African swine fever (ASF)

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INTRODUCTION

African swine fever (ASF) is a highly lethal viral disease of domestic pigs that manifests as an acute or peracute haemorrhagic fever. Subacute and chronic forms have been described but are unusual under natural conditions. It is caused by a large DNA virus, Asfivirus, the only member of the family Asfarviridae, which was created for it in 2000. It is the only known biologically-transmitted DNA arbovirus. All ASF viruses are considered to belong to a single serotype, but more than 20 genotypes have been identified, most of which occur naturally in eastern and southern Africa. In spite of half a century of research no vaccine is available, although efforts to develop one are ongoing. Its classical area of distribution is southern and eastern Africa, where it exists in an ancient sylvatic cycle between warthogs (Phacochoerus aethiopicus) and argasid ticks of the Ornithodoros moubata complex. It was first recognised as an entity different from classical swine fever in Kenya and was described as such in 1921. Soon after that it was reported from Angola and South Africa. Its arrival in Portugal, probably from Angola, in 1957 and again in 1959, with spread to Spain and subsequently to a number of countries including Brazil demonstrated its catastrophic effects on highly developed pig industries. Its eradication from the Iberian Peninsula took more than 30 years, and it is endemic in the Italian island of Sardinia, as well as in most sub-Saharan African countries where pigs are kept. It was introduced into the Caucasus in 2007 and efforts to eradicate it are ongoing.

(Also refer to the World Animal Health Information Database (WAHID))
Livestock Health, Management and Production › High Impact Diseases › Vector-Borne Diseases › African Swine Fever

Video link: [http://www.youtube.com/watch?v=dkGcsuS7qHM](http://www.youtube.com/watch?v=dkGcsuS7qHM)

**EPIDEMIOLOGY**

ASF classically is confined to the southern and eastern savannas of sub-Saharan Africa, where it exists in an ancient cycle between warthogs and eyeless tampans that live in their burrows. Warthogs are impervious to the pathogenic effects of the virus and develop no clinical signs of disease. Neonatal warthogs spend the first 4 – 6 weeks of life in the burrows where they are born. *Ornithodoros* tampans that live in the burrows feed on the young warthogs and can infect them with ASF virus. In spite of colostral antibodies to the virus, the young warthogs when infected develop a very high viraemia, sufficient to infect further tampans, although they apparently suffer no ill effects from the virus. The tampans also feed on older warthogs, but these do not become viraemic owing to their immunity, and are therefore not able to transmit the virus. Although ASF virus can be detected in lymphoid tissues of warthogs, it has proven difficult to infect domestic pigs by feeding them on warthog tissues. Other African wild pigs, bush pigs (*Potamochoerus porcus*, *P. larvatus*) and giant forest hogs (*Hylochoerus meinertzhageni*), are susceptible to infection with ASF virus but are also resistant to the pathogenic effects. Their role in the epidemiology of ASF, if any, is unknown.
In sub-Saharan Africa where the disease is endemic, ASFV has lived in an inapparent sylvatic cycle with its invertebrate host the tampan and its vertebrate host, wild pigs, mainly warthogs.

Tampans (*Ornithodoros* sp.) that live in pig shelters (kholas) in which pigs are kept at night have been found to be infected with ASF virus.

In large parts of Africa, ASF virus exists in an ancient cycle between warthogs and soft-shelled, eyeless tampans that live in warthog burrows often found at the base of termite mounds.

When domestic pigs were introduced into Africa, exposure to ASF virus resulted in lethal disease that killed up to 100% of affected pigs. In Kenya and South Africa it was soon noticed that ASF appeared to be associated with proximity to warthogs, and by ensuring a good separation through the use of double fencing it was possible to prevent the disease in domestic pigs. However, the involvement of *Ornithodoros* in maintaining and transmitting the virus was first discovered in Spain, where *O. marocanus* (= *O. erraticus*) lives in rural pig sties. Investigation of *Ornithodoros* from warthog burrows demonstrated that these tampans are able to transmit the virus vertically, trans-stadially, and sexually (males to
females). O. marocanus transmits the virus trans-stadially and sexually, but vertical transmission has not been demonstrated. Ornithodoros tamps develop slowly and are capable of long periods of survival in a dormant state when no hosts are available. Ornithodoros marocanus is able to maintain ASF virus in infective amounts for at least five years. An outbreak of ASF that occurred in Portugal in 1999, five years after formal declaration of freedom from ASF, was associated with introducing pigs into shelters that were occupied by O. marocanus. In Malawi, a cycle of maintenance and transmission has been demonstrated between free-ranging domestic pigs and O. moubata tamps that infest the shelters the pigs occupy at night. Experimental studies have shown that several species of Ornithodoros are capable of transmitting ASF virus to pigs.

