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INFECTIOUS DISEASE: REVIEW ARTICLE

An Update on the Epidemiology and Pathology of African Swine Fever

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Summary

African swine fever (ASF) is one of the most important infectious diseases of swine and has major negative consequences for affected countries. ASF is present in many sub-Saharan countries, Sardinia and several countries of eastern and central Europe, where its continuous spread has the swine industry on heightened alert. ASF is a complex disease for which no vaccine or treatment is available, so its control is based on early detection and rapid control of spread. For a robust and reliable early detection programme it is essential to be able to recognize the clinical signs and pathological changes of ASF, keeping in mind that in most cases the first introductions don't show high mortality nor characteristic clinical signs or lesions, but fever and some hemorrhagic lymph nodes. Knowledge of the main characteristics of this infection, including its current distribution and routes of transmission, is also essential for preventing and controlling ASF. This review addresses each of these topics and aims to update knowledge of the disease in order to improve early detection of ASF in the field and allow implementation of public health programmes.

Keywords: African swine fever; clinical signs; differential diagnosis; lesions

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Introduction

African swine fever (ASF) is one of the most important infectious diseases of swine and is present in many African countries, some eastern and central European countries and Sardinia. ASF must be notified to the World Organization of Animal Health (OIE) and its presence leads to immediate restrictions on the pig and pork trade.

ASF is caused by infection with a complex DNA virus, the ASF virus (ASFV), which is the only member of the family Asfarviridae (Dixon *et al.*, 2005). ASFV is a large enveloped virus of approximately 200 nm in diameter that contains double-stranded DNA of 170–193 kilobase pairs (Dixon *et al.*, 2013). ASFV is composed of more than 50 structural proteins and produces more than 150 proteins in infected macrophages (Salas and Andrés, 2013), many of which are highly immunogenic. Hence, infection causes a strong humoral immune response that persists for long periods of time. Nevertheless, the antibodies produced are not able to neutralize ASF infection effectively (Neilan *et al.*, 2004) and serotyping is not possible. Consequently, classification is based on genotyping procedures through the analysis of some genome regions, such as the C-terminal region of the gene encoding vp72 (Bastos *et al.*, 2003). Based on the differences observed in this region, circulating isolates of ASFV have been classified into 22 genotypes (Boshoff *et al.*, 2007).

ASFV replicates in mononuclear phagocytic cells of both domestic and wild swine. The virus infects monocytes and macrophages (Malmquist and Hay, 1960), but infection of T or B lymphocytes has never been observed (Minguez *et al.*, 1988). The virus also replicates in endothelial cells (Carrasco *et al.*, 1996a), hepatocytes, renal tubular epithelial cells (Gómez-Villamandos et al., 1995a) and neutrophils. After initial replication in these primary sites, ASFV spreads through the blood or the lymphatic system, where it persists for long periods of time in the absence of neutralizing antibodies and moves towards secondary sites of replication.

ASFV also replicates in soft ticks of the genus *Ornithodoros*, which act as virus reservoirs. These ticks are involved in the epidemiological cycle of ASF in eastern and southern Africa (*Ornithodoros moubata*) and have also been observed during infection on the Iberian Peninsula (*Ornithodoros erraticus*). Other *Ornithodoros* spp. have been demonstrated to be susceptible to ASF infection (European Food Safety Authority, 2010). ASFV strain Georgia 2007/1 can also replicate in *O. erraticus* ticks (Diaz *et al.*, 2012), but the potential role of these ticks in virus transmission is still unknown in other European regions.

Despite efforts made over past decades, there is no vaccine available for preventing and controlling ASFV infection. Several strategies have been studied; however, the lack of neutralizing antibodies, genetic variability and presence of some gaps in knowledge about ASF pathogenesis and immune modulation make discovery-based approaches difficult.

African Swine Fever Epidemiology and Main Routes of Introduction

ASF was first described in Kenya in 1921 (Montgomery, 1921) and since then it has spread rapidly to other African countries. ASF first escaped from Africa in 1957 to reach Portugal via contaminated waste containing infected pig products that were used to feed pigs. After this incursion, which was rapidly controlled, ASFV re-entered Portugal in 1960 and this time it spread to the whole Iberian Peninsula, where it persisted for more than 30 years (Arias and Sánchez-Vizcaíno, 2002). During this period (1960–1995), ASFV spread sporadically to other countries in Europe and America (i.e. Brazil, Dominican Republic, Cuba and Haiti). ASF has been eradicated from all of these countries except from the Italian island of Sardinia, which has been affected since 1978 (Sánchez-Vizcaíno and Arias, 2012; Costard *et al.*, 2013a, b).

ASF has also continued to spread on the African continent and reached West African countries and some islands that were previously free of the disease. Some authors have stated that this spread started in 1994 due to a combination of factors including: (1) increasing pig production on the continent, (2) presence of non-symptomatic pigs that could act as reservoirs to spread the disease without being noticed and (3) globalization. These factors, together with the economic crisis of the present century, could be the origin of the spread of ASFV to Eastern Europe (Penrith and Vosloo, 2009; Sánchez-Vizcaíno *et al.*, 2013).

In 2007, ASFV entered Georgia through the port of Poti (Beltran-Alcrudo *et al.*, 2008), potentially via contaminated food used to feed pigs. From this region, ASFV spread rapidly through the country and affected neighbouring countries including Armenia, Azerbaijan and the Russian Federation. In this geographical region, ASFV affects domestic and wild boar and has spread to the north and west. In 2012, the first outbreaks were declared in Ukraine, followed by Belarus in 2013 (Sánchez-Vizcaíno *et al.*, 2013). From these countries, ASF continued to spread before reaching the European Union (EU) borders in 2014, when several dead wild boars were found in Lithuania and Poland. Since then, several cases have been reported in Estonia and