African Swine Fever: an Overview

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Key words

Swine – Swine fever virus – Epizootology – Diagnosis – Monitoring – Control methods – Disease control – Africa.

Summary

The African swine fever (ASF) is an expanding devastating viral disease currently threatening the pig industry worldwide. The virus is an icosahedral cytoplasmic deoxyribovirus (ICDV) of the *Asfarviridae* family. It is an arbovirus (transmitted by ticks) and it is the only arbovirus that contains DNA. Wild suids of Africa, mainly the warthog and bushpig, are the original vertebrate hosts of ASF. Domestic pigs are infected by ticks (*Ornithodoros moubata)* or by direct contact with wild suids. Transstadial and transovarian infections occur in ticks. Recently, ASF caused high morbidity and mortality in domestic pigs in Nigeria and other West African countries previously known to be free of the disease. No protective vaccination has been discovered; hence, a slaughter policy with adequate compensation, strict quarantine of pigs and their products at borders are necessary to stop the current outbreaks of ASF in Africa, particularly in West Africa. To eradicate ASF it has become very urgent to further promote awareness of ASF, to cook swill before serving to pigs, and to enforce regulations against free-ranging pigs. Research in vaccine production against ASF should be intensified. Governments must improve veterinary ambulatory and diagnostic services as well as the information network since ASF is a notifiable disease.

■ INTRODUCTION

The African swine fever (ASF) is known as *peste porcina africana*, *pestis africana suum*, *maladie de Montgomery*, warthog disease, *peste porcine africaine*, *Afrikaanse varkpes*, *Afrikanische Schweinepest* (52). Recently, the ASF virus has been allocated to a new genus, *Asfarvirus*, of the family *Asfarviridae* (22). ASF in domestic pigs varies from peracute, acute, subacute, and chronic to inapparent infections. Clinical manifestations of ASF may resemble those of hog cholera (19).

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■ INITIAL OUTBREAKS

Montgomery (21) first reported ASF from East Africa, while Steyn (53) later reported it in South Africa. De Kock et al. (4) described outbreaks occurring in 1933 and 1934 in South Africa as serious and involving 11,000 swine of which more than 8000 died, 2000 were slaughtered and only 862 were considered as survivors. Velho (55, 56) mentioned enzootic ASF in free-ranging swine in Angola. ASF was restricted during the early period with several severe outbreaks occurring in Eastern and Southern Africa (52). ASF was first reported in West Africa in Senegal in 1978 (6) and later in Cameroon in 1982, killing about 80% of all pigs in the latter country.

In 1957, outbreaks with high mortality occurred in Portugal (50, 52), and in Spain in 1960 (47). France experienced outbreaks in 1964, Italy in 1967 and Belgium in 1985 (52). Emergency eradication measures were taken which consisted essentially in slaughtering all infected and exposed swine, restricting swine and

pork movements, and cooking all garbage containing pork before allowing it to be fed to swine. This approach quickly eradicated the initial outbreaks in Europe (13, 52). ASF was thought to have been probably carried from Spain to Cuba in 1971, from where it spread throughout the Caribbean, and then occurred in Brazil in 1978 (52). It is generally believed that garbage containing uncooked pork in airplanes from Spain or Portugal was responsible for the outbreaks, since initial outbreaks occurred in swine fed uncooked garbage from international airports (13). Also, initial outbreaks in Malta and Sardinia were reported in swine fed uncooked garbage from seaports (48). In Cuba, emergency eradication measures were quickly and decisively applied in which more than 400,000 swine in the province of Havana were killed. Also, movements of swine and pork were restricted, while feeding of raw garbage to swine was forbidden. These efforts quickly eradicated ASF (diagnosed in May) by August 1971 with no recurrence reported ever since (14).

■ CURRENT OUTBREAKS

Southern Africa

A narrow zone in the north of South Africa has been delineated as an enzootic zone wing due to the presence of infected warthogs (35, 49, 52). Also, continued outbreaks have been reported in South Africa (27, 35), Zambia (26, 36), Mozambique (29), Botswana (28), and ASF outbreaks were reported for the first time in Malawi in 1989 and up to 1991 (25). ASF has been reported in Madagascar since 1998 (7, 28) and was also reported in the Democratic Republic of Congo (31, 32).

East Africa: Kenya

ASF outbreaks occurred in Nairobi in 1994 (37) after an absence of 30 years. In Kenya, there was no evidence for the involvement of warthogs or ticks, and the disease is believed to have resulted from movements of infected pigs from enzootic areas. Currently, outbreaks of ASF have been reported in Kenya (37).