Apart from transmission by tamps, ASF is a highly contagious disease that is capable of very rapid spread in domestic pigs. Infected pigs shed large quantities of the virus in all their excretions and secretions, and in an outbreak the usual sources of infection are infected pigs and objects contaminated by infected pigs (fomites). Pigs may shed virus for up to 48 hours before clinical signs appear. Airborne transmission has been demonstrated experimentally to occur only over short distances (two metres or less) in closed housing without barriers between the pigs, so is unlikely to be of any importance. Of a large number of arthropods investigated, apart from Ornithodoros, only stable flies of the genus Stomoxys are capable of maintaining and transmitting the virus mechanically for up to 48 hours. Since the dose of virus required to cause disease is fairly high (10^4 to 10^5 HAD50 to infect 50% of pigs by the oral route), water (e.g. rivers, lakes) is an unlikely source of infection, and animals such as rodents, birds and even dogs and cats would be unable to carry sufficient ASF virus to cause infection. Neither vertical nor sexual transmission has been demonstrated to occur.
When protected in a suitable protein environment, ASF virus is resistant over a wide range of temperatures and pH. Meat from infected pigs is therefore an important source of infection for pigs that scavenge for their food or that are fed swill that could contain uncooked or undercooked pork. Exposure to 56°C for at least 20 minutes is required to inactivate the virus. The virus can remain viable for 15 weeks in chilled meat and up to six months in processed hams, and may persist longer in frozen meat. However, ASF virus is rapidly destroyed by desiccation and exposure to sunlight. Pig sties in a tropical country were shown, even without cleaning after the pigs occupying them had died of ASF, to be safe after five days but not after three. Because of its ability to remain stable within a pH range of 4 - 10, ASF virus is not destroyed by pH-dependant disinfectants or by maturing pork.

In naïve pig populations, up to 100% of infected pigs die. However, a small proportion of domestic pigs have an inherent resistance to the pathogenic effects of ASF virus, and survive infection with few or no clinical signs, the fact that they were infected only being indicated by the presence of antibodies later. Such pigs eliminate the virus rapidly and do not become long-term carriers, although the antibodies may persist for at least 3 years, and viral DNA may be detectible in lymphoid tissues for at least eight months.
However, in sufficiently large and continuous populations of pigs, as may be found in densely populated areas with pigs raised in traditional free-range systems in Africa, or the dense European wild boar population of Sardinia, virus circulation may be maintained indefinitely, since there are always new pigs to infect. If pigs are infected with a virus of lower virulence, as has been reported in Angola and possibly DRC, as well as in Spain and in countries infected from Spain, some pigs may develop subacute or chronic forms of ASF. These pigs remain infectious to other pigs until they finally succumb, usually less than a year after the first infection. In the Iberian Peninsula it is likely that attempts to produce a vaccine may have resulted in viruses of lower pathogenicity, since the ASF virus is, like most DNA viruses, relatively stable over time, and the virus that infected Sardinia, for example, remains highly virulent in spite of more than 30 years of circulation.

The introduction of ASF to areas outside sub-Saharan Africa, as well as possibly to West Africa, although this is debatable, is generally attributed to pigs having access to airport or harbour swill that contained infected meat. This usually occurs either when scavenging pigs have access to disposal sites or when the material becomes available to be fed to pigs by their owners.

The first extra-African outbreak occurred in Portugal in 1957, and was rapidly eradicated. However, it was re-introduced in 1959, spread to Spain in 1960 and became established in the Iberian Peninsula until 1993, when Spain was declared free, followed by Portugal in 1994. During this period ASF occurred in a number of western European countries as well as in Cuba, Haiti, Dominican Republic, and Brazil. It was eradicated from all of them except for the island of Sardinia, where sporadic outbreaks have occurred ever since its introduction in 1978.

