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African Swine Fever: Analysing its Epidemiology, Pathogenesis and Control Strategies: A Review

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ABSTRACT

African Swine Fever Virus (ASFV) poses a significant threat to global swine populations, with devastating economic and agricultural implications. This review article provides a comprehensive examination of various facets of ASFV, encompassing its structure, entry mechanism, transmission dynamics, clinical signs, pathogenesis, diagnosis and control strategies. The complex virion architecture, including the multilayered core and distinctive outer envelope, is explored, shedding light on key elements influencing the virus's stability and infectivity. The intricate mechanisms governing ASFV entry into host cells are discussed, emphasizing the interplay between viral proteins and cellular receptors. Insight into the virus-host interaction provides a foundation for understanding the initial stages of infection, influencing subsequent pathogenesis. Transmission dynamics, a critical aspect of ASFV epidemiology, are examined, encompassing both direct and indirect modes of spread. Factors influencing the persistence of ASFV in diverse environments and the role of vectors in disease dissemination are explored to elucidate the complex transmission pathways. Clinical signs and pathogenesis of ASFV infection are thoroughly reviewed, outlining the diverse manifestations in swine species. The immunopathological responses and host factors influencing disease severity are discussed, enhancing our understanding of ASFV pathobiology. A comprehensive understanding of diagnostic tools is pivotal for timely and accurate disease detection, enabling swift intervention measures. In conclusion, this review provides a nuanced and integrative overview of ASFV, offering valuable insights for researchers, veterinarians and policymakers engaged in combatting this significant threat to the swine industry.

Key words: African Swine Fever Virus (ASFV), Control Strategies, Epidemiology, Pathogenesis, Virion architecture.

The African swine fever virus (ASFV) causes African swine fever (ASF), a viral disease affecting swine. It leads to high mortality in domestic pigs while remaining asymptomatic in the natural suid reservoir hosts. The disease results in significant economic losses that cannot be avoided without an effective vaccine. The primary methods of disease control include quarantining and culling infected animals (Penrith *et al*., 2009). ASFV is a double-stranded DNA virus known for its complex molecular structure. It belongs to the Asfarviridae family, Asfivirus genus and Asfuvirales order. It stands out as the only DNA virus transmitted by arthropods, specifically soft ticks of the Ornithodoros genus (Parker *et al*.,1969). Soft ticks, such as Ornithodoros moubata in Africa and Ornithodoros erraticus in Europe, play essential roles in the sylvatic transmission cycle of the virus.

African swine fever disease was first identified in the 1920s in Kenya (Montgomery *et al*., 1921). Initially, it was confined to Africa, but during the mid-20th century, it expanded to Europe, South America and the Caribbean. Aggressive control measures led to the eradication of the disease in Europe, except for Sardinia, during the 1990s. However, in 2007, ASF extended beyond Africa into the Caucasus, particularly in Georgia and by 2014, it had reached the eastern region of the European Union. Subsequently, the disease was reported in various EU nations, including Poland and the Baltic states (Pejsak *et al*., 2014; Wozniakowski *et al*., 2016).

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In eastern and southern Africa, the genetic characterization of the ASFV, based on sequence variations in the C-terminal region of the B646L gene encoding the main capsid protein p72, revealed the presence of 22 distinct genotypes (Bastos *et al*., 2003; Boshoff *et al*., 2007). Western Africa played a role in exporting ASFV genotype I to Europe in 1960 (Boklund *et al*., 2018). In 2007, genotype II of ASFV was imported from eastern Africa and rapidly spread throughout Europe, eventually entering China *via* Russia in 2018 (Dixon *et al*., 2019). On August 3, 2018, the World Organization for Animal Health reported the first case of

ASFV strain belonging to genotype II in Shenyang, Liaoning Province, China (Zhou *et al*., 2018; Pikalo *et al*., 2019).

