

African Swine Fever

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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, African 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.

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).

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 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

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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 37°C and 15 days at 4°C. ASFV can also persist for a year and a half in blood stored at 4°C, 150 days in boned meat stored at 39° F, 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 the soft ticks belonging to 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 70°C for 30 minutes to inactivate ASFV; 30 minutes at 60°C 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 symptoms 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, vomiting, 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; 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

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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 runt animals infected with very low viral doses had few or no clinical signs, including fever, before death. Warthogs and bush pigs usually become infected asymptotically or have mild disease.

Post Morte Lesions

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