ASF was first formally reported from West Africa when Senegal reported outbreaks to the OIE in 1978, followed by Cameroon in 1982. However, it has since been reported that there was an outbreak in Nigeria in 1973 and that it occurred in Guinea Bissau and the Cape Verde islands as early as 1960. Since restriction fragment length polymerase (RFLP) studies demonstrated the Cameroon virus to be the same as the one that infected Europe, it was suggested that the outbreaks may have been due to a re-introduction into Africa of the virus from Europe. However, genotype I viruses are widespread in West and Central Africa and movements of pigs and their products largely unknown.

In 1996 ASF appeared for the first time in Côte d’Ivoire. Although it was apparently eradicated by the end of the year, this marked the start of a rolling pandemic that affected Benin, Togo, Nigeria, Ghana and, in 2003, Burkina Faso. At the same time Madagascar was infected and ASF became endemic in the island. Finally, in 2007, Georgia and subsequently its neighbours Armenia, Russia and Azerbaijan were infected, as well as the island of Mauritius. Molecular genetic studies indicated that all of the West African outbreaks were caused by genotype I viruses, while the Caucasus and Mauritius were infected by a genotype II virus previously only reported from Mozambique and Madagascar. Molecular studies have demonstrated that a large number of genotypes occur in eastern and southern Africa, while only genotype I has been identified in western Africa.
Key to distribution map for ASF

- **Yellow**: ASF is endemic in domestic pigs and warthogs
- **Red**: ASF is endemic in domestic pigs (and/or Eurasian wild boar)
- **Orange**: ASF occurs sporadically in domestic pigs due to movement of pigs/pork
- **Green**: ASF occurs sporadically in domestic pigs due to warthog contact
- **Dark Green**: ASF has occurred historically but has been eradicated

**PATHOGENESIS**

Infection of domestic pigs most frequently occurs via the oro-nasal route and virus is first detected in the pharyngeal tonsils and mandibular lymph nodes. Virus is also detected early in these organs after parenteral infection by blood-sucking *Ornithodoros* ticks or intramuscular injection. Occasionally virus has first been detected in bronchial or gastric lymph nodes. Spread of the virus is haematogenous, with 90% of the virus associated with erythrocytes; virus is also associated with peripheral leukocytes. The level of viraemia in acute cases of ASF is very high, often reaching $10^8$ HAD50/ml.

The target cells for primary replication are reticular cells, monocytes and macrophages, with a predilection for the antigen-presenting cells of the monocyte-macrophage system. Lymphoid tissues (lymph nodes, spleen, tonsils) therefore contain the greatest amounts of ASF virus. The virus enters macrophages via
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Receptors on the cell membrane. Replication occurs in the cytoplasm in ‘viral factories’ and virions are associated with ribosomes. Replication in other cell types including endothelial cells, pericytes, glomerular mesangial cells, renal collecting duct epithelial cells, hepatocytes, neutrophils and megakaryocytes has been observed, usually in later stages of infection with less virulent viruses, and is believed to have little effect on the pathogenesis of the disease.

Acute ASF is characterized by haemorrhage in multiple organs as a result of consumption coagulopathy and increased vascular permeability due to inflammatory mediators released by infected macrophages.
Widespread destruction of lymphocytes and marked lymphopenia are features of ASF but replication does not occur in lymphocytes. The loss of lymphocytes is due to apoptosis (programmed cell death) that results from the release of cytokines by affected macrophages, in particular tumour necrosis factor α (TNF-α) and interleukin 1α (IL-1α). A protein product P21 of a highly conserved ASF virus gene is present throughout the infection cycle but it does not prevent massive destruction of lymphocytes. Although the loss of lymphocytes must result in immune suppression, which is probably to the advantage of the ASF virus, pigs usually die of acute ASF before secondary infections are observed. In the subacute and chronic cases of ASF reported, especially from the Iberian Peninsula, secondary infections undoubtedly contributed to the clinical signs and pathology.

