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 (ASF) is an important viral disease of pigs that has spread extensively throughout the world since 2007 and has become a serious threat to pork production. 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 effective treatment, and severely affected pigs usually die. The spread of ASF virus (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 can act as biological vectors. 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.

For many years, African swine fever was mostly confined to sub-Saharan Africa, where viruses from wild warthogs, thought to be the original host, had become widely established in domesticated pigs. In 2007, ASFV was accidentally introduced into the Caucasus region of Eurasia, most likely in pig swill. This highly virulent virus caused outbreaks on pig farms, became established in wild boar, and has since spread across much of Europe and Asia. It was recently introduced to the island of Hispaniola (Haiti and the Dominican Republic) in the Caribbean, and there is a possibility it might occur among wild boar in parts of the Middle East. The virus has also diversified, generating isolates of widely varying virulence, which are less likely to be recognized quickly. Controlling this outbreak has been difficult, with reports of recurrence in wild boar populations where ASFV was thought to have been eliminated, and a number of difficulties in developing an effective vaccine that does not revert to virulence.

Etiology

African swine fever results from infection by African swine fever virus, an enveloped virus in the genus *Asfivirus* and family Asfarviridae. ASFV isolates differ greatly in virulence, ranging from highly pathogenic viruses that kill most pigs to strains that result only in seroconversion. At least 24 genotypes of ASFV have been identified, many from wildlife cycles in Africa. A number of these genotypes have also been found at some time in domesticated pigs, though only some are adapted to efficient transmission in these animals.

One genotype I virus has been endemic in Sardinia (Italy) since the 1960s, while the virus introduced into Eurasia in 2007 belongs to genotype II. Although the latter virus was highly virulent when introduced, its descendants have diversified, with isolates of varying virulence now circulating in Europe and Asia. In addition, a genotype I virus of low virulence, possibly introduced via an unauthorized vaccine, was identified in some herds in China and has recombined with genotype II viruses. At least some of these recombinants, which have since been found in other parts of Eurasia, are highly virulent.

Species Affected

African swine fever affects members of the pig family (Suidae). Species known to be susceptible to infection include domesticated pigs and wild boar (both subspecies of *Sus scrofa*), warthogs (*Phacochoerus* spp.), bush pigs (*Potamochoerus larvatus* and *Potamochoerus porcus*), giant forest hogs (*Hylochoerus* spp.) and some wild Asian suids such as *Sus barbatus* and *Sus cebifrons*. Most of these animals can develop clinical signs, although infections in warthogs mostly seem to be subclinical or mild. Some older reviews and textbooks suggest that peccaries (*Tayassu* spp.) may 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 to ASFV. Known maintenance hosts for the virus include domesticated pigs, wild boar and warthogs, while the role of other wild suids is unclear.

Zoonotic potential

There is no evidence that ASFV infects humans.

The Center for Food Security & Public Health

Institute for International Cooperation in Animal Biologics

Iowa State University College of Veterinary Medicine

World Organisation for Animal Health Founded as OIE

USDA

Geographic Distribution

African swine fever is endemic in much of sub-Saharan Africa and some nearby islands, e.g., Madagascar. Outbreaks were seen occasionally outside Africa before 2007, affecting parts of Europe, the Caribbean (Cuba, Hispaniola) and South America (Brazil). These viruses were all eventually eradicated, with the exception of a genotype I virus that has persisted for decades on the Mediterranean island of Sardinia (Italy).

A genotype II virus introduced into the Republic of Georgia in 2007 has since spread to domesticated swine and/or wild boars in many countries in Europe and Asia. In 2021, viruses from this outbreak became established in Hispaniola (Dominican Republic and Haiti), which is part of the Greater Antilles group of islands in the Caribbean. They were also found in wild boar in Iran in 2010, though there have been no reports of ASFV in the Middle East since then. Genotype I viruses, possibly introduced via the use of live attenuated vaccines, have been detected in some herds in China, and genotype I/II recombinants have since been found in China and other locations, including Vietnam and Russia.

Although some countries in the affected regions maintain an ASF-free domestic pig population, most reports of apparent virus elimination from wild boar in Europe have been followed by either recrudescence after low levels of virus circulation among boar (e.g., Estonia) or apparent reintroduction from neighboring countries (e.g., Czech Republic).