West Africa

Apart from Senegal, Cameroon, Cape Verde Island and Guinea Bissau that were affected by ASF in 1978, new countries in West Africa have been currently infected by ASF. Such new countries include Côte d'Ivoire (26), Benin (33), Togo (34), Nigeria (23) and Ghana (28). Côte d'Ivoire was affected by ASF in 1996, Benin, Togo and Nigeria have been reporting outbreaks since 1997 (6). Outbreaks of ASF have recently been reported in Senegal, Cameroon, Ghana, Togo, and Benin (30). It is assumed that the Cameroon mountain at the Nigeria border with the border control of pigs' movements have in the past prevented the spread of ASF to Nigeria.

Although warthogs are widespread in the West African savannah, *Ornithodoros moubata* has not been reported in the region while the few warthogs that have been tested (2 from Nigeria, 2 from Senegal and 3 from Benin) proved negative for ASFV (42). Furthermore, the outbreaks as far as can be ascertained started in the coastal zone, where there are no warthogs. The infection can therefore be assumed to have originated with movements of infected domestic pigs or their products.

Europe and United States

African swine fever outbreaks have been reported in Portugal in 1999 (28) and in Italy since 1993 (16). The disease has also been reported in wild boars in Spain since 1991 (43). High risks of ASF in the Netherlands (12) and US hogs (20) have yet to occur.

■ EPIZOOTIOLOGY

The African swine fever virus (ASFV) is the only DNA virus that can be classified as an arbovirus. It replicates to a high titer within the larval cells of the developing egg of the argasid tick, *Ornithodoros moubata* (49). In Africa and Southern Europe, *Ornithodoros* ticks act as reservoirs. Ticks readily become infected while engorging on infected swine. They can retain the virus and transmit it to swine for as long as eight years or probably throughout their life span (14, 44). ASFV is transmitted from tick to tick sexually, transovarially and transstadially (45, 49, 52). Thus it is quite reasonable to believe that the virus is primarily a virus of ticks (44).

It has also been indicated that any bloodsucking insects, such as mosquitoes, horseflies, etc., feeding on infected viremic pigs and subsequently on non-infected swine within the same herd or other herds within the vicinity could transmit ASFV (14). Also, propagation of ASFV in mosquito cell cultures growing at 26-28°C has been achieved (10).

The original vertebrate hosts of the ASF virus are the wild suids of Africa, especially the warthog and to a lesser extent the bush pig (52). African wild suids, the warthog (*Phacochoerus africanus*), the bush pig (*Potamochoerus porcus*) and the giant forest hog (*Hylochoerus* spp.) only become infected with no clinical expression and are the reservoirs of infection for the disease in Africa. The collared peccary *(Tayassu tajacu*) in the USA, the white-lip peccary (*Tayassu albirostris)* of South and Central America are also supposed to become infected healthy carriers (52). The wild boar in Europe is not a reservoir of infection because it is killed by the disease as is the domestic pig.

The epizootiological conception of ASF in Africa could be divided into an old silvatic cycle dependent on inapparent maintenance in warthogs or other wild suids. The new cycle is based in the virus maintained in domestic pigs (52). Wild suids are already infected as piglets by *Ornithodoros moubata* ticks, which transmit the virus transstadially and transovarially. Then the infection is also carried from the population of wild suids to domestic pigs by *O. moubata,* and is a vector-borne disease up to this stage. The contact between infected wild suids and domestic pigs usually results in a disease outbreak in domestic pigs (52). While no excretion of the virus could be observed in warthogs, infected domestic pigs are continuously excreting the virus in nasal secretions, blood, feces and urine. The infection being a contact disease among domestic pigs, it is transferred orally and nasally. Due to the high resistance of the virus to environmental influences, the oral infection with contaminated feed is very important in domestic pigs (52).

■ CLINICAL SIGNS

Clinically, the first sign of ASF is reduced appetite but, more often, sudden death in pigs is the first indication noted by farmers (19, 52). Clinical manifestations of ASF may resemble hog cholera (19). Initially, the diarrhea is mucoid, while in some pigs it later becomes bloody. Almost all pigs that develop severe bloody diarrhea die. Affected pigs spend much time lying down, but when aroused, they often eat limited amounts of food and frequently drink some water. Body temperature ranges between 40.5-42.2°C and hyperemia of the skin is usually marked over the mouth, ear, under the belly, over the hindquarters and around the fetlocks, especially in white pigs (8, 52). Hemorrhages are also found in many internal organs, especially in lymph nodes.

Pregnant sows often abort at any stage of pregnancy soon after clinical signs develop. Petechial and ecchymotic hemorrhages may

occur on the fetal membranes and skin of the aborted fetus (18). An incubation period of 2-19 days leads to either a peracute or acute course of ASF, which is characterized by high fever, thirst, inappetance, apathy, staggering gait, muscle tremors and dyspnea with increased respiration (52). The acute stage may last for seven days and the subacute up to 70 days. Generalized hemorrhages are consistent with the acute form of the disease (43). During the peracute course of ASF, death may appear suddenly without clinical symptoms. During natural infection, only a few pigs in a group are initially sick and the spread of the disease throughout the remainder of the herd may take several weeks with low virulent strains, or only a few days with highly virulent viruses. Incubation periods vary from two to five days in experimentally infected pigs (10, 52).