Haemorrhage in multiple organs is the most striking feature of ASF and its cause has been extensively debated. Thrombocytopenia is sometimes but not always a feature of ASF, and it was speculated that this could be due to a direct effect of the virus on thrombocyte production or survival. However, replication in megakaryocytes has been found to be minimal in most studies. It is generally accepted that impaired haemostasis is due to increased vascular permeability as a result of the effects of active substances released by infected and destroyed macrophages. The most important vascular changes are thought to result from the effect of inflammatory mediators secreted by the infected macrophages, including prostaglandin E2, that activate the clotting cascade and finally cause disseminated intravascular coagulation. Death is due to shock and/or excessive fluid exudation in the lungs. The latter is ascribed to activation of pulmonary intravascular macrophages with consequent release of cytokines, including TNF-α and IL-1α, and oxygen radicals. Thrombocytopenia if present is likely due to consumption coagulopathy.

### DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

#### Clinical signs and pathology

The incubation period for ASF is 5 – 15 days. The first indication of ASF is unusually high mortality among domestic pigs of all ages. Clinical signs are high fever, which is characterised by pigs huddling together as if cold, loss of appetite, lethargy and, in white-skinned pigs, marked skin flushing, particularly of the extremities and ventral body. As the disease progresses, haemorrhages become visible on the skin and mucosa.
In the acute stage of ASF white-skinned pigs become flushed to cyanotic, particularly the ears, lower legs, and ventral abdomen. Recumbency, accompanied by a high fever, indicated by congestion, haemorrhages and cyanosis of the ventral area and extremities.

Other, more variable signs include ocular and nasal discharges, vomiting, constipation followed by diarrhoea with blood-stained faeces, abdominal pain, hind limb weakness resulting in incoordination and a swaying gait, respiratory distress, and, in disease of longer duration, nervous signs. Sows may abort at any stage of pregnancy due to the high fever. At necropsy haemorrhages are observed in organs and on serosal surfaces. The body cavities generally contain blood-tinged fluid. The spleen is usually enlarged and congested, and occasionally infarcts may be present. The lymph nodes, in particular submandibular, hepatogastric and mesenteric nodes, are markedly enlarged and haemorrhagic, often resembling blood clots. Sometimes there is severe lung oedema. Severe congestion of the gastric fundus, sometimes with ulceration, and haemorrhagic enteritis may be present. Histologically the most characteristic changes are widespread karyorrhexis of lymphoid tissues, effacement of the SS sheaths in the spleen, and fibrinoid vasculitis. The virus targets macrophages, especially in lymphoid tissues but also in other organs and in bone marrow, and most of the changes observed can be ascribed to the release of cytokines by macrophages in which virus is replicating. These cause inflammation with increased vascular permeability, resulting in oedema and haemorrhage. Although the virus does not replicate in lymphocytes, widespread apoptosis of both B and T cells occurs. Death is usually due to shock and disseminated intravascular coagulation, but some pigs die as a result of massive lung oedema.
Fibrinoid necrosis of arterioles

Pyknosis and karyorrhexis in lymphoid tissues

Fibrous pericarditis seen in cases of subacute and chronic ASF

The lungs do not collapse and are enlarged due to the accumulation of fluid, resulting in prominent interlobular septa and straw-coloured to blood-tinged fluid in body cavities.
Pinpoint haemorrhages in the renal cortex

The mucosa of the stomach is often deeply congested to haemorrhagic and sometimes necrotic

Video link: http://www.youtube.com/watch?v=mF2oeROP0CU

As indicated earlier, the subacute and chronic forms of ASF appear to be rare. Subacute ASF is characterised by a fluctuating fever, and death, which occurs within weeks, is often due to pneumonia, generally caused by secondary bacterial invaders. Pigs that suffer chronic ASF are generally in poor condition, with long dull coats, ulcerous skin lesions including over bony points, and often have arthritis, pneumonia, and cardiac damage that can lead to congestive heart failure, with lesions such as fibrous epi- and pericarditis evident at necropsy. Ulcers at the ileo-caecal junction, as described for classical swine fever, have been described in what apparently were chronic cases of ASF in Angola.
Chronically infected pigs are usually severely emaciated and stunted, with a long dull hair coat. Signs of pneumonia may be present as well as lameness and ulcers over bony points.

A field diagnosis is based on high mortality in pigs of all ages and the typical clinical signs and lesions. Although described as typical, none are pathognomonic, and laboratory confirmation is essential.