In India, there were a total of 11 ASF outbreaks reported on May 21, 2020, in Assam and Arunachal Pradesh states, resulting in the death of 3,701 pigs (WOAH, 2020). Between February 24, 2020 and April 10, 2020, seven ASF outbreaks were reported in the state. The disease initially appeared in the Arunachal Pradesh region and, within a month, had spread to the Dhemaji region of Assam. Subsequently, it was observed in the Sivasagar region on March 7, 2020, Biswanath on March 20, 2020, another region of Sivasagar on March 23, 2020 and simultaneously in different regions of Kamrup Metro and Sivasagar districts on April 2, 2020 (Patil *et al*., 2020).

The ASFV can persist in the natural environment for extended periods due to the complex structure of its particles and genome. Domestic pigs are highly susceptible to ASFV infection, which can occur through contact with infected pigs, exposure to people and objects carrying the virus and contact with virus-contaminated environments (Gaudreault *et al*., 2020).

Effective control of ASF necessitates stringent quarantine measures and rapid, precise laboratory diagnosis, which plays a crucial role in providing valuable epidemiological data, early disease detection and limiting the spread of ASFV. An ideal ASF diagnosis method should consistently demonstrate high sensitivity and specificity while being designed for ease of use and high-throughput applications. Polymerase chain reaction (PCR) is regarded as the standard tool for early ASF diagnosis due to its superior sensitivity, specificity and capacity to detect the ASFV genome in clinical samples from domestic pigs, wild swine and ticks (Gallardo *et al*., 2015).

The ASFV is characterized by a large genome and ongoing research focuses on identifying protective antigens and virulence genes. While previous studies have demonstrated that protective immunity can be achieved with attenuated vaccines, challenges such as virulence, immunogenicity, viral phenotype, antigen diversity and the absence of cross-protective immunity continue to impact live attenuated ASF vaccines. Currently, the primary measure to contain the disease is the implementation of movement restrictions, banning swill feed, stringent biosecurity practices and the use of rapid and accurate laboratory diagnosis (Yong-Joo *et al*., 2021).

The review aims to provide a comprehensive overview of African swine fever (ASF), a viral disease affecting swine caused by the African swine fever virus (ASFV). The review covers the history and spread of ASF from its identification in Kenya in the 1920s to its expansion into Europe, South America and Asia. It highlights the complexity of the ASFV, its unique transmission and the genetic diversity of ASFV genotypes in different regions. Furthermore, the review addresses specific outbreaks, such as those in India and emphasizes the need for effective control measures, including quarantine, culling and stringent biosecurity practices. The importance of rapid and precise laboratory diagnosis, particularly using polymerase chain reaction (PCR), is highlighted for early detection and limiting the spread of ASF.

Structural characteristics and genetic composition of the virus particle

African swine fever virus presents itself as a colossal icosahedral DNA virus, characterized by virus particles ranging in diameter from 260 to 300 nm. The virus exhibits a complex, multi-layered structure, comprising distinct components arranged from the outermost layer to the innermost core (Wang *et al*., 2021).

Outer envelope identified by proteins p12/pE402R:

The outermost layer is the outer envelope, which is said to be derived from the host cellular membrane during the budding process. Notably, traces of protein pE402R (CD2v) have been identified on the surface of budding virions. The protein p12 plays a pivotalt role in facilitating ASFV's entry into host cells by promoting the adsorption of virus particles to specific receptors on the host cell membrane (Angulo *et al*., 1993).

Capsid (pE120R/pB438L/P72):

At the core of ASFV's structure lies the capsid, composed of 2,760 pseudo-hexameric capsomers and 12 pentameric capsomers. These capsomers are constructed through the arrangement of p72 protein molecules, adopting a double jelly-roll structure in groups of three for the pseudo-hexameric capsomers and five for the pentameric capsomers (Wang *et al*., 2019). The presence of protein pB438L is crucial for forming the capsid's vertices. Additionally, pE120R is an integral component of the virus capsid (Alejo *et al*., 2018).

Inner Envelope (p12/p17/pE183L/pE248R/pH108R/pE199L/P22):

Inside the capsid, p17 and pE183L (p54) play essential roles in orchestrating the assembly of the capsid layer. Simultaneously, p12, pE248R and pE199L are actively engaged in facilitating ASFV's entry into host cells (Wang *et al*., 2021).