Transmission

African swine fever can be transmitted either directly between animals or by the soft tick genus *Ornithodoros,* which acts as a biological vector. Domesticated pigs and wild boar can shed ASFV in most or all secretions and excretions including oronasal fluid, urine, feces and semen. While some pigs may begin to shed this virus 2 days before the onset of clinical signs, some studies suggest that the majority of virus shedding occurs late in the illness. Blood and tissues contain large amounts of virus, and massive environmental contamination may result if blood is shed during necropsies or pig fights, or if a pig develops bloody diarrhea. Although animals that recover can apparently clear the virus completely, some pigs may become chronically infected and continue to shed the virus. There is currently no evidence that viable ASFV persists long-term in a latent state in asymptomatic, healthy animals that have no evidence of viremia. Information about virus shedding in other suids is more limited; however, virus replication is much lower in adult warthogs, which do not seem to readily transmit the virus by direct contact.

Most pigs are thought to become infected with ASFV via inhalation or ingestion. Because this virus 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 animals might be important in some outbreaks. Fairly high doses of the virus are generally required to infect a pig in solid feed, though ingestion of the virus in liquids seems to be more efficient. Inhalation requires only low viral doses. Aerosolized viruses may contribute to transmission within a building or farm, but current evidence suggests that this only occurs over relatively short distances. Boars can infect sows by artificial insemination as well as during natural breeding. Transplacental transmission has also been demonstrated; however, all of the infected fetuses reported to date either died *in utero* or shortly after birth.

Ornithodoros spp. ticks, which can infect suids either through bites or when they are eaten by the pig, are biological vectors for ASFV. In some parts of Africa, these viruses cycle between juvenile warthogs and soft ticks of the *Ornithodoros moubata* complex, which live in their burrows. Transstadial, transovarial and sexual transmission has been demonstrated in these ticks. A similar cycle can occur between domesticated pigs and the *Ornithodoros* spp. ticks that colonize their pens, and *O. erraticus* helped maintain ASFV on the Iberian Peninsula during the European outbreaks in the 1950s and 1960s. *Ornithodoros* spp. ticks are long-lived, and ASFV has been reported to persist in their colonies for several years (e.g., 5 years in *O. erraticus*). However, they can eventually clear the virus if they are not reinfected. Other blood-sucking insects such as various biting flies (e.g., *Stomoxys* spp., tabanids), mosquitoes, hard ticks or swine lice (e.g., *Haematopinus suis*) might be able to transmit ASFV mechanically for a short time, though their significance in transmission, if any, is still unclear. In addition, some potential mechanical vectors, such as swine lice, have limited mobility.

ASFV can also be spread on fomites, including vehicles, feed and equipment. Except in animal tissues, most studies of its persistence under controlled laboratory conditions, using various fomites including soil and unprocessed grains, have reported survival times ranging from less than a day to approximately 1-2 weeks, at temperatures from 4ºC (39ºF) to around 20ºC (68ºF). Survival was generally longer under cold conditions and when the virus was protected in feces or other organic matter. There are, however, a few outliers among these studies. One reported that ASFV was infectious for more than 16 weeks in sterilized soil samples at 4ºC, and for either 2 or 3 weeks at 22ºC (72ºF), depending on whether or not sheep erythrocytes were added to the soil. Another study found that, in three different sterilized feed components, the virus remained viable for 5 days to 4 months at 4ºC, 1 day to at least 21 days at 20ºC , and < 1 day to at least 7 days at 35ºC (95ºF). Particularly long survival was found in sterilized soybean meal. Whether studies of virus persistence in sterilized materials accurately reflect real world conditions is uncertain, as the presence of other microorganisms can be detrimental to the survival of some viruses.

