African Swine Fever

Peste Porcine Africaine,
Fiebre Porcina Africana,
Pestis Africana Suum,
Maladie de Montgomery,
Warthog Disease,
Afrikaanse Varkpes,
Afrikanische Schweinepest

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Importance

African swine fever is a serious, highly contagious, viral disease of pigs. African swine fever virus (ASFV) can spread very rapidly in pig populations by direct or indirect contact. It can persist for long periods in uncooked pig products, facilitating its introduction into new areas. This virus can also become endemic in feral or wild suids, and transmission cycles between these animals and *Ornithodoros* ticks can complicate or even prevent eradication. ASFV isolates vary in virulence from highly pathogenic strains that cause near 100% mortality to low–virulence isolates that can be difficult to diagnose. There is no vaccine or treatment.

African swine fever is a serious problem in many African countries. Changes in production practices and increasing globalization have also increased the risk of its introduction into other regions. Past outbreaks occurred in Europe, South America and the Caribbean, and the cost of eradication was significant. The swine herds of Malta and the Dominican Republic were completely depopulated during outbreaks in these countries. In Spain and Portugal, ASFV became endemic in the 1960s and complete eradication took more than 30 years. It still remains present on the island of Sardinia. In 2007, Africa swine fever was introduced into the Caucasus region of Eurasia, where it has spread widely among wild boar and domesticated pigs. This virus has caused outbreaks in pigs as far west as the easternmost countries of the E.U., and it has also been detected in wild boar in Iran. In 2018, African swine fever was detected in multiple locations in China.

Etiology

African swine fever results from infection by the African swine fever virus, which belongs to the genus *Asfivirus* in the family Asfarviridae. More than 20 genotypes of ASFV have been identified, most from wildlife cycles in Africa. The virus introduced into the Caucasus belongs to genotype II, while viruses endemic in Sardinia belong to genotype I. ASFV isolates differ greatly in virulence, from highly pathogenic viruses that kill most pigs to strains that result only in seroconversion.

Species Affected

African swine fever affects members of the pig family (Suidae). Species that can be infected include domesticated swine, Eurasian wild boars (*Sus scrofa scrofa*), warthogs (*Phacochoerus* spp.), bush pigs (*Potamochoerus larvatus* and *Potamochoerus porcus*) and giant forest hogs (*Hylochoerus* spp.). Warthogs and bush pigs, which are generally asymptomatic, are thought to be wildlife reservoirs for the virus in Africa. Some older reviews and textbooks suggest that peccaries (*Tayassu* spp.) may also become infected without clinical signs, although one attempt to infect collared peccaries (*Tayassu tajacu*) in 1969 was unsuccessful. Recent reviews state that that peccaries are not susceptible.

Zoonotic potential

There is no evidence that ASFV infects humans.

Geographic Distribution

African swine fever is endemic in most of sub-Saharan Africa including the island of Madagascar. Outbreaks have been reported periodically outside Africa. The virus was eventually eradicated in most cases, although it remains endemic on the island of Sardinia (Italy) in the Mediterranean. In 2007, ASFV was introduced into the Caucasus region of Eurasia, via the Republic of Georgia, and it has spread to domesticated pigs and/or wild boars in a number of countries in this region. As of 2015, infections had been reported as far west as Lithuania, Latvia and Poland. Viruses that apparently originated from this outbreak have also been found in wild boar in the Middle East (Iran). China reported African swine fever in domestic pigs at multiple location within the country in 2018.

Transmission

African swine fever can be transmitted either with or without tick vectors as intermediaries. After direct (non-tickborne) contact with the virus, ASFV is mainly

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thought to enter the body via the upper respiratory tract. This virus has been found in all secretions and excretions of sick domesticated pigs, with particularly high concentrations in oronasal fluid. There may, however, be species differences among the Suidae. For instance, concentrations of ASFV appear to be much lower in adult warthogs, compared to pigs, and adult warthogs might not transmit the virus by direct contact. In pigs, aerosolized viruses may contribute to transmission within a building or farm, although current evidence suggests that this only occurs over relatively short distances. Because ASFV can persist in blood and tissues after death, it is readily spread by feeding uncooked swill that contains tissues from infected animals. Some reports suggest that the cannibalism of dead pigs may be important in transmission. In addition, massive environmental contamination may result if blood is shed during necropsies or pig fights, or if a pig develops bloody diarrhea. How long pigs can remain infected is still uncertain. Several studies have reported finding ASFV in the tissues of domesticated pigs for as long as 3 to 6 months, and virus shedding and transmission for at least 70 days after experimental inoculation. However, there are also studies where pigs could not transmit the virus for longer than a month. Currently, there is no evidence that the virus persists long-term in a latent state.