**Laboratory confirmation**

The samples of choice are spleen and lymph nodes on ice but not frozen. If maintenance of the cold chain is a problem the samples may be preserved in 50% glycerol-saline. Additionally, a set of samples from various organs (spleen, lymph nodes, lung, liver, kidney, brain) may be taken in 10% buffered formalin for histopathological examination and immunohistochemistry. If only live sick pigs are available, whole blood in anticoagulant (EDTA) may be submitted for PCR and blood in heparin-containing tubes for viral isolation. It should be noted that serum is not a useful sample for diagnosis of acute or peracute ASF, as most pigs die before antibodies can be detected. Commonly used laboratory tests to detect the presence of virus are polymerase chain reaction (PCR) and the direct fluorescent antibody test (FAT). For more detailed information on the diagnostic techniques, please refer to the OIE Manual. Although the OIE Manual points out that the FAT is not as sensitive as PCR, in countries where FAT is used it will detect ASF virus in an outbreak, when large amounts are present in blood or tissue samples, and is a more robust test than PCR when laboratory conditions are not ideal. The gold standard for diagnosis remains viral isolation and the observation of haemadsorption or cytopathic effects.

**Differential diagnosis**

The most important differential diagnosis for ASF is classical swine fever (CSF). The two diseases are indistinguishable on clinical signs and pathological lesions, and laboratory confirmation is essential. Bacterial septicaemia is probably the second most important differential diagnosis, in particular erysipelas, the acute form of which is characterised by high fever and a severely congestive picture. Usually younger age groups are affected, although acute erysipelas may occur in pigs of all ages, mortality is lower and there is a response to antimicrobial treatment. Other diseases that may be
confused with ASF are possibly PRRS, particularly the more virulent forms that result in high mortality like the ‘high fever disease’ recently reported in China and Viet Nam, which is associated with a highly pathogenic strain of PRRS; porcine dermatitis and nephropathy syndrome, part of the porcine circovirus 2 associated disease (PCVAD) complex; and porcine trypanosomosis caused by Trypanosoma simiae. High mortality over a range of ages may occur with acute toxicoses, but these generally occur en masse rather than over a period of time as more pigs become infected with ASF. Poisoning with coumarins results in a haemorrhagic picture but usually only affects individual pigs. Stachybotryotoxicosis from mouldy feed results in widespread karyorrhexis that can be confused with the microscopic lesions of ASF.

CONTROL / PREVENTION

Since there is no vaccine, prevention of ASF relies upon implementing strict biosecurity measures that will prevent contact between the ASF virus and domestic pigs. On account of both the high mortality caused by ASF and the drastic measures required to control outbreaks, prevention is extremely important. Fortunately, since ASF is not an air- or water-borne disease and the infective dose is fairly high, the biosecurity measures required are relatively simple. Where the sylvatic cycle is the main source of infection, prevention relies on keeping pigs in pig-proof premises to avoid contact with warthogs and their tampans. In the infected zone in South Africa this is achieved by double fencing to ensure that even if warthogs approach the outer fence any tampans that might drop off them would not cross the space between the two fences. A solid wall will also offer adequate protection. The most important factor is that a pig-proof fence implies that it extends below the soil surface sufficiently to prevent pigs (wild or domestic) from digging their way through under the fence. The existence of accredited pig farms that have never experienced ASF in the control zone in north-eastern South Africa where the warthogs are infected bears witness to the success of these measures. It is underlined by the fact that when pigs in the control zone are allowed to roam freely, or are kept in facilities that are not adequately pig-proofed, ASF is almost inevitable.

Road blocks are important to prevent movement of pigs and pork products but its efficacy may be very limited in the circumstances that prevail in large parts of Africa.
In countries with or without the sylvatic cycle, where outbreaks are more frequently caused by movement of infected pigs and their products and scavenging or swill feeding, additional measures are required. Although controlling the movement of pigs and their products in areas where ASF is endemic is strongly recommended, in practice this is extremely difficult. Heavy traffic on roads and the potential for off-road movement prevent adequate control even if resources to implement it are available. It is therefore important to emphasise that the safety of pigs depends on the will and ability of their producers to protect them with on-farm biosecurity measures. These consist of limiting access to the pigs, providing at least a change of footwear to people who have to enter the area where the pigs are kept, and not feeding swill that could contain uncooked or under-cooked pork unless it has been thoroughly cooked first. While disinfectant footbaths have a place in on-farm biosecurity, because in multi-house facilities the provision of new footwear for each house would be impractical, it must be recognised that these alone are not enough. If disinfection is not accompanied by the removal of organic material from the soles of footwear, it will fail to inactivate ASF virus within the material. Footbaths should therefore be provided with a rough mat, preferably a metal scraper, as well as fresh disinfectant at the right strength daily. Disinfectants that are effective against ASF virus include 2% caustic soda (NaOH), 2% sodium hypochlorite, and various registered commercial virucidal products. Detergents are also effective because they destroy the lipid envelope of the virus. Clearly, in order to apply biosecurity measures, producers have to confine their pigs. This is generally not attractive to producers accustomed to low-input traditional pig production systems where the pigs largely fend for themselves. Their willingness to change will depend largely on market incentives, social pressure, and the availability of support and information to enable them to construct affordable but adequate housing, feed their pigs economically, and maintain an acceptable level of hygiene to prevent the diseases that come with confining pigs.