Studies on the persistence of ASFV in water, which might protect an enveloped virus from desiccation, reported that it remained viable in sterilized or distilled water for as long as 3-7 weeks at 22-24°C (72-75ºF), and for at least 2 months either at 4-6°C (39-46ºF) or when frozen at -16 to -20°C (-4 to 3ºF). A study that used samples of river water found that ASFV remained viable for at least 42 days at 4°C,

28 days at 15°C (59ºF), and 14 days at 21°C, but survived longer in distilled water. While these studies are likely to be applicable to water sources such as drinking troughs, their relevance to natural water sources such as rivers or lakes, where added viruses would be diluted to much lower levels, is unclear. The survival of ASFV in water appears to be reduced by the presence of ciliates but prolonged by freshwater snails. It has been reported to accumulate in the tissues of snails and leeches; however, feeding these snails to pigs did not result in infections, and the virus appeared to enter an abortive replication cycle in the leeches.

ASFV can persist for relatively long periods in refrigerated, frozen or preserved pig tissues, including a year and a half in blood and approximately 5 months in boned meat, both stored at 4ºC, and 140 days in salted dried hams, with one study suggesting that it might persist for years in frozen carcasses. A study that used pork products made from experimentally infected animals detected live viruses in dry cured salami at 18 days but not 26 days after processing, in dry cured pork belly at 60 but not 137 days, and in dry cured loin at 83 but not 137 days. Studies that examined various porcine tissue and organ samples, generally as a proxy for virus survival in carcasses in the environment, reported that, depending on the substrate, ASFV persisted for periods ranging from a month to 2 years when frozen at -20ºC or stored at 4°C, though infectious virus usually could not be found in tissues after 1-2 weeks at 20ºC.

There is limited field data on ASFV survival outside the laboratory, where it might be destroyed more rapidly on fomites by sunlight and desiccation, and by rigor mortis and various decomposition processes in carcasses. Some studies found that pigs did not become infected when placed in pens that had housed animals with clinical signs 3-5 days earlier, if there was either no gross contamination with blood or any visible blood had been washed away and bloodstained areas decontaminated. In another report, researchers could not recover any infectious virus from pens 7 days after the removal of experimentally infected pigs with clinical signs that included bloody diarrhea. No infectious virus was found by three groups of researchers who examined the soil where naturally infected wild boar carcasses had been removed, looked for the virus in the bones of buried and unburied boar carcasses, or tested soil and organ samples from wild boar carcasses buried 18 days to 15 months earlier, though PCR tests detected viral nucleic acids in each of the 3 experiments. Viable ASFV was also reported to be absent from composted pig carcasses after 3 days.

Disinfection

ASFV has been shown to be susceptible to a number of disinfectants including sodium hypochlorite, sodium hydroxide (caustic soda) and glutaraldehyde, as well as certain phenols, iodophors, peroxygens (e.g., potassium peroxymonosulfate), acids (citric, acetic), quaternary ammonium compounds and multi-component disinfectants such as Virkon™. As with other viruses, some disinfectants are more effective than others in the presence of significant soiling, low environmental temperatures can affect the efficacy of certain agents, and prolonged contact time may be needed in some circumstances.

Heat is also effective in destroying this virus. Although 30 minutes at 60ºC (140ºF) is reported to be sufficient to inactivate ASFV in serum and body fluids, unprocessed meat must be heated to at least 70ºC (158ºF) for 30 minute. Recommendations from different agencies for the heat inactivation of pig swill have varied. A recent study's theoretical calculations, based on observations of inactivation in small amounts of several different swills, suggest that inactivation may occur within a few minutes at 90ºC (194 ºF) but requires at least 2 hours at 70ºC. Whether all of the swill will reach a given temperature must, however, also be considered. ASFV in serum-free medium can be inactivated by pH less than 3.9 or greater than 11.5.

Incubation Period

The incubation period in domesticated pigs is reported to be 4-19 days in naturally-acquired cases and 2-21 days after experimental inoculation, with highly virulent viruses often affecting animals within a week. Some comparative studies suggest that wild boar might develop clinical signs more quickly than pigs after oronasal exposure.

Clinical Signs

African swine fever can present as a peracute, acute, subacute or chronic illness. Some infected animals may also seroconvert without becoming ill. The course of the disease is generally correlated with the virulence of the virus; however, a given virus can cause more than one form, and some animals in a herd may remain asymptomatic or develop only transient signs while others become severely ill. 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.