ASFV can spread on fomites, including vehicles, feed and equipment. In feces kept at room temperature, this virus was estimated to survive for several days in some reports, and for at least 11 days in one study where the sample was stored in the dark. One study reported that ASFV remained infectious longer in urine than feces, with estimated survival times of 3 days at 37ºC and 15 days at 4ºC. ASFV can also persist for a year and a half in blood stored at 4ºC, 150 days in boned meat stored at 39ºF, 140 days in salted dried hams, and several years in frozen carcasses.

Vector-mediated transmission is through the bites of *Ornithodoros* spp. soft ticks. In some regions of Africa, ASFV is thought to cycle between juvenile common warthogs (*Phacochoerus africanus*) and soft ticks of the *Ornithodoros moubata* complex, that live in their burrows. Transstadial, transovarial and sexual transmission have been demonstrated in these ticks. A similar cycle is thought to exist between domesticated pigs and the *Ornithodoros moubata* complex ticks that colonize their pig pens in Africa. *Ornithodoros erraticus* acted as a biological vector during an outbreak on the Iberian Peninsula in Europe, and additional species of *Ornithodoros* have been infected in the laboratory. *Ornithodoros* spp. ticks are long-lived, and colonies have been demonstrated to maintain ASFV for several years (e.g., 5 years in *O. erraticus*). However, the ticks can eventually clear the virus if they are not reinfected. There is no evidence that hard ticks act as biological vectors for ASFV.

Other bloodsucking insects such as mosquitoes and biting flies might be able to transmit ASFV mechanically. Stable flies (*Stomoxys calcitrans*) can carry high levels of the virus for 2 days. Under experimental conditions, these flies could transmit ASFV 24 hours after feeding on infected pigs.

Disinfection

Many common disinfectants are ineffective against ASFV; care should be taken to use a disinfectant specifically approved for this virus. Sodium hypochlorite, citric acid (1%) and some iodine and quaternary ammonium compounds are reported to destroy ASFV on some nonporous surfaces. In one recent experiment, either 2% citric acid or higher concentrations of sodium hypochlorite (e.g., 2000 ppm) could disinfect the virus on wood; however, citric acid was more effective.

Unprocessed meat must be heated to at least 70ºC for 30 minutes to inactivate ASFV; 30 minutes at 60ºC is sufficient for serum and body fluids. This virus can also be inactivated by pH < 3.9 or > 11.5 in serum-free medium.

Incubation Period

The incubation period is 5 to 21 days after direct contact with infected pigs, but it can be less than 5 days after exposure to ticks. Acute disease typically appears in 3 to 7 days.

Clinical Signs

African swine fever can be a peracute, acute, subacute or chronic disease. Severe cases that affect large numbers of animals may be readily recognized; however, some herds develop milder clinical signs that are easily confused with other diseases. Some animals can seroconvert without developing clinical signs.

Sudden deaths with few lesions (peracute cases) may be the first sign of an infection in a herd. Acute cases are characterized by a high fever, anorexia, lethargy, weakness and recumbency. Erythema can be seen, and is most apparent in white pigs. Some pigs develop cyanotic skin blotching, especially on the ears, tail, lower legs or hams. Pigs may also have diarrhea, constipation and/or signs of abdominal pain; the diarrhea is initially mucoid and may later become bloody. There may also be visible signs of hemorrhagic tendencies, including epistaxis and hemorrhages in the skin. Respiratory signs (including dyspnea), nasal and conjunctival discharges, and neurological signs have also been reported. Pregnant animals frequently abort; in some cases, abortions may be the first signs of an outbreak. Leukopenia and thrombocytopenia of varying severity may be detected in laboratory tests. Death often occurs within 7 to 10 days.