Core-Shell (p5/p14/p34/p37/p150/p15/p35/p8/pS273R):

ASFV's core-shell formation involves the enzymatic breakdown of two virus polyprotein precursors, pp220 and pp62, through the viral protease pS273R. This process results in the generation of various mature products that collectively constitute the core-shell (Revilla *et al*., 2018).

Nucleoid (p10/pA104R):

The innermost core of ASFV particles houses the nucleoid, where p10 and pA104R act as DNA-binding proteins, contributing to the management of the virus's genetic material (Urbano and Ferreira, 2020).

In summary, ASFV's virion structure is characterized by its immense size and intricate, multi-layered organization.

Each layer and associated proteins within the virus play distinct roles in various stages of the virus's life cycle, including entry, replication and genetic management (Alejo *et al*., 2018).

Proteins constituting the structure of ASFV

The ASFV virion contains many structural proteins, some of which play a role in forming the virus particles. Researchers have identified up to 70 of these proteins and at least 16 are believed to be involved in the assembly process (Alejo *et al*., 2018) (Table 1).

Among these, the primary capsid protein, p72 (ORF B646L), plays a crucial role in facilitating the assembly of the icosahedral capsid on the inner envelope (García *et al*., 1998). Additionally, mature proteins originating from polyprotein pp220 (CP2474L) and polyprotein pp62 (CP530R) combine to form the core-shell that encloses the DNA-containing nucleoid (Andrés *et al*., 2002).

Another essential component is the membrane protein p17 (D117L), which is necessary for the assembly of the capsid layer on the inner envelope (Suárez *et al*., 2010). Furthermore, the phosphoprotein p14.5 (E120R), acting as a capsid component, is involved in facilitating intracellular virus transport (Andrés *et al*., 2001). The polyprotein processing enzyme, encoded by ORF S273R and identified as a cysteine proteinase, plays a pivotal role in processing viral polyproteins. Vital DNA-binding proteins p10 (K78R) and pA104R, situated in the nucleoid of mature ASFV particles, are implicated in viral processes within this domain. Notably, pA104R, through knockdown experiments using small interfering RNAs, has been established to participate in vital functions such as viral transcription, DNA replication and genome packaging (Frouco *et al*., 2017).

Temporal dynamics of ASFV infection: from adsorption to viral release

The infection mechanism of the virus involves six key events namely adsorption, penetration, uncoating, biosynthesis, packaging and shedding. After binding to the host receptor, ASFV is internalized within 30 minutes through either direct adsorption or the macropinocytosis pathway. It enters early endosomes at 1-30 minutes post-infection and progresses to late endosomes between 30 to 90 minutes. Shelling and genome release occur in late endosomes, followed by three gene expression stages: early (4-6 hours post infection, intermediate (6-16 hours post infection) and late (8-24 hours post infection). Virus particles assemble in a designated "virus factory" around 16-24 hours post infection, with release from the cell membrane occurring approximately 24 hours post-infection (Gaudreault *et al*., 2020).

Viral entry mechanism

The ASFV infectious cycle commences with viral adsorption and entry into the host cell *via* receptor-mediated endocytosis. Rab GTPase proteins regulate endosomal maturation, with viral decapsidation occurring in mature endosomes. Following fusion with endosomal membranes, naked cores are released into the cytosol for replication. The cytoplasmic replication cycle involves microtubule-mediated transport to a perinuclear area, forming viral factories. Structural protein p54 interacts with dynein, facilitating transport. The perinuclear viral factory, near the microtubule organizing center, accumulates viral proteins and DNA, leading to the assembly of icosahedral intermediates and mature virions through DNA encapsidation (Covadonga *et al*., 2012).

Transmission

African swine fever is one of the pig diseases with the highest mortality. ASFV is generally spread by contact with infectious animals and fomites, ingestion of contaminated pig products and tick bites. However, ASFV transmission and maintenance varies substantially between countries. In sub-Saharan Africa, the disease is endemic and circulates through a cycle of infection involving domestic pigs, bushpigs, warthogs and soft ticks of the Ornithodoros species (Plowright *et al*., 1969). In areas of the Caucasus, Eastern Europe and the Baltic countries, the disease circulates among domestic pigs (*Sus scrofa domesticus*) and European wild boars (*Sus scrofa*), causing similar clinical signs and mortality in both populations (Gogin *et al*., 2013). ASFV is likely to have been introduced into Georgia *via* imports of contaminated pig products from Eastern Africa or Madagascar (Rowlands *et al*., 2008). Since then, the disease has subsequently spread to Eastern

Table 1: ASFV: Architectural proteins and assembly-related proteins.

