g. cresols, 1 Stroke Environ® (Calgon Vestal), Tek-Trol (Bio-Tek Industries, Inc.) at 1-2% concentrations, not inactivated by organic debris, disinfectant properties are enhanced by warm temperatures, and diminished by cold temperatures and moisture, contact with skin is



corrosive and the use of goggles and rubber gloves is recommended.
Notification Yes.
Guarantees required under EU Legislation
Guarantees required by EAZA Zoos
Measures required under the Animal Disease Surveillance Plan
Measures required for introducing animals from non-approved sources
Measures to be taken in case of disease outbreak or positive laboratory findings
Conditions for restoring disease-free status after an outbreak
Contacts for further information
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