Subacute African swine fever, caused by moderately virulent isolates, is similar to acute ASF but with less severe clinical signs. Abortions may be the first sign. Fever, thrombocytopenia and leukopenia may be transient in this form; however, hemorrhages can occur during the period of thrombocytopenia. Affected pigs usually die or recover within 3 to 4 weeks.

Chronic disease was described in Europe when ASFV was endemic on the Iberian Peninsula. Some authors speculate that the strains that cause this form might have originated from live attenuated vaccine strains investigated at that time. Pigs with the chronic form can have intermittent low fever, appetite loss and depression. The signs may be limited to emaciation and stunting in some animals. Other pigs develop respiratory problems and swollen joints. Coughing is common, and diarrhea and occasional vomiting have been reported. Ulcers and reddened or raised necrotic skin foci may appear over body protrusions and other areas subject to trauma. Chronic African swine fever can be fatal.

Signs in wild boar inoculated with a highly virulent isolate were similar to those in domesticated pigs; however, some runted animals infected with very low viral doses had few or no clinical signs, including fever, before death. Warthogs and bush pigs usually become infected asymptomatically or have mild disease.

Post Mortem Lesions

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The gross lesions of African swine fever are highly variable, and are affected by the virulence of the isolate and the course of the disease.

Numerous organs may be affected, to varying extent, in animals with acute or subacute African swine fever. The carcass is often in good condition in animals that die acutely. There may be bluish-purple discoloration and/or hemorrhages in the skin, and signs of bloody diarrhea or other internal hemorrhages. The major internal lesions are hemorrhagic, and occur most consistently in the spleen, lymph nodes, kidneys and heart. In animals infected with highly virulent isolates, the spleen can be very large, friable, and dark red to black. In other cases, the spleen may be enlarged but not friable, and the color may be closer to normal. The lymph nodes are often swollen and hemorrhagic, and may look like blood clots; the nodes most often affected are the gastrohepatic and renal lymph nodes. Petechiae are common on the cortical and cut surfaces of the kidneys, and sometimes in the renal pelvis. Perirenal edema may be present. Hemorrhages, petechiae and/or ecchymoses are sometimes detected in other organs including the urinary bladder, lungs, stomach and intestines. Pulmonary edema and congestion can be prominent in some pigs. There may also be congestion of the liver and edema in the wall of the gall bladder and bile duct, and the pleural, pericardial and/or peritoneal cavities may contain straw-colored or blood-stained fluid. The brain and meninges can be congested, edematous or hemorrhagic. Animals that die peracutely may have few or poorly developed lesions.

In animals with chronic African swine fever, the carcass may be emaciated. Other possible post–mortem lesions include focal areas of skin necrosis, skin ulcers, consolidated lobules in the lung, caseous pneumonia, nonseptic fibrinous pericarditis, pleural adhesions, generalized lymphadenopathy and swollen joints. Some lesions may result from secondary infections.

Aborted fetuses can be anasarcous and have a mottled liver. They may have petechiae or ecchymoses in the skin and myocardium. Petechiae can also be found in the placenta.

Diagnostic Tests

African swine fever can be diagnosed by virus isolation. This virus can be detected in blood from live animals or tissues (especially spleen, kidney, tonsils and lymph nodes) collected at necropsy. ASFV is not found in aborted fetuses; in cases of abortion, a blood sample should be collected from the dam. Cell types used for virus isolation include pig leukocyte or bone marrow cultures, porcine alveolar macrophages and blood monocyte cultures. One recent study used MARC-145 (African green monkey kidney) cells. ASFV-infected cells may be detected by their ability to induce hemadsorption of pig erythrocytes to their surfaces. A few non-hemadsorbing isolates can be missed with this test; most of these viruses are avirulent, but some do produce acute, symptomatic disease. PCR or immunofluorescence can also be used to detect the virus, and PCR can be used to confirm its identity.