Proteins involved in assembly	Genomic coding region	Viral components and affiliated elements
P72, P49 and P14.5	B646L, B438L and E120R	Capsid
P17 and pE183L	D117L and E183L	Inner envelope
p5, p14, p34, p37 and p150	CP2475L	Core shell
Histone-like DNA-binding protein and DNA binding protein p10	A104R and K78R	Nucleoid
Topoisomerase II	P1192R	Yet to determine its associated affiliation

Source: Urbano and Ferreira *et al*., 2020.

Europe and the Baltic countries, most likely through movements of infected wild boars and domestic pigs and contaminated pig products.

Pig-to-pig transmission

Several experimental studies demonstrated that direct contact with infectious domestic pigs is an effective mechanism of ASFV transmission. Susceptible pigs housed together with pigs infected with the ASFV strains from Lithuania and Georgia became infected by direct contact after one to nine days post-exposure (dpe) (Gallardo *et al*., 2015). When contact pigs were separated from the infectious pigs by solid partitions to prevent direct pig contact between pens, the transmission occurred after six to 15 dpe (Guinat *et al*., 2014). The basic reproduction number (R0, *e.g*, the average number of newly infected animals caused by one infectious animal) was estimated for the Malta ASFV strain at 18.0 (95 per cent confidence interval [CI]: 6.9 to 46.9) and for the Georgia and Russia ASFV strains at 1.4 (95 per cent CI 0.6 to 2.4), 2.8 (95 per cent CI 1.3 to 4.8) and 9.8 (95 per cent CI 3.9 to 15.6) (Guinat *et al*., 2014), depending on the transmission scenarios.

Feed-to-pig transmission

ASFV can persist for months in pork meat, fat and skin and in different types of pork products, such as sausages and salami stored under experimental conditions at negative and room temperature. Therefore, swill feeding, a common practice in the traditional pig production systems with free-ranging and backyard pigs globally (Costard *et al*., 2009) could play an important role in the ASFV transmission to domestic pigs.

Wild boar-to-pig transmission

Experimental studies demonstrated that wild boars were as susceptible as domestic pigs to ASFV infection using highly virulent ASFV strains from Armenia (2008) and Chechnya (2009) (Gabriel *et al*., 2011). Wild boars developed non-specific clinical signs, similar to those observed in domestic pigs, including fever, loss of appetite, diarrhoea and lethargy and died within seven to nine days, regardless of age or sex. ASFV is therefore likely to transmit between wild boars by contact with infectious wild boars, infectious free-ranging pigs or carcases of infected pigs or wild boars improperly disposed of by farmers or hunters (Gabriel *et al*., 2011).

Fomites-to-pig transmission

Studies have provided the range of possible environmental sources for ASFV transmission to domestic pigs. ASFV can persist for weeks in blood, faeces and urine excreted in the environment by infected pigs (Parker *et al*., 1969).

Tick-to-pig transmission

One experimental study has, however, indicated that ASFV Georgia strain was able to replicate in *O. erraticus* ticks that are commonly found in Southern Europe and remain for at least 12 weeks (Diaz *et al*., 2012). However, ASFV was not able to replicate in hard ticks (Ixodes ricinus and Dermacentor reticulatus), that are also commonly found in Europe, suggesting a limited vector competence for this tick family (Diaz *et al*., 2012).

Clinical signs

The clinical presentation and the gross pathological lesions of ASF in domestic pigs may vary depending on the virulence of the virus isolate, the route and dose of infection and host characteristics. The clinical courses observed in ASF in domestic pigs can be described as per acute (or hyperacute), acute, subacute, or chronic (Salguero, 2020)

Peracute ASF

Highly virulent strains are typically responsible for this clinical course, characterized by a very rapid clinical course, with high fever (up to 42°C), anorexia, lethargy and sometimes sudden death without signs of disease (Salguero, 2020).