Pigs with peracute African swine fever die rapidly, usually with few or no clinical signs, while animals with acute cases typically have a high fever with nonspecific signs such as anorexia, lethargy, weakness and recumbency. Erythema may be evident, particularly in white animals, and some animals develop cyanotic skin blotching. The latter sign tends to be noted most often on the ears, snout, tail and perianal region, abdomen, lower legs and/or hams. Gastrointestinal signs including diarrhea, constipation, vomiting and signs of abdominal pain are also common. The diarrhea is initially mucoid and may later become bloody. Some pigs can also develop other hemorrhagic signs, such as epistaxis or cutaneous petechiae and ecchymoses. Respiratory signs (including dyspnea), nasal and conjunctival discharges are common in some outbreaks, 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. In some herds, abortions may be the first sign of these outbreaks. Thrombocytopenia and leukopenia may be transient in this form; however, petechiae or other hemorrhagic signs can sometimes be seen. Pigs with the subacute form of African swine fever usually die or recover within 3-4 weeks. In one outbreak, most sows that had recovered clinically but were still PCR positive either aborted or gave birth to stillborn and/or mummified fetuses. Some of these sows also produced live neonates with congenital tremors and ataxia that died within the first few days. Viral antigens and nucleic acids were found in the brains of the piglets with neurological signs, suggesting that African swine fever was the cause.

Chronically infected pigs usually have nonspecific signs such as an intermittent low fever, appetite loss and depression, and may be thin, emaciated and/or stunted. Some animals also cough or have other signs of respiratory issues, develop swollen joints, or have gastrointestinal signs such as diarrhea and/or occasional vomiting. Ulcers and reddened or raised necrotic skin foci may appear over body protrusions and other areas subject to trauma. Some of the clinical signs seen in these animals may be due to secondary infections. Chronically infected animals may eventually die.

Signs in experimentally infected wild boar are similar to those in domesticated pigs; however, some studies reported that they became ill more rapidly and died sooner. Skin lesions may also be absent or less evident in wild boar, with erythema usually limited to areas of thinner or hairless skin such as the abdomen and inguinal region. Some runted animals inoculated 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 of Africa swine fever are highly variable, and are influenced by the virulence of the isolate and the course of the disease. Animals that die peracutely may have few or poorly developed lesions, while subacutely infected animals sometimes have more widespread organ involvement than those with acute cases.

The carcass is often in good condition in animals that die relatively soon after they become infected, but there may be bluish-purple discoloration and/or hemorrhages in the skin, and some pigs have external indications of bloody diarrhea or other internal hemorrhages. The major internal lesions are hemorrhagic, and can be seen most consistently in the spleen, lymph nodes, kidneys and heart. The spleen can be very large, friable, and dark red to black in some pigs; in others, it is enlarged but not friable, with a color closer to normal. The lymph nodes are often swollen and hemorrhagic, and may look like blood clots. Many reports indicate that the abdominal lymph nodes, including the gastrohepatic, mesenteric, ileocecal and renal nodes, are affected most often; however, the submandibular and medial retropharyngeal nodes also had similar lesions in some animals exposed to the virus via the oronasal route. Petechiae are frequently noted on the cortical and cut surfaces of the kidneys, and sometimes in the renal pelvis, and perirenal edema may be evident. Hemorrhages, petechiae and/or ecchymoses may also be found in other organs including the urinary bladder, lungs, stomach and intestines, and pulmonary edema and congestion can be prominent in some pigs. Other reported lesions include congestion of the liver, edema in the wall of the gall bladder and bile duct, and straw-colored or blood-stained fluid in the pleural, pericardial and/or peritoneal cavities. In some cases, the brain and meninges are congested, edematous or hemorrhagic.

Lesions that have been noted in aborted fetuses in some reports include generalized edema (anasarca), a mottled liver, hemorrhagic tonsils, excess abdominal fluid, and petechiae or ecchymoses in the skin, myocardium and other organs. Petechiae may also be detected in the placenta. Five newborn piglets with congenital tremors had enlarged and hemorrhagic mandibular and inguinal lymph nodes, petechiae in the kidney, and congested meninges. One piglet also had hydranencephaly.

