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 an important viral disease of pigs that has become a serious threat to worldwide pork production since 2007. African swine fever virus (ASFV) usually circulates in sub-Saharan Africa, where it is thought to have originated in wild warthogs but has become a common virus in domesticated pigs. ASF viruses range from highly pathogenic strains that may kill nearly the entire herd to less virulent isolates that cause a milder, nonspecific illness difficult to recognize as African swine fever. There is no vaccine and no effective treatment, and severely affected pigs usually die. The spread of ASFV is facilitated by a number of factors, including its persistence for long periods in uncooked pork products, which may be fed to pigs in food scraps (pig swill), and its ability to become established in wild or feral suids. In some areas, control is complicated by the establishment of the virus in *Ornithodoros* ticks, which occurs in addition to direct transmission between animals. One tick vector hindered eradication efforts during a previous outbreak in Spain and Portugal, where complete elimination of the virus took more than 30 years.

In 2007, ASFV was accidentally introduced into the Caucasus region of Eurasia, most likely in pig swill from Africa. This highly virulent virus caused outbreaks on pig farms, but it also became established in wild boar, and has been spreading slowly and steadily in these animals, with occasional larger jumps attributed to transmission by people or the transport of domesticated pigs. As of June 2019, infected wild boar have been found as far west as the Baltic region, parts of Central Europe (e.g., Poland, Hungary) and Belgium. While outbreaks in domesticated herds have been eradicated, it is still uncertain whether the virus can be eliminated from wild boar. In 2018, the same virus was detected in China, where it appears to have spread widely before the outbreak was recognized. African swine fever has since been reported in domesticated pigs in several other southern Asian countries, as well as in wild boar, and the virus appears to be spreading quickly in some parts of Asia. There are fears that it could be transported from Eurasia to other locations, including the Americas, as at least one virus was in the past. One report from 2010 described finding ASFV in wild boar in Iran, though there are no other reports indicating its presence in the Middle East.

Etiology

African swine fever results from infection by African swine fever virus (ASFV), an enveloped virus in the genus *Asfivirus* and family Asfarviridae. More than 20 genotypes of ASFV have been identified, many from wildlife cycles in Africa. Some of these viruses also occur in domesticated pigs. The virus introduced in 2007 into the Caucasus belongs to genotype II, while a virus that has been endemic in Sardinia (Italy) since the 1960s is of genotype I.

ASFV isolates differ greatly in virulence, from highly pathogenic viruses that kill most pigs to strains that result only in seroconversion. The genotype II virus currently circulating in Eurasia is highly virulent and remains the predominant strain, though less virulent viruses have been reported sporadically during this outbreak.

Species Affected

African swine fever affects members of the pig family (Suidae). Species known to be susceptible to infection include domesticated swine and wild boar (both subspecies of *Sus scrofa*), warthogs (*Phacochoerus* spp.), bush pigs (*Potamochoerus larvatus* and *Potamochoerus porcus*) and giant forest hogs (*Hylochoerus* spp.). Most of these animals can develop clinical signs, although infections in warthogs seem to be subclinical or mild. 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. Warthogs are thought to be the primary wildlife reservoirs for the virus in Africa, although other wild suids might also play a role. Domesticated pigs also maintain ASFV.

Zoonotic potential

There is no evidence that ASFV infects humans.

Geographic Distribution

African swine fever is endemic in much of sub-Saharan Africa including the island of Madagascar. Outbreaks have been seen occasionally outside Africa, but the virus was almost always eradicated. It has, however, persisted on the Mediterranean island of Sardinia (Italy), where free-range production systems, uncontrolled pig movements and socioeconomic factors complicate control efforts. In 2007, ASFV was introduced into the Caucasus region of Eurasia, via the Republic of Georgia, and it has spread to domesticated swine and/or wild boars in a number of countries in this area. As of June 2019, infections had been reported as far west as the Baltic states, Romania, Bulgaria, Poland, Hungary and Belgium. In most cases, the virus appears to be spreading in wild boar, but domesticated pigs were also affected in some nations. Viruses that apparently originated from this outbreak were found in wild boar in Iran in 2010, but there have been no reports of ASFV in the Middle East since then. In 2018, a virus from Eurasia was detected in domesticated pigs in China. Since then, it has spread to pigs in other Asian countries including Vietnam, Mongolia, Cambodia, Lao and North Korea. Infected wild boar have also been detected in this region.