In the acute form of the disease death occurs in 2-9 days in 90% of cases.

Once a diagnosis of ASF has been confirmed, stamping out of all affected and in-contact pigs should commence. Deep burial and/or burning is recommended so that the meat is not consumed.
Following stamping out, all carcases are disposed of by deep burial and disinfection of the premises.

For commercial farmers with good facilities, the concept of compartmentalization (http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.08.01_ASF.pdf) opens the way for them to be able to access export markets even in areas where ASF is endemic. The OIE accepts and describes the principle of compartments that are free of one or more diseases but the guidelines for the measures to be applied should be developed by national authorities in collaboration with the industry.

In the event of an outbreak of ASF, as in the case of most highly contagious diseases, the recommended measures are quarantine, movement control and eradication by ‘stamping out’ of all infected and in-contact pigs, with destruction of the carcases. The exercise may be extended to include all of the pigs in a defined area, whether or not they are infected. The success of these measures depends on early diagnosis of the disease, adequate resources in terms of people and equipment to apply the measures, and enough funds for rapid payment of market-related compensation for healthy pigs slaughtered.

Delayed diagnosis usually results in spread of the disease with multiple foci to be controlled and many pigs to be killed, and even when the spread is limited, few developing countries have the resources to apply the measures effectively enough to prevent increased movement of pigs in order to avoid them. It is furthermore increasingly argued that ‘stamping out’ wreaks more damage than the disease itself, and is ethically, environmentally and aesthetically unacceptable. Without rapid and market-related compensation, it also serves as an imperative for clandestine movement and sale of pigs. Where export is not a consideration, most countries resort to at most ‘modified stamping out’, killing only sick animals and allowing those that survive to live. Since there is no long-term carrier state in domestic pigs, this does not necessarily prolong the course of the outbreak, provided the owners understand that they should not sell pigs from an infected herd and should not bring in new pigs for at least two months after the outbreak.

If this method is adopted, it is vital that the animal health officers visiting farms observe strict biosecurity practices to avoid carrying ASF from one farm to another. The cooperation of pig producers is key to the success of modified stamping out, and depends on their understanding the way in which the disease
spreads and how this can be prevented. If owners of infected herds agree, stamping out the entire herd may be a safer approach, but the wholesale slaughter of all the pigs in a defined area is not justified because ASF is not directly transmitted over distances. Where healthy pigs are slaughtered within infected herds, producers may agree to compensation in the form of nucleus breeding stock once the outbreak is over.

The OIE recommends that premises be kept free of pigs for 40 days before restocking, but in practical terms this depends on circumstances. It can be much shorter if the premises are isolated and there is no active ASF close by, whereas it may be longer if the disease is still prevalent in the area. Producers who wish to resume pig farming are advised to introduce sentinel pigs at approximately 10% of the stocking rate with access to all parts of the piggery. If they have shown no sign of disease after 4 – 6 weeks the premises can be considered free of ASF virus and more pigs can be introduced. Where tampans in pig sties are involved in maintenance of ASF, as occurred in the Iberian Peninsula, the situation is more problematic. Tampans can survive and remain infective for up to five years in a dormant state, and an outbreak occurred in Portugal in 1999, five years after eradication was complete, when pigs were introduced into sties that had been unoccupied for the entire period post eradication owing to the presence of tampans. Acaracides are generally not effective in freeing premises of tampans, and burning down the structures is recommended.
MARKETING AND TRADE / SOCIO-ECONOMICS

Like all transboundary diseases, ASF is a highly trade-sensitive disease and, for a country that exports pork, there are serious economic consequences of an outbreak in terms of trade bans. For this reason the incursion of ASF into an area as distant from Africa as the Caucasus raised alarm about the possibility of it reaching Europe. Although ASF has been present within the EU, in Sardinia, since 1978, this is not regarded as a particular threat because it has remained confined to that island since its introduction. However, the great majority of countries where ASF occurs are not important exporters of pork, and the socio-economic consequences are at the level of households rather than governments. An exception occurred in Georgia and Armenia, neither of which export pork, but whose most important trading partners chose to ban all agricultural products, including wine, lest they were contaminated with ASF!