The carcasses of pigs with chronic African swine fever are often in poor condition, thin or emaciated. These animals may have a variety of lesions including focal areas of skin necrosis, skin ulcers, consolidated lobules in the lung, caseous pneumonia, nonseptic fibrinous pericarditis, pleural adhesions, generalized lymphadenopathy and swollen joints.

Diagnostic Tests

Commonly recommended samples to detect ASFV, its nucleic acids and antigens include blood from live animals or 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. Most secretions and excretions are also likely to contain virus during the clinical stage in live animals, but there is little or no published information for using these samples in diagnostic testing, except in aggregate pen-based surveillance, such as ropes for saliva testing. Other types of samples, such as ear punches, have also been investigated for PCR-based surveillance of carcasses.

A blood sample from the dam has usually been recommended in cases of abortion, as ASFV was not thought to occur in the fetus. However, some fetuses were recently found to have at least low levels of viral DNA in tissues such as the tonsils, spleen, lymph nodes, liver and lung, as well as the placenta. ASFV was successfully isolated from the placenta, amniotic fluid and fetal blood after abortions caused by a low virulence isolate, though the fetal tissues showed little or no immunostaining for viral antigens. Both viral nucleic acids and antigens were detected in the brain and cerebrospinal fluid of neonatal piglets with congenital

tremors; however, the virus appeared to be absent from other tissues including the spleen, kidney and lymph nodes.

Clinical cases of African swine fever are often diagnosed with various PCR tests. Loop-mediated isothermal amplification (LAMP), recombinase polymerase amplification (RPA) and recombinase- aided amplification (RAA) assays to detect ASFV nucleic acids have also been published. ASFV antigens may be found in tissues or buffy coat samples with ELISAs or immunofluorescence. Rapid penside lateral flow devices for antigen detection have also been described, and may be available as commercial tests in some countries. Antigens are easiest to find in acute cases, and antigen-detection tests sometimes have relatively low sensitivity; thus, they are best employed as herd tests, in conjunction with other assays. A hemadsorption "autorosette" test was also used in the past to detect ASFV directly in peripheral blood leukocytes, but it has mostly been replaced by PCR.

If necessary, ASFV can be isolated in primary porcine cells including pig leukocyte or bone marrow cultures, porcine alveolar macrophages or blood monocyte cultures. Primary cells are complicated to work with, and reports of cell lines that might be able to replace these cells, such as MA-104 cells, have been published. Additional cell lines (e.g., COS-1 cells, Vero cells) can be used to propagate viruses that have been adapted to cell culture, but none of these cells is susceptible to all field strains, limiting their usefulness for diagnosis. Traditionally, ASFV-infected cells have been identified in cultures by their ability to induce hemadsorption of pig erythrocytes to their surfaces, but a few non-hemadsorbing isolates, including some that cause chronic disease, can be missed with this test. Virus-infected cells can also be detected by PCR or immunofluorescence, and PCR can be used to confirm the virus's identity.

Pigs with acute disease often die before seroconverting; however antibodies to ASFV persist for long periods in animals that survive. The currently used serological assays include ELISAs, immunoblotting, indirect fluorescent antibody (IFA) and indirect immunoperoxidase (IPT) tests, and penside lateral flow assays to detect antibodies have been commercialized in some countries. 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

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, including quarantines of new animals, reduce the risk of introducing ASFV onto a farm. Separation of the herd from wild suids, their environments and carcasses (e.g., with double fencing), must also be considered in many areas. In the past, many ASFV-free countries used heat treatment to inactivate any viruses that might be present in pig swill. Due to the risk that ASFV 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 in domesticated pigs with 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, and minimum quarantines were much longer (e.g., 6 years rather than 40 days) where the tick vectors were thought to be involved in transmission. Because ASFV often spreads relatively slowly through a herd, some Asian countries where this virus has become common have recently implemented partial or selective culling of herds, rather then complete depopulation, as part of their control efforts. The success of this measure remains to be determined, but potential benefits include an incentive for early reporting of outbreaks, as well as reduced losses for farmers.