Transmission

African swine fever can be transmitted either with or without tick vectors as intermediaries. Domesticated pigs can shed ASFV in all secretions and excretions including oronasal fluid, urine and feces. Significant virus shedding can begin 2 days before the onset of clinical signs. Blood contains large amounts of the virus, and massive environmental contamination may result if blood is shed during necropsies or pig fights, or if a pig develops bloody diarrhea. Information about virus shedding in other suids is more limited; however, virus replication appears to be much lower in adult warthogs than pigs, and they are not thought to transmit the virus by direct contact.

ASFV can most likely enter the body through various mucous membranes after direct (non-tickborne) contact with infected pigs or the environment, but most animals are thought to be infected by inhalation or ingestion. Higher doses of the virus are generally required to infect a pig in solid feed, compared to inhalation, but ingestion of virus in liquids also seems to be efficient. Aerosolized viruses may contribute to transmission within a building or farm, but current evidence suggests that this only occurs over relatively short distances. Because ASFV can persist in tissues after death, it can be spread by feeding uncooked or undercooked pig swill that contains tissues from infected animals. Cannibalism of dead pigs might be important in some outbreaks.

Vector-mediated transmission occurs through the bites of some members of the soft tick genus *Ornithodoros*. In some parts of Africa, ASFV cycles between juvenile common warthogs (*Phacochoerus africanus*) and soft ticks of the *Ornithodoros moubata* complex, which 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 in Africa and the *Ornithodoros moubata* complex ticks that colonize their pens. *Ornithodoros erraticus* acted as a biological vector on the Iberian Peninsula during outbreaks 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, they 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. ASFV was found in swine lice (*Haematopinus suis*) collected from experimentally infected pigs. 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. Pigs also became infected when they were fed stable flies that had been been fed on infected blood. Fairly large numbers of flies were used to infect pigs in both of these experiments, but it is possible that transmission by other blood-sucking flies is more efficient.

How long pigs can remain infected with ASFV is uncertain. Some studies have found this virus in the tissues of domesticated pigs for as long as 2-6 months after experimental inoculation, and there are reports of virus shedding and transmission for at least 70 days. In other reports, pigs transmitted ASFV for less than a month. Longer transmission seems to be associated with less virulent viruses, which can cause chronic infections and persistent viremia. Recent studies that used highly virulent or moderately virulent ASFV circulating in Europe found that recovered pigs did not infect naive pigs via prolonged close contact after live virus could no longer be isolated from their blood. Some of these pigs were still PCR-positive at the time. Currently, there is no evidence that ASFV persists long-term in a latent state.

ASFV can spread on fomites, including vehicles, feed and equipment. It is reported to survive for several days in feces or urine at room temperature, and in feces for at least 11 days in one study where the sample was stored in the dark. One study estimated the infectious period for urine, based on the half-life of ASFV and the estimated dose required for infection, as 3 days at 37ºC (99ºF) and 15 days at 4ºC (39ºF). Feces was estimated to remain infectious at these temperatures for 4 and 8 days, respectively. ASFV is also reported to persist for a year and a half in blood or approximately 5 months in boned meat, both stored at 4ºC, and 140 days in salted dried hams, A recent study, which used pork products made from experimentally infected animals, isolated virus from dry cured salami at 18 days but not 26 days after processing, from dry cured pork belly at

60 but not 137 days and from dry cured loin at 83 but not 137 days. Pigs fed salami held for 26 days and pork belly or loin at 137 days, respectively, did not become infected. One source suggests that it may persist for several years in frozen carcasses, and unpublished findings from a report in the 1960s indicated that at least small amounts of infectious virus might persist in forest soil for nearly 4 months, in freshwater for up to 7 weeks in summer and approximately 6 months in winter, and on wooden boards or bricks buried in dirt for 2-3 months. However, this study injected pigs with virus, which may not be applicable to natural exposure. More recent investigations detected nucleic acids for several days to weeks in the soil where wild boar carcasses had been removed, but infectious virus could not be found. Few studies have examined virus transmission to pigs from fomites or other environmental sources, but in one recent report, pigs became infected when they were placed in pens that had housed animals with African swine fever, but not when the pens were left empty of pigs for 3 days or longer. These pens contained feces and urine, but visible blood had been washed away and bloodstained areas decontaminated. The length of virus persistence is likely to be influenced by the level of virus contamination.