Acute ASF

This clinical form is cause by highly or moderately virulent isolates and it is the typical course observed in naïve farms very quickly after the first fatal cases are reported. The clinical course is characterized by high fever, with temperatures of 40-42°C, lethargy, anorexia and inactivity. Many affected animals show centripetal cyanosis, easily found in the ears, snout, limbs, abdomen, tail and perianal area. Other clinical signs may include nasal discharges, sometimes stained with blood (epistaxis), vomiting and diarrhoea, which can be also blood-stained (melaena). At the post-mortem examination, the most characteristic lesion of acute ASF is the haemorrhagic splenomegaly with a very enlarged spleen, dark in colour and friable at sectioning, occupying a large space within the abdominal cavity. The second most important lesion described in acute ASF is multifocal haemorrhagic lymphadenitis. Lymph nodes can have multifocal or extensive haemorrhages that can produce a marbled appearance (Salguero, 2020).

Subacute ASF

This clinical form is usually observed in animals infected by moderately virulent isolates, with similar clinical signs as those observed in acute ASF, although normally less marked. Affected pigs show moderate to high fever and the mortality rate ranges from 30 to 70%, with pigs dying at 7-20 after infection (Salguero, 2020).

Chronic ASF

This clinical form is caused by the infection of low-virulence isolates and has been observed, quite infrequently, in the Iberian Peninsula and the Dominican Republic. It has been hypothesised that this low virulence isolates and the associated chronic form, have evolved from ASFV isolates employed in early vaccine trials carried out in the Iberian Peninsula in the 1960's. This clinical form is characterized by multifocal necrosis in the skin and arthritis, growth retardation emaciation, respiratory distress and abortion (Salguero, 2020).

Epidemiology

The African swine fever virus is a highly contagious viral disease that affects domestic pigs and wild suids. Its epidemiology varies across different regions, driven by host species, vectors and the interplay between them. In endemic areas, ASF can manifest in chronic or subclinical forms, leading to decreased mortality rates (Allaway *et al*., 1995; Fasina *et al*., 2010; Owolodun *et al*., 2010). In the Iberian Peninsula, chronic forms of ASF were linked to infections with low-virulence ASFV viruses, possibly due to the use of live attenuated vaccines during the 1960s (Sanchez-Vizcaino *et al*., 2012).

In Africa, ASFV resistance has been proposed due to acquired immunity from prior exposure to lower virus doses or related viruses with reduced virulence, possibly originating from circulation in domestic pig populations (Penrith *et al*., 2004). Additionally, local pig breeds were suggested to be genetically less susceptible to ASFV, although increased resistance was not inheritable (Penrith *et al*., 2004). The evolutionary study of ASFV with p72 protein from the ASFV of the B646L genes indicated that they play a vital role in the evolutionary process. study also helps other researchers to predict and develop vaccines or drugs that might alter the pathogenic characteristics of the ASFV and control the spread of viruses and disease (Penrith *et al*., 2009).

Sub-clinically infected, chronically infected, or recovered pigs play crucial roles in ASF's epidemiology, contributing to disease persistence in endemic areas and causing sporadic outbreaks (Allaway, 1995; Boinas *et al*., 2004; Leitao *et al*., 2001; Sánchez-Vizcaíno and Arias, 2012; Wilkinson 1984). Although no long-term carrier state has been proven, infected pigs can remain carriers for several weeks, transmitting the virus to susceptible pigs through direct contact, tick bites, or ingestion of contaminated meat and products (Wilkinson, 1984).

Wild suids, such as warthogs (*Phacochoerus africanus*), are considered the original vertebrate hosts of ASF and are involved in a sylvatic cycle with ticks of the O. moubata complex (Plowright *et al*., 1969). Warthogs act as asymptomatic carriers and ASFV persists in their lymph nodes, primarily transmitted to them by infected soft ticks in burrows (Thomson *et al*., 1980).