A chronic course may sometimes appear and may last for 2-15 months. It is characterized by intermittent fever, emaciation, swelling of the joints and sheaths of the tendons, keratitis and atrophy of the bulbus (52). Coughing often occurs in the chronic form. Diarrhea and occasional vomiting may be present. Morbidity can approach 100% and mortality can also be above 80%. Chronically infected domestic pigs excrete the virus during the whole course of the disease (52).

■ PATHOLOGY

The usual route of an ASF infection in pig is via the mouth or the upper respiratory system (9). The ASF virus provokes proliferation of lymphocytes and reticular cells. After four days, degeneration becomes more prominent than proliferation. The gross pathological changes are similar to those of hog cholera characterized by lesions of the circulatory system (52). Generally, the lesions are proportional to the amount of damage to the circulatory system. Also, these lesions are usually marked in acute cases that are due to highly virulent viruses, whereas they may be minimal or absent when due to low virulent viruses causing inapparent or chronic forms of the disease (14, 52). The circulatory system is probably the most severely damaged tissue of the body as evidenced by congestion, edema, ascites, hydrothorax, hydropericardium, infarctions and occasional necrosis in many visceral organs, and changes in the blood. Petechial and ecchymotic hemorrhages are often seen on the epicardium, around engorged coronary blood vessels on the epicardium and endocardium. Also, they are found around attachments of the chordae tendinae to the valves and endocardium. Lymph nodes are hemorrhagic and edematous. Severely affected lymph nodes may look like hematomas. Spleen may be enlarged and lungs usually appear congested. Pneumonia may be seen in chronic cases (40). Kidneys and urinary bladder are usually seen with petechial and ecchymotic hemorrhages. Leucopenia is marked in acute ASF, with increase in the percentage of neutrophil mature forms especially and with concomitant drop in lymphocyte numbers (5).

In chronic ASF hypergammaglobulinemia is often marked (38). Also, the brain and meninges are often congested, edematous and may contain multiple hemorrhages (46, 52). Since the necropsy lesions resemble those of hog cholera, salmonellosis and erysipelas, laboratory confirmation of ASF is imperative.

■ DIAGNOSIS

A positive diagnosis of ASF may be by either virus detection or demonstration of ASF-specific antibody (10). The tests for detection of the ASF virus in infected tissues are listed hereafter.

Antibody detection

Enzyme-linked immunosorbent assays (ELISA) are the most recently developed serologic techniques for detecting antibodies. The technique is now widely used and recommended by the Office international des épizooties (OIE) (24, 51, 52). Sera from recovered animals can be screened by indirect immunofluorescence (IIF), immunoelectroosmophoresis (IEOP) and ELISA (52). Other tests for screening of antibodies against ASFV are the agar gel precipitation test (AGPT), complement fixation test (CFT), hemadsorption inhibition (HADI), immunofluorescence plaque assay (IPA), and immunoblot (2, 3, 41, 52). Routinely, radial immunoassay (RIA), reverse radial immunodiffusion (RRID), iodine agglutination test (IAT) and solid phase radioimmunoassay are used.

Virus isolation

For virus isolation, the hemadsorption (HA) test has been extensively used (2, 15, 54). The ability of ASF to produce HA in leukocyte cultures is still regarded by many as its most meaningful identifying feature. However, some non-hemadsorbing strains can be missed. Other tests to detect the ASF virus include animal inoculation, leukocyte culture for the autorosette test and electron microscopic examination of tissues (52).

Detection of genome nucleic acids

Methods to detect genome nucleic acids from the African swine fever virus in infected tissues are restriction fragment length polymorphisms (RSLP), polymerase chain reaction (PCR) and use of DNA probes (1).

Antigen detection

Methods for detecting the ASF virus antigen in infected tissues include direct immunofluorescence on buffy coat cells and on tissue smears or cryostat section. The method is very useful in acute cases of ASF. The virus is present in large amounts in the blood and in any tissue with a rich blood supply (10). The ELISA test has been adapted for detection and assay of antibodies against ASFV by some laboratories (43). It is the most sensitive test to detect singular perhaps chronically infected animals (24, 51) and has been recommended by OIE (24, 51, 52).

However, none of the above tests can be expected singularly to detect the disease under all conditions (10). Therefore, rapid diagnosis is of prime importance, while speed, convenience as well as accuracy and sensitivity must be considered in selecting the tests to be applied routinely (10, 52).