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 and some iodine and quaternary ammonium compounds are reported to destroy ASFV on some nonporous surfaces. In one 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 (158ºF) for 30 minutes to inactivate ASFV; 30 minutes at 60ºC (140ºF) is sufficient for serum and body fluids. Virus in serum-free medium can also be inactivated by pH < 3.9 or > 11.5.

Incubation Period

The incubation period is reported to be 4 to 19 days in naturally-acquired cases.

Clinical Signs

African swine fever can present as a peracute, acute, subacute or chronic disease, and some animals may seroconvert without becoming ill. The course of the disease is generally correlated with the virulence of the virus, although a given virus can cause more than one form. Even in herds infected with highly virulent isolates, severely ill pigs are sometimes uncommon until the later stages of an outbreak, with most affected animals initially having mild, nonspecific clinical signs.

Sudden deaths with few lesions (peracute cases) may be the first sign of an infection in some herds. 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 experience diarrhea, constipation or vomiting and/or display signs of abdominal pain; the diarrhea is initially mucoid and may later become bloody. There may also be other hemorrhagic signs, including epistaxis and hemorrhages in the skin. Respiratory signs (including dyspnea), nasal and conjunctival discharges, and neurological signs have been reported. Pregnant animals frequently abort. Leukopenia and thrombocytopenia of varying severity may be detected in laboratory tests. Death often occurs within 7-10 days.

Subacute African swine fever is similar, but with less severe clinical signs. Fever, thrombocytopenia and leukopenia may be transient; however, hemorrhages can occur during the period of thrombocytopenia. Abortions are sometimes the first sign of an outbreak in this form. Affected pigs usually die or recover within 3 to 4 weeks. Petechiae and cyanotic lesions have been reported in some recovering animals.

Pigs with the chronic form have nonspecific signs such as an intermittent low fever, appetite loss and depression. Other signs may be limited to emaciation and stunting, but some 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 cases.

Post Mortem Lesions

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The gross lesions are highly variable, and are influenced 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 gastrohepatic and renal lymph nodes are affected most often. 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 of these 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 may also be found in the placenta.

Diagnostic Tests

Clinical samples generally include blood from live animals and tissues (especially spleen, kidney, tonsils, lymph nodes, liver, heart and lung) collected at necropsy. The spleen and lymph nodes usually contain the highest concentrations of virus, and viral DNA may persist longer in the spleen than other internal organs after death. Nucleic acids may also be detected in the bone marrow, which can be useful when other tissues from carcasses are not available or usable, and the intra-articular tissues of joints are sometimes tested in chronic cases. ASFV does not occur in aborted fetuses; in cases of abortion, a blood sample should be collected from the dam.

ASFV is usually isolated in primary porcine cells, including pig leukocyte or bone marrow cultures, porcine alveolar macrophages or blood monocyte cultures. ASFV-infected cells in the culture may be identified by their ability to induce hemadsorption of pig erythrocytes to their surfaces. However, a few non-hemadsorbing isolates can be missed with this test. Most of the latter viruses are avirulent, but some do produce illnesses, including chronic disease. Other methods to detect virus-infected cells include PCR and immunofluorescence, and PCR can be used to confirm the virus's identity. PCR can also detect nucleic acids directly in clinical samples. A wide variety of PCR tests have been described, with some real time assays reported to be more sensitive than others. Loop-mediated isothermal amplification assays (LAMPs) have been published.

ASFV antigens may be found in tissue smears or cryostat sections, as well as in buffy coat samples, using ELISAs or immunofluorescence. Antigens are easiest to detect in acute cases; the tests are less sensitive in subacutely or chronically infected animals. They are best employed as herd tests, and in conjunction with other assays. 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. Rapid penside lateral flow devices for antigen detection have been published.

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 assays include ELISAs, immunoblotting, indirect fluorescent antibody (IFA) and indirect immunoperoxidase (IPT) tests. The ELISA is prescribed for international trade, and is generally confirmed by immunoblotting, but IFA or IPT can also be used for confirmation.

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

Biosecurity measures (e.g., fences, restricted visitor access, good hygiene, disinfection of footwear or the use of dedicated footwear, closed herds, quarantines of new animals) help prevent virus introduction onto farms. Separation of the herd from wild suids, their environments and carcasses, as well as measures to prevent accidental human transport of ASFV, must be considered. In the past, many ASFV-free countries used heat treatment to inactivate viruses in pig swill and prevent the entry of ASFV. 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), some nations have completely forbidden feeding swill to pigs. In areas where this is not feasible, some sources recommend boiling the swill for at least 30 minutes, with frequent or continuous stirring. Solid walls without cracks are considered the optimal building material to discourage the establishment of *Ornithodoros* ticks and facilitate control. Acaricides are generally ineffective where wooden, stone, earth or overlapping metal walls/ fences provide hiding places for these ticks.

