

African swine fever: an epidemiological overview

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Summary

African swine fever is a highly infectious, viral disease affecting domestic pigs with mortalities approaching 100%. The disease severely impacts on the socio-economic status of farmers. The disease is endemic in a number of countries in sub-Saharan Africa, in Sardinia (Italy), the Russian Federations, the Caucasus and it has shown possibilities of spread within Eastern Europe. Involvement of all stakeholders in the pig value chain in designing and implementing of prevention and control strategies could be explored in future control programs. This may call for investing resources in changing disease management behavior in managing this disease.

Introduction

African swine fever (ASF) was first described by Montgomery in 1921 in Kenya where it caused a disease with nearly 100% mortality in domestic pigs. It is considered endemic in most of sub-Saharan Africa (SSA) and in Sardinia, Italy. The disease has spread to the Caucasus and the Russian federation (Figure 1) with recent developments in eastern Europe suggesting that ASF could have a geographical expansion (FAO, 2012).

ASF is caused by African swine fever virus (ASFV), a large icosahedral DNA virus of the genus *Asfivirus* and family *Asfarviridae* (Dixon et al., 2005). The virus genome is about 170-190 kilobase pairs, depending on the isolate (Blasco et al., 1989), encoding about 160-175 genes. Basing on the p72 portion of the genome, at least 22 genotypes have been identified basing on a partial nucleotide sequencing of the p72 gene (Boshoff et al., 2007) and all of which are present in Africa. Subtyping is based on full-length sequencing of the p54 gene. Further discrimination among isolates can be done by analysis of the central variable region (CVR) with the B602L gene (Gallardo et al., 2009).

An ASF disease is manifested as subacute, acute, chronic and subclinical forms depending on the virulence of the viral strain. These have varying lesions characterized by extensive haemorrhages and lymphoid tissue destruction (Mebus, 1988). It causes a highly contagious disease with severe socio-economic impact affecting smallholder and commercial pig farmers and causing losses to governments in terms of loss in trade, control measures in case of outbreaks and compensation.

The pig industry has been growing and especially in many countries in SSA, pig numbers have increased by more than 50% in a decade. Since most of this increase had been in smallholder or backyard systems with low levels of biosecurity, the sector growth comes along with disease control and prevention challenges (FAO, 2012). This, together with increasing movement of people and pig products is largely responsible for the ASF incursion into new areas.

Transmission

The transmission and maintenance of ASF occurs in a sylvatic cycle and/or a domestic pig cycle. Depending on the presence or absence of wild suids and arthropod vectors and the type of pig production system, the epidemiology of ASF varies substantially between countries, regions and continents (Costard et al., 2009). The cycles are;

(a) Sylvatic cycle

The role of wild pigs is well described for warthogs in eastern and southern Africa (Thomson, 1985; Plowright et al., 1994), although information is lacking from other African regions and for other wild pig species (Jori et al., 2007). Maintenance of the virus is dependent on soft ticks of the species *Ornithodoros* species. Young warthogs become infected when bitten by infected *Ornithodoros* spp ticks while in the burrows and develop a transient viraemia lasting two to three weeks. This is sufficient to infect viraemic ticks feeding on infected individuals (Thomson et al., 1980). The role of bush pigs (*Potamochoerus larvatus*) remains largely unexplained although their role as free-living hosts of ASFV has been demonstrated both experimentally and under natural condition (Oura et al., 1998; Luther et al., 2007). Although bushpigs develop sufficient viraemia to infect *Ornithodoros* spp and domestic pigs, they seem to require high levels of virus of virus to become infected than domestic pigs (Anderson et al., 1998). In Africa, the giant forest hog (*Hylochoerus meinertzhageni*) has also been reported although its role is still considered negligible (Penrith et al., 2004). The wild boar (*Sus scrofa*) and feral pigs are susceptible to

ASFV and show clinical signs and mortality similar to domestic pigs. In Russia and the Caucasus region, it has been suggested that ASF has been spreading along trade routes with occasional infection of wild boar (FAO, 2010).

(b) Domestic pigs

Most isolates of ASF cause an acute haemorrhagic fever with mortality approaching 100%. Moderately virulent and low virulent isolates have also been described and recovered pigs can remain persistently infected for periods of 6 months or more (Wilkinson, 1984; Oura et al., 2005). Recovered pigs may transmit the virus to uninfected pigs directly or through infected pork. Poorly disposed of carcasses, fomites (equipment, vehicles and clothing) can be a source of infection.

(c) Transmission between sylvatic cycle and domestic pigs

The *Ornithodoros* spp. soft vectors and wild suids are a potential risk to domestic pigs. Adult warthogs may transport the *Ornithodoros* to areas where there domestic pigs are reared thus exposing them to ASFV. Domestic pigs that come in contact with warthog carcasses or feed on their faeces may be exposed to ASFV (Thomson et al., 1980), a route which may also be possible with bushpigs and giant forest hogs which do not come into contact with *Ornithodoros* spp. (Roger et al., 2001). Although the sylvatic involving warthogs occurs throughout southern and eastern Africa, the majority of ASF outbreaks in recent decades have been associated with movement of infected pig and pork (Penrith and Vosloo, 2009).