Other wild suids, like bushpigs and red river hogs, may have a role in ASF's epidemiology, although their exact involvement is not fully understood (Haresnape *et al*., 1985; Jori and Bastos, 2009). ASFV replication has been demonstrated in bushpigs, but the mechanisms of transmission to other hosts remain unclear (Anderson *et al*., 1998; Oura *et al*., 1998).

In Europe, wild boars and feral pigs have shown similar susceptibility to ASFV as domestic pigs (Jori and Bastos, 2009; McVicar *et al*., 1981). ASF outbreaks in wild boars tend to fade out but can facilitate disease spread in areas with free-range pigs, primarily through direct contact, fomites, or ingestion of infected carcasses (Laddomada *et al*., 1994; Mur *et al*., 2012a; Perez *et al*., 1998; Ruiz-Fons *et al*., 2008). In India a case report indicated that in a Government Pig Farm at Kanke, Ranchi (Jharkhand) ASFV caused 95.3% mortality in pigs wiping out 1240 pigs out of 1300 stock in various age group in span of 30 days (Shreya *et al*., 2024).

Soft ticks, particularly O. *moubata*, play a crucial role in ASFV transmission. They serve as biological vectors and reservoirs for the virus, infecting both domestic and wild pigs during blood meals. Infected ticks can retain the virus for extended periods and transmit it to susceptible hosts, contributing to the persistence of ASF in the absence of viraemic hosts (Plowright *et al*., 1969).

Overall, ASF's epidemiology is complex and varies regionally, influenced by factors such as host species, vectors and ecological interactions. Understanding these dynamics is essential for effective ASF control and prevention strategies. In total, since January 2021 ASF has been reported as present in five different world regions in 41 countries, affecting more than 828,000 pigs and more than 23,000 wild boars (data reported through INs and FURs), with more than 1,000,000 animal losses (WOAH, Feb 2023).

Pathogenesis

The infection is characterized by a complex pathogenesis involving various stages of viral replication and immune response which are listed below (Salguero *et al*., 2020):

Initial infection

ASFV initially enters pigs through an oral-nasal route or via the bite of an infected soft tick. The virus then targets the tonsils or regional lymph nodes, where it starts replicating.

Lymphatic and hematogenous spread

Within 2-3 days, the virus spreads through the lymphatic and circulatory systems to secondary organs for replication and then to various other organs in the body.

Target cells

Monocytes and macrophages are the primary target cells for ASFV infection. Despite being a DNA virus, ASFV replicates in the cytoplasm of these cells, not the nucleus.

Cellular changes

Infected monocyte-macrophages exhibit distinct changes, including cellular swelling and the formation of intracytoplasmic inclusion bodies. Viral factories are visible under electron microscopy.

Cell death

The virus induces cell death in the infected monocyte-macrophages, which can occur through apoptosis or necrosis. ASFV carries genes involved in programmed cell death, affecting the survival of infected cells.

Destruction of lymphoid organs

ASFV infection results in the massive destruction of lymphoid organs and tissues, including the spleen, lymph

nodes, thymus and tonsils. This leads to a significant loss of B and T lymphocytes and macrophages.

Cytokine Storm

Virus replication in monocyte-macrophages triggers their activation and an increase in the secretion of proinflammatory cytokines such as IL-1, TNF-α and IL-6. This cytokine storm is responsible for the widespread induction of apoptosis in neighbouring lymphocytes.

In summary, ASFV infection follows a multi-stage pathogenesis, starting with initial infection in lymphoid tissues, replication in monocyte-macrophages and leading to the destruction of lymphoid organs and tissues. The activation of infected cells and the subsequent cytokine storm contribute to the depletion of lymphocytes, a characteristic feature of ASFV infection. This complex interplay between the virus and the host's immune response plays a crucial role in the disease's progression and severity (Francisco *et al*., 2020).

Diagnosis and control

Effective containment of ASFV demands stringent quarantine measures and accurate laboratory diagnostics for early detection and epidemiological data gathering (Gallardo *et al*., 2019). Real-time PCR (qPCR) is the preferred method due to its sensitivity, specificity and high-throughput capabilities in detecting ASFV genomes. Various PCR assays, including conventional and qPCR, have been developed and validated using the highly conserved p72 protein gene encoding ASF (Mur *et al*., 2013). Isothermal amplification, while a cost-effective alternative for field use, may have limitations in detecting the virus in recovered or carrier animals.