Most sub-Saharan African countries do not have highly developed pig industries. The great majority of pigs are kept by smallholders in traditional husbandry systems. Under these systems, pigs are at most confined seasonally, and are for at least part of the year able to find their own food. Although these animals are not accorded a high commercial value, they are generally valued by their owners as a source of food as well as a source of household income, being sold opportunistically when there is a demand for them. In the forested areas of West Africa, which are largely unsuitable for cattle production, pigs replace cattle as the ceremonial animal of choice. Pig production in these areas also serves to reduce the quest for ‘bush meat’ and thus contributes to the conservation of biodiversity. The loss of large numbers of pigs to ASF therefore has effects that range from inability to pay school fees or make the appropriate...
contributions to weddings and funerals in Africa to increased illegal hunting of rare wild species. The seriousness of these consequences should not be underestimated, and ASF should be prevented. At the same time it must be remembered that for poor pig owners the value of being able to retain pigs that survive ASF and even to eat the meat from pigs that die of it will outweigh the fear of perpetuating the disease, and therefore drastic control measures, unless prompt and acceptable compensation is assured, will be unwelcome.

The costs of an ASF outbreak in a high value pig industry can be astronomical. Apart from the producers, major losers in the industry are abattoirs, meat retailers, and feed manufacturers. There are also major costs to the government for implementation of control measures and compensation for compulsory slaughter. In commercial industries producing millions of pigs the logistical challenges of stamping out and disposal of carcasses are already accepted to be insurmountable. The effects in smaller industries when calculated in terms of job losses and lost trade opportunities may be equally catastrophic, given that most of the people involved are not wealthy in the first place. For example, as a result of the outbreaks in Côte d’Ivoire in 1996, 300 women who made their living in the rural areas from slaughtering pigs were deprived of their only income.

When control measures are imposed, owners of healthy herds often suffer the most, as they are unable to move their pigs to the abattoir for slaughter and both feed costs and space become problematic. In the end more pigs may have to be killed on-farm and disposed of simply for welfare reasons. If at all possible, identifying healthy herds and allowing controlled movement to the abattoir for slaughter can assist control, because the availability of uninfected pork for legal sale may limit clandestine trade in pork of unknown origin that is likely to take place when pork becomes scarce.

ASF virus naturally infects only members of the pig family and humans cannot become infected

It has been noted that the economic effects of ASF may encourage transboundary spread of the disease. When pigs start dying, many owners are anxious to sell their pigs that are still apparently healthy before they die of disease, as well as the meat of pigs that have died. This often leads to a drop in the price of pigs and pork, making these commodities more attractive to neighbours. For example, pigs usually fetch
a higher price in Benin than in neighbouring Togo, but when ASF broke out in Benin in 1997 the price dropped to below the price of pigs in Togo, and the first outbreaks there were traced to pigs bought in Benin, probably in the incubation phase of ASF.

Market forces are likely to prove important in controlling and possibly eradicating ASF from countries where it is endemic in domestic pigs. To break the circulation in domestic pig populations, a change from free-range husbandry to more conventional management systems is essential. This requires inputs from the owner in terms of housing, feed and provision of water that will not be affordable unless a good price can be obtained for the pigs. Apart from ASF, there are other problems that can be resolved if the pigs are confined, notably porcine cysticercosis, which is currently the most important cause of epilepsy in human populations in sub-Saharan Africa, and there are initiatives to support a change in husbandry methods to eliminate this neglected zoonosis. However, efforts to train pig farmers are most likely to succeed if there are real economic benefits for them in improving their husbandry methods.

IMPORTANT OUTBREAKS

High mortality means that any outbreak of ASF will be of some importance, if only to the owners of the affected pigs. However, the ASF events listed below are considered to be of particular importance because of the wide-ranging effect that they have had.