Current control measures in wild boar mainly focus on reducing their numbers by hunting and/or trapping, as higher population densities are thought to facilitate maintenance of the virus, and attempts to discourage the movement of infected animals with fencing or other means. In some countries, authorities also search for and remove wild boar carcasses. Campaigns to eliminate the virus from wild boar populations with these measures have often met with limited success, and appear to be more effective when there is a single geographical focus of infection. 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 also been published. Eradication of ASFV from some wild reservoirs in Africa, such as warthogs, appears unlikely, but compartments where African swine fever is controlled and barriers (double fencing) prevent contact with wild reservoirs have been established in some regions where warthog-mediated introduction is a concern.

Vaccines have been difficult to develop against ASFV, which has a large and complex genome. Although two live attenuated vaccines have been licensed in Vietnam, pigs can shed the vaccine viruses, raising concerns about reversion to virulence or recombination with field strains. Some vaccinated animals also experienced health problems or died, and both vaccine viruses were recently shown to revert to virulence on serial passage in pigs. One resulted in mild clinical signs and prolonged viremia after 5 passages, and the other caused severe clinical signs and high mortality after 3-4 passages.

Morbidity and Mortality

ASFV currently circulates in warthogs as well as domesticated pigs in parts of Africa, and in both pigs and wild boar in parts of Eurasia. While previous experiences in Europe suggested that this virus would eventually die out in wild boar if it was not reintroduced from pigs, this has not happened. Instead, it has spread slowly in boar populations and diversified, independently of its occurrence in farmed pigs. In Estonia, initial high mortality rates among wild boar, together with control measures, resulted in a significant decline in the numbers of these animals and the apparent disappearance of the virus; however, it continued to circulate at low levels (which were not detected at the time by surveillance) and the recovery of the boar population was accompanied by the resurgence of the virus.

The importance of wild boar in infecting domesticated pigs (or vice versa) may differ between regions. In some areas, especially those with large numbers of backyard pigs, most farms seem to become infected from other farms rather than wild suids. Outbreaks appear to be somewhat seasonal in both pigs and wild boar, though the reported patterns differ between pigs and boar, and between farms in different countries. Patterns of pig marketing, including changes in the demand for pork products, and other cultural factors are generally thought to account for the seasonality in domestic herds, though it remains possible that other factors, such as arthropod-mediated transmission, might also play a role. In wild boar, seasonality might be caused by changes in wild boar interactions (e.g., mating season), as well as longer virus survival under cold conditions; however, it is possible that the observed variations are just an artifact of human observations, for instance the increased likelihood of detecting dead boar carcasses or infected animals at times when more people are in the woods.

The morbidity and mortality rates for African swine fever can be influenced by the virulence of the isolate, the dose of the virus, and animal factors such as previous exposures and general health. Morbidity rates can approach 100% in some naive herds of domesticated pigs, while cumulative mortality ranges from < 5% with mild or attenuated viruses to 100% from highly virulent viruses. The mortality rate is usually around 30-70% in subacute cases. 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 a new region, with an increased incidence of milder cases once it becomes endemic. Because ASFV sometimes takes days to weeks to spread through a herd, initial herd mortality rates may be low even when the case fatality rate is high.

Both domesticated pig and wild boar populations can maintain a variety of ASF isolates of varying virulence. Some viruses have been shown to cause more severe disease in wild boar than pigs, though it is not known whether this is true for all or most strains. Chronic African swine fever was first described during outbreaks on the Iberian Peninsula, leading to speculation that the viruses that cause this form might have originated from live attenuated vaccine strains tested at the time. However, it can also be caused by virulent viruses, including some recent Eurasian strains. Pigs that recover from African swine fever are often resistant to illness upon subsequent exposure, though they may become reinfected and shed the virus. However, this protection does not necessarily extend to all other strains. Certain populations of pigs in Africa seem to be relatively resistant to African swine fever, but the basis for this resistance is not known.

Internet Resources

CIRAD Pigtrop (Pig Production in Developing Countries)

Food and Agriculture Organization of the United Nations (FAO). Recognizing African Swine Fever. A Field Manual.

FAO. Updates on the ASF Situation Worldwide (with links to African swine fever information)

FAO: African Swine Fever: Detection and Diagnosis – A Manual for Veterinarians (English version)

The Merck Veterinary Manual

United States Animal Health Association. Foreign Animal Diseases

World Organization for Animal Health (WOAH)

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals; WOAH Terrestrial Animal Health Code

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