African Swine fever (ASF)

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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. Omithodoros 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.
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* tampans 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* tampans 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 $10^5$ HAD$_{50}$ 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.
ASF is endemic in domestic pigs and warthogs

ASF is endemic in domestic pigs (and/or Eurasian wild boar)

ASF occurs sporadically in domestic pigs due to movement of pigs/pork

ASF occurs sporadically in domestic pigs due to warthog contact

ASF has occurred historically but has been eradicated