- The wave of ASF that swept through Europe after the introduction of ASF into the Iberian Peninsula in 1959/60, reaching the Caribbean and Brazil, and resulting in ASF becoming endemic in the Iberian Peninsula for more than 30 years and apparently permanently in the island of Sardinia.
- The West African pandemic that started in Côte d’Ivoire in 1996 and resulted in ASF becoming endemic in several countries that were not previously infected.
- The outbreaks in the Caucasus that started in Georgia in 2007 and swept through the region, where it appears to have become established in the southern dependencies of Russia bordering the Caucasus with spread to more than 130 foci, some in north-western Russia not far from the borders of Estonia and Finland, creating fears of another introduction into Western Europe.

An increase in ASF activity and movement has been observed in eastern Africa since 1993. Outbreaks occurred in Mozambique south of the Save river for the first time and in Kenya for the first time in 30 years. In 1997/8 ASF was introduced into Madagascar for the first time, where it has become endemic, and from which it spread to Mauritius in 2007, requiring considerable efforts to eradicate it. Tanzania also suffered outbreaks in various parts of the country, and outbreaks occurred in provinces in Zambia not previously affected. Although the sylvatic cycle is present in these countries, all the outbreaks were traceable to movement of pigs and pork and not to contact with the wild hosts and vectors. This is
important because it indicates a change in the epidemiology of the disease that is probably linked to the way pigs are kept and marketed, and has most likely been the source of the outbreaks in the Caucasus.

FAQs

1. Can humans get ASF?

No, ASF is not a zoonosis.

2. Will eating meat from pigs that have died of ASF be harmful to one’s health?

The fact that it is infected with ASF virus will not harm one’s health, but if the pig was clinically sick it would have had a high fever and the carcass would have been haemorrhagic, which reduces the keeping quality and flavour of the meat, partly owing to an increased bacterial load owing to increased permeability of the gut. Some of the bacteria or their toxins might cause disease in humans.

3. Which other animals can get ASF?

ASF virus naturally infects only members of the pig family. In the attempts to produce vaccines and for other purposes experimental infections have been produced in rabbits and even in a goat, but this would not happen under field conditions.

4. Do pigs that recover from ASF become lifelong carriers of the virus?

Pigs that recover from ASF and are healthy (i.e. not suffering from a chronic form of the disease and obviously unwell) do not become lifelong carriers of the virus. They may shed virus sporadically for up to 30 days post infection, but after that are not able to infect in-contact naïve pigs. Sensitive tests (PCR) may detect virus in the blood for longer, although this may no longer be viable. Virus may be detectable in tissues for up to six months, but again may not be viable. However, it would be prudent not to slaughter pigs until at least six months after recovery in case the meat ended up in uncooked swill and some infective virus remained.

5. Why do some pigs survive without showing ill effects when most of the pigs die of ASF?

The mechanism for survival of these pigs is not known, but some pigs appear to have an inherent immunity to the pathogenic effects of ASF, similar to that of African wild pigs. Studies have shown that this ability to survive is not simply inherited by offspring.

6. Why is there no vaccine for ASF?
Efforts to produce a conventional vaccine against ASF failed for various reasons. Firstly, it was difficult to produce a vaccine that would protect pigs without causing clinical ASF. Secondly, the protection afforded, if any, did not extend to infection with heterologous viruses. Now that there is a good understanding of the molecular biology of the virus and the pathogenesis of the disease, there is more hope that a vaccine will be developed.

7. **Is there any treatment for ASF?**

There is no treatment for ASF.

8. **Which disinfectants work against ASF?**

The most practical disinfectants to use are 2% sodium hypochlorite (bleach), 2% sodium hydroxide (caustic soda), detergents, and some of the commercial virucidal products.

9. **Are aerosols important in the transmission of ASF?**

Aerosol transmission has not been demonstrated, and airborne transmission has been shown to occur only over short distances (<2m) in closed barns where the pigs are not separated by physical barriers.

10. **Can ASF virus come from pig feed, as opposed to swill containing uncooked pork?**

ASF virus can only come from pig feed if the feed has been contaminated by the blood, saliva or excretions of pigs suffering from ASF and there is sufficient moisture for the virus to remain viable.

REFERENCES