Molecular epidemiology of ASFV

Advances made in molecular typing methods have contributed considerably to improved understanding of the epidemiology of ASF. Differentiation between ASFV isolates depends on genetic methodologies. Earlier comparative studies utilized restriction fragment length polymorphisms (RFLPs) (Wesley and Tuthill, 1984) but these methods have now been replaced by PCR amplification and nucleotide sequencing. RFLP analyses revealed that outbreaks in domestic pigs in Europe, the Caribbean, and Cameroon in west Africa between 1957 and 1986 were closely related, indicating that the disease had spread over several continents, probably

because of a single spread from a wildlife source in Africa into domestic animals (Wesley and Tuthill, 1984; Vinuela, 1985).

Phylogenetic analysis using different gene regions has made it possible to compare many more isolates. The first such comparison, including a large number of viruses from many geographical origins, demonstrated that analyses of the B646L gene (encoding one of the major structural proteins, VP72), could successfully distinguish 10 major ASFV genotypes on the African continent—of which five corresponded to the geographical groupings distinguished by RFLP analysis (Bastos et al., 2003). The largest group comprised isolates from 24 countries in Europe, South America, the Caribbean and West Africa, the so-called ESAC-WA genotype or genotype I with a highly conserved B646L gene (Bastos et al., 2003). Nine other genotypes occurred in East and southern Africa where the sylvatic cycle occurs and provided evidence of spill-over from the sylvatic cycle to domestic animals. More detailed studies using the B646L gene region identified 13 genotypes in eight countries in East Africa. Significantly, genotype I, thought to be present only in the domestic pig cycle, was found in a sylvatic cycle in East Africa (Lubisi et al., 2005). In addition, a homogeneous pig associated lineage-linked outbreaks that had occurred in Mozambique, Zambia and Malawi over a 23-year period. In southern Africa, a further six novel genotypes were identified based on sequencing of the B646L gene, bringing the total number to 22 (Boshoff et al., 2007). As in East Africa (Lubisi et al., 2005), some genotypes in southern Africa were country specific, while others had transboundary distributions (Boshoff et al., 2007). These data have clearly demonstrated that greater genetic variation occurs where the sylvatic cycle is present and that occasional transmission occurs between the sylvatic and domestic cycle in addition to long-term circulation of conserved viruses within domestic pigs. Analysis of other gene regions was carried out to assist with outbreak tracing. Analyses of the central variable region (CVR) within the B602L open reading frame identified 12 differently sized products within the ESAC-WA genotype (Phologane et al., 2005). Sequencing from a larger set of isolates from this genotype (Nix et al., 2006) revealed 19 subgroups. The large conserved B646L genotype VIII, which defines virus causing outbreaks between 1961 and 2001 in four East African countries, was further characterized into seven discrete amino-acid lineages while a combined B646L–CVR analysis identified eight lineages (Lubisi et al., 2007).

According to Costard et al. (2009), the current approach for molecular discrimination is therefore to use the B646L gene for genotyping, and either sequencing the CVR of closely related isolates, or combined PCR of several other gene regions to distinguish sub-groups. This approach was used recently to reveal that the ASFV isolates introduced into the Caucasus and Mauritius were both genotype II (Rowlands et al., 2008). Genotype II has been found circulating in domestic pigs in Mozambique, Zambia and Madagascar (Bastos et al., 2003; Bastos et al., 2004; Penrith et al., 2007) and it is suggested that this virus may have been introduced to Georgia from infected meat taken from ships in the Black Sea port of Poti and being fed to domestic pigs (FAO, 2008).

Risk factors, control and prevention

In endemic areas in Africa, local spread is associated with free-range pig production system, local pig movements and a lack of biosecurity (Costard et al., 2009). It is not uncommon to encounter pig movements during (ASF) outbreaks in areas where pig movement controls (quarantine) are in place (Figure 2). Within the pig value chain, the management practices and infrastructure on pig farms and the practices in the trade and processing of pork products present potential risk (Figure 3) for ASF spread. In the Caucasus and the Russian federation, ASF spreads progressively along trade routes in areas where smallholder pig production is common, with occasional infection of the wild boar (FAO, 2010).

While ASF was eradicated in the majority of non-African countries that had outbreaks of ASF, it has not been the case in SSA (Penrith et al., 2013). Stamping out through culling, compensation and strict zoo-sanitary measures have been largely used. In Africa, in addition to stamping out, quarantine and movement control must be rigorously applied with ASF outbreaks. However, the measures which include movement bans and bans on sale of pork have largely been ineffective because it has been impossible to enforce them (Penrith et al., 2013).

As a long-term measure, governments in the developing world may need to increase investment in livestock particularly in research as suggested by Perry and Grace (2009) since the role of livestock in poverty-reduction is recognised. There may be need for investing development resources in changing disease management behavior (Perry and Grace, 2009) in managing diseases like ASF in SSA.

Conclusions

ASF remains endemic in most countries in SSA and in Sardinia Italy. The disease is also currently endemic in the Russian Federation since 2008 and has shown tendencies of geographical expansion in Eastern Europe. In most of these cases, there has been involvement of smallholder farms with free-ranging pigs, illegal movement and sale of pigs and pig products and management practices (behavior) that could spread disease. Although the sylvatic cycle exists in Africa, most outbreaks have been associated with movement of pig and pork products. Although some countries have succeeded in eradicating ASF, there is need for increased awareness of all stakeholders in the pig industry in the areas that are affected. The success of prevention and control strategies will largely depend on cooperation from all actors in the pig value chain. Governments need to increase investment in livestock especially in research and also efforts made towards changing disease management behavior for diseases like ASF.

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Figure captions:

Figure 1: Distribution of ASF (Source: OIE WAHID 2013)

Figure 2: Movement of sick and in-contact pigs and their products is very risky.

Figure 3: Poor to no slaughter infrastructure makes ASF control difficult.