Some areas have successfully eradicated African swine fever outbreaks by standard stamping out measures (e.g., slaughter of infected and in–contact animals, sanitation,

disinfection, movement controls and quarantines), but more complex measures were needed in some regions. On the Iberian Peninsula, ASFV became established in wild boar and *Ornithodoros erraticus* ticks in the 1960s, 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, but the minimum quarantine is 6 years if vectors are thought to be involved in transmission.

Current control measures in wild boar mainly focus on reducing their numbers, as higher population densities are thought to facilitate maintenance of the virus, and attempting to discourage the movements of infected animals. Authorities in the Czech Republic apparently controlled a single focus of infection in wild boar with intensive measures that included fencing, trapping and targeted hunting. Biosecurity measures to reduce the risk of transporting ASFV during hunting (e.g., the use of leak-proof vessels to remove carcasses and store offal, limits on vehicles in infected areas, precautions for cleaning and disinfecting tools) have been recommended. Eradication of ASFV from some wild reservoirs in Africa, such as warthogs, appears unlikely. However, compartments where African swine fever is controlled and barriers (double fencing) prevent contact with wild reservoirs have been established in some parts of Africa where warthog-mediated introduction is a concern.

No vaccine is currently available.

Morbidity and Mortality

The morbidity rate for African swine fever can approach 100% in naïve herds of domesticated pigs. Cumulative mortality depends on the virulence of the isolate, and can range from < 5% to 100%. It is usually 30-70% in subacute cases. However, viruses can sometimes take days to weeks to spread through a herd, and initial herd mortality rates may be low even when the case fatality rate is high. Less virulent isolates are more likely to kill pigs with concurrent diseases, pregnant or nursing sows, and young animals. Morbidity and mortality rates also tend to be higher when ASFV is introduced into new regions, with an increased incidence of subacute and subclinical cases once it becomes endemic. Chronic African swine fever was first described during outbreaks on the Iberian Peninsula, and some authors speculated that the viruses that cause this form might have originated from live attenuated vaccine strains tested at the time. However, chronic disease has since been reported in pigs that were experimentally infected with recent European strains, and it has also been seen in Angola. Some populations of pigs in Africa are reported to be more resistant to African swine fever than others, but the basis for this resistance is not known.

The role of wild suids in spreading African swine fever differs between regions. Warthogs cause some outbreaks in Africa, but, at present, domesticated pigs seems to be driving virus spread in many African countries. In the current outbreaks in Europe, ASFV appears to persist in wild boar populations independently of outbreaks among domesticated pigs. Although previous experiences suggested that the virus would eventually die out in these animals if it was not reintroduced, neither explosive outbreaks nor self-extinction has been reported to date. Instead, the virus has been spreading slowly and steadily across Europe in wild boar. Why this is occurring is unclear. The high density of animals in many areas probably plays a role, but virus spread has also occurred in areas where wild boar density is low. Other factors likely to influence ASFV transmission rates include wild boars' social structure, where family groups are prominent, and exposure to infectious carcasses. In 2019, one study found that the incidence of ASFV had decreased for the first time in wild boar in Estonia. The density of wild boar is relatively low in this area, and decreased further after virus introduction, possibly due to the effects of the disease as well as deliberate measures to decrease animal numbers. Whether this will eventually lead to the extinction of ASFV in Estonia is still uncertain.

There is currently no evidence that *Ornithodoros* ticks play any role in the current European outbreak, but some authors note that there is relatively little information on their distribution, and members of the *O. erraticus* complex were known to occur in the Caucasus at one time. Recent studies of exposure to *Ornithodoros* have reported strong positives among backyard pigs in the southern parts of the Russian Federation. There is little or no information about whether wild boar or ticks play any role in the outbreaks in southern Asia, but the virus is spreading rapidly in some domestic herds.

Internet Resources

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

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

FAO. Updates on the ASF Situation Worldwide (with links to African swine fever information)
http://www.fao.org/ag/againfo/programmes/en/empres/ASF/situation_update.html

FAO: African Swine Fever: Detection and Diagnosis – A Manual for Veterinarians (English version)
http://www.fao.org/3/a-i7228e.pdf

The Merck Veterinary Manual
http://www.merckvetmanual.com/

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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