Many available ELISA kits for detecting African swine fever virus antibodies target specific proteins like p72, p30 and p54. However, the discovery of low-virulence viruses with deleted genes has led to a demand for more sensitive tests that cover a wider range of detection targets in ASFV detection (Zhang *et al*., 2023).

Indirect enzyme-linked immunosorbent assay (ELISA) methods were developed by different research groups to identify African swine fever virus (ASFV) strains. Purified CD2v extracellular domain protein was used as the detection antigen, demonstrating excellent specificity for CD2v-deleted ASFV and high sensitivity, allowing identification of infected clinical serum samples diluted up to 1:2,560 (Jiang *et al*., 2022). A prokaryotic recombinant pB602L protein which is a late non-structural protein with strong antigenicity achieved high specificity and sensitivity having no cross-reactions with antibodies from other tested swine viruses, detecting anti-ASFV antibodies in serum samples diluted up to 1:6,400 (Yang *et al*., 2022). An ELISA kit developed using prokaryotic expression of proteins p22 and p30, exhibited high sensitivity and outperformed a commercial kit by detecting positive serum samples at dilutions as high as 1:12,800 (Li *et al*., 2023). Other Serological tests like complement antigen-based methods, while blood adsorption and virus isolation are sensitive but labour-intensive confirmatory tests (Zhang *et al*., 2023).

Commercial kits for ASFV genomic testing, especially real-time PCR kits, have become prevalent. Hemadsorption inhibition assays (HADIA) provide valuable insights into ASFV strain phenotyping, aiding in immunopathology and vaccine research (Li *et al*., 2022).

Preventing and controlling African swine fever virus (ASFV) encompasses a multifaceted approach. Following are the summarised approaches for an effective control of disease which were implied in many countries so far (Liu *et al*., 2021).

ASFV vaccine development

The primary goal is to create a vaccine capable of effectively preventing ASFV infections in domestic pig populations. The challenges lie in the virus's complex structure and the multitude of proteins it encodes, making it difficult to pinpoint key antigenic epitopes (Gavier *et al*., 2020). Despite decades of research, a fully effective vaccine remains elusive. Live-attenuated virus vaccines (LAVs) have shown promise, with recent gene-deleted LAVs demonstrating potential. However, challenges such as the lack of a suitable cell line for production and the need for a differential labelling technique to distinguish vaccinated animals from infected ones hinder progress (Borca *et al*., 2020).

Anti-ASFV drugs

Over the last few decades, certain compounds and commercially accessible drugs exhibiting anti-African swine fever virus (ASFV) activity *in vitro* have been identified. The inhibitory potential of aUY11, an aromatic nucleoside derivative was highlighted which not only against viruses like Influenza A virus (IAV) and hepatitis C virus (HCV) but also demonstrated significant anti-ASFV activity (Colpitts *et al*., 2013). Nevertheless, investigations into these compounds have been limited to cellular-level studies under *in vitro* conditions and their prospective impacts on ASFV-infected pigs are yet to be established (Liu *et al*., 2021).

ASFV-resistant pigs development

Selective breeding and genetic approaches are being explored to develop pigs with inherent resistance to ASFV. The objective is to create a swine population that is more resilient to ASFV infections, potentially reducing the susceptibility of pigs to the virus and limiting its spread within the population. A Chinese research team disclosed the identification of LS-2, domestic pigs resistant to African swine fever virus (ASFV). This ground breaking discovery marked the first successful identification of ASFV-resistant domestic pigs globally, signifying a crucial advancement in the screening and development of pigs resistant to ASFV. (Chen *et al*., 2020).

Efficient disinfection practices

Proper disinfection protocols are crucial for preventing the transmission of ASFV. This involves the implementation of

thorough and effective disinfection measures on farms and in pig-rearing facilities. Disinfection helps minimize the presence of the virus in the environment, reducing the risk of contamination and subsequent outbreaks (Block *et al*., 2001).