PCR is often used to detect ASFV nucleic acids in clinical samples. It can be employed with putrefied samples, which are unsuitable for virus isolation and antigen detection, as well as with fresh tissues or blood. One study reported that, after death, the levels of viral DNA were highest in the spleen, and persisted longest in this tissue. There is also a published report describing the use of PCR with tonsil scrapings from live, experimentally infected animals, as well as blood or nasal swabs. Isothermal amplification methods are also in development.

ASFV antigens may be found in tissue smears or cryostat sections, as well as in buffy coat samples, using ELISAs or immunofluorescence. These tests are best employed as herd tests, and in conjunction with other assays. Antigens are easiest to detect in acute cases; these tests are less sensitive in subacutely or chronically infected animals. A hemadsorption “autorosette” test can also be used to detect ASFV directly in peripheral blood leukocytes; however, this test has mostly been replaced by PCR, which is easier to evaluate.

Serology may be useful, particularly in endemic regions. Pigs with acute disease often die before developing antibodies; however, antibodies to ASFV persist for long periods in animals that survive. Many serological tests have been developed for the diagnosis of African swine fever, but only a few have been standardized for routine use in diagnostic laboratories. Currently used tests include ELISAs, immunoblotting and indirect fluorescent antibody (IFA) tests. The ELISA is prescribed for international trade, and is generally confirmed by immunoblotting (although IFA can also be used).

Treatment

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

Control

Disease reporting

A quick response is vital for containing outbreaks in ASFV-free regions. Veterinarians who encounter or suspect African 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 the past, heat treatment was used to inactivate viruses in pig swill (scraps fed to pigs) and prevent the entry of ASFV into areas free of this disease. Due to the risk that this and other viruses may not be completely inactivated (for example, if parts of the swill do not reach the target temperature), feeding swill to pigs has now been completely forbidden in some countries.

Some areas that experienced ASFV outbreaks successfully eradicated the virus by the slaughter of infected and in–contact animals, safe carcass disposal, sanitation, disinfection, movement controls and quarantines, and the prevention of contact with wild suids and infected ticks. However, the length and complexity of eradication campaigns differed with the local conditions. On the Iberian Peninsula, for example, ASFV had become established in wild boars and *Ornithodoros erraticus* ticks, and complete eradication took decades. Pigpens with infected ticks were destroyed or isolated as part of this campaign. Current regulations in the EU allow pig farms to be restocked as soon as 40 days after cleaning and disinfection, if an African swine fever outbreak occurs in the absence of vectors; however, the minimum quarantine is 6 years if vectors are thought to be involved in transmission. *Ornithodoros* ticks apparently did not become chronically infected during outbreaks in South America, and this (together with the absence of virus in wildlife or feral pigs) simplified eradication.

Eradication of ASFV from some wild reservoirs in Africa, such as warthogs, appears unlikely. However, compartments where African swine fever is controlled and barriers prevent contact with wild reservoirs have been established in some regions. No vaccine is currently available.

Morbidity and Mortality

In domesticated pigs, the morbidity rate can approach 100% in naïve herds; however, viruses may take days or several weeks to spread through the herd. The mortality rate depends on the virulence of the isolate, and can range from <5% to 100%. Highly virulent isolates can cause nearly 100% mortality in pigs of all ages. Less virulent isolates are more likely to be fatal in pigs with a concurrent disease, pregnant animals and young animals. Mortality also tends to be high when ASFV is introduced into new regions, with an increased incidence of subacute and subclinical cases once it becomes endemic. In subacute disease, the mortality rate ranges from 30% to 70%, and may differ between age groups. In some situations, rates can be as high as 70-80% in young pigs but less than 20% in older animals.

Internet Resources

CIRAD Pigtrop (Pig Production in Developing Countries)
http://pigtrop.cirad.fr/home

Food and Agriculture Organization of the United Nations. Recognizing African Swine Fever. A Field Manual.
http://www.fao.org/docrep/004/X8060E/X8060E00.HTM

The Merck Veterinary Manual
http://www.merckvetmanual.com/mvm/index.html

United States Animal Health Association. Foreign Animal Diseases
http://www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf

World Organization for Animal Health (OIE)
http://www.oie.int

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/

OIE Terrestrial Animal Health Code
http://www.oie.int/international-standard-setting/terrestrial-code/access-online/

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