High-Level Biosecurity

Stringent inspection and quarantine measures are essential at international airports, shipping terminals and railway stations to prevent the introduction of pork products by international passengers. Proper disposal of leftover food from international flights, ships, or trains is crucial. In the event of a confirmed ASFV-positive farm, a 3-km protection zone and a 10-km surveillance zone should be established around the infected area, with strict restrictions on pig transportation in these zones. The affected pig farm must undergo depopulation and the culled pigs should be either incinerated, deep-buried, or composted. Additionally, the farm area and all equipment must undergo thorough disinfection, cleaning and drying for a minimum of 40 days (Guinat *et al*., 2017).

Repopulation of pigs

After an ASFV outbreak, strategic repopulation is necessary to restore pig populations and rebuild the swine industry. This involves carefully reintroducing healthy and disease-resistant pigs into affected areas. The process requires meticulous planning to prevent the recurrence of the virus and ensure the long-term health of the swine population (Liu *et al*., 2021).

In summary, the prevention and control of ASFV involve a multi-pronged approach that addresses vaccine development, drug discovery, genetic resistance in pigs, efficient disinfection, high-level biosecurity and strategic repopulation. Each of these components plays a crucial role in managing the complex challenges posed by ASFV and safeguarding the well-being of swine populations.

Effectively managing African Swine fever disease is crucial, bearing significant implications for a country's food security and economic stability. An epidemiological study conducted on 68 outbreaks in China between August and November 2018 unveiled that irregular transport of live pigs and pork products contributed to 19% of the cases, while 46% were attributed to vehicles and individuals carrying the virus and 34% to slop feeding (Li *et al*., 2022). Another epidemiological investigation in Kerala, India, indicated that swill feed was likely the primary source of infection, leading to outbreaks in four nearby farms (Jagadish *et al*., 2023). All these studies suggest that for effective control of disease a total restriction on swill feed and strict biosecurity as the only measures even when an effective vaccine is available shortly.

Research on antivirals has identified iododeoxyuridine and adenosine analogues targeting S-adenosylhomocysteine hydrolase as effective against ASFV *in vitro* (De Clercq, 2009). Porcine IFN-gamma and siRNAs have also shown inhibitory effects, while CRISPR/Cas9 targeting specific gene regions has inhibited ASFV (Esparza *et al*., 1988; Hübner *et al*., 2018; Erik *et al*., 2019).

Vaccination with inactivated ASFV vaccines is deemed non-protective (Stone *et al*., 1967). Recombinant virus ASFV-G-DA137R has shown a completely attenuated phenotype, serving as a potential live attenuated vaccine candidate (Tran *et al*., 2022). Genetically engineered vaccines targeting various ASFV proteins have been studied, with ASFV-G-DI177 showing effective protection against virulent strains (Uma *et al*., 2021). However, vaccines alone cannot replace the need for behaviour change in the pork value chain to effectively control ASFV spread, considering challenges in achieving high vaccination coverage and the virus's extended survival period in pork products (Dixon *et al*., 2020).

CONCLUSION

Over a century of spread and evolution, ASF has threatened the world's pig herds more than ever. As there is no ASF vaccine, control of the ASF epidemic largely depends on biosecurity measures. At the same time, practitioners have weak biosecurity awareness and strict biosecurity has not formed a strong line of resistance to viruses. Although several countries banned the ASF vaccine, the development of an ASF vaccine will be beneficial for the control and eradication of ASF based on the current situation. The complex structure of ASFV, its large genome and the diversity of viral genomes among different strains add to the difficulty of developing a vaccine. The structure and function of the main ASFV proteins, infection and immune mechanisms must be fully understood and major immunogens must be identified. Despite the availability of basic information about the ASFV replication mechanism and the process of virus entry/internalization and endosomal transport, many details about the specific process of virus replication remain unclear. Until a vaccine becomes available, biosecurity may be the most important measure to prevent the spread of ASF. The research on ASFV media should be accelerated, including animal factors, human factors and environmental factors. Only through understanding the role various factors play in the spread of the virus can more purposeful measures to prevent and control be taken. Similarly, ASF pathological diagnosis and laboratory diagnostic technology research also occupy an important position in the prevention and control of ASF.

Conflict of interest

Authors declare that they do not have any conflict of interest.

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