Comparative assessment of diagnostic techniques

From the long-time experience of Spanish workers in diagnosing ASF, the HA test was generally regarded as *the* differential test for ASFV. The test has been the most widely used means to isolate the virus from field specimens. Spanish workers assigned a sensitivity of 98.9% to the HA test and also have used it as the basis for evaluating the other diagnostic tests (10). Although indirect IIF was reported to have a sensitivity of 82% in detecting antibodies in tissue extracts of ASF-positive cases, it is not suitable for testing large numbers of serum samples.

More than 90% of ASF infected pigs can be detected by the demonstration of specific antibodies against ASFV and the technique is suitable for testing large serum samples (10). Thus techniques based on antibody detection, such as ELISA and immunoblotting assay, are currently the most frequently used (41).

■ SEROSURVEILLANCE

Up till now, a safe and effective vaccine against ASF has not been developed, hence rapid diagnosis, drastic slaughter and quarantine methods are required in controlling and eradicating ASF. Effectiveness of close surveillance by early diagnosis and rapid destruction of infected and exposed swine has been demonstrated in outbreaks in France, Italy, Cuba and South Africa (10).

A rapid and sensitive method recommended by OIE in broad programs of surveillance, especially one that detects ASF-specific antibodies in chronically infected animals, is ELISA (24, 51, 52) and has now been widely used. Antibodies to the ASF virus can be detected as early as three to four days after infection. Other antiviral antibodies are produced later especially the IgG class, which becomes readily detectable 14 days after infection if the animal is still alive. Although antibodies can persist throughout the life of the animal, there is no evidence that these antibodies are capable of neutralizing virus infectivity and therefore are probably not protective (52).

Once ASF has become enzootic in domestic pigs, serosurveillance will help in detecting surviving carriers. A simple and most effective means of recognizing such animals is the identification and demonstration of specific antibodies to ASFV. It is important that in enzootic areas investigation of a new outbreak starts with the detection of specific antibodies; hence serosurveillance becomes an important component of any control strategy of ASF.

■ TREATMENT AND CONTROL

There is yet no effective drug treatment or cure for ASF. Also, it has not been possible so far to produce effective inactivated or attenuated live vaccines or immunoprotection (42). Hess and Pan (11) did not find the humoral or cellular immune system impaired in pigs that had recovered from ASF. However, the inability of antibodies to neutralize the virus dims the hope for an effective vaccine. Application of attenuated virus strains as vaccine antigens has produced outbreaks of chronic diseases, which contributed to further spreading the infection, while sera from recovered animals also only gave incomplete protection against the lethal infection (52).

It has been suspected that inapparent carriers among domestic pigs are playing a major role in maintaining the disease in the enzootic areas (10). Thus, the stamping out policy as applied in the past will be the only means of eradicating the current outbreaks of the disease especially in the new West African countries affected, i.e. all sick/infected and exposed swine will have to be eliminated and disposed of from the affected premises. The standard sanitary measures of quarantine, decontamination and carefully controlled and monitored restocking must be rigidly enforced. All slaughtered sick and exposed pigs must be disposed of by burial or incineration. Some surface active agents containing o-phenylphenol and iodine compounds are effective in destroying the ASF virus on environmental surfaces and in waste materials (10). However, controlled processing and distribution of healthy-appearing animal carcasses for human consumption may speed up eradication and substantially reduce the cost of an eradication program (10), especially in poor African countries with the heavy burden of protein malnutrition. The inability of governments to compensate farmers limits the effectiveness of an eradication policy in the currently affected areas of West Africa. Also, rigorous prevention of contact between domestic pigs and wild reservoirs through free-ranging pigs must be maintained during outbreaks (46).

Swill from airports, seaports and other areas must be cooked before fed to swine. Control of ectoparasites like *Ornithodoros* ticks and hematophagous flies should be routinely carried out in pig farms, especially during ASF outbreaks. A claim that the pig louse, *Haematopinus suis*, can transmit the ASF virus has not been substantiated by further studies, while it has been established that the stable fly, *Stomoxys calcitrans*, can retain high levels of virus for two days. It was also found out that the fly can transmit the virus to pigs 24 hours after feeding on an infected pig (42). Since ASF is an expanding disease whose costs of eradication far exceed those of prevention, no country that looks forward to a thriving swine industry and agricultural products that are acceptable in world trade should ignore the threat of ASF (10).

REFERENCES

1. ALCARAZ C., RODRIGUEZ F., OVIEDO J.M., EIRAS A., DE DIEGO M., ALONSO C., ESCRIBANO J.M., 1995. Highly specific confirmatory western blot test for African swine fever virus antibody detection using the recombinant virus protein. *J. Virol. Method:* 111-119.

2. COGGINS L., 1968. Modified haemadsorption inhibition test for African swine fever virus. *Bull. Epizoot. Dis. Africa,* **16**: 61-64.

3. COGGINS L., HEUSCHELE W.P., 1966. Use of agar diffusion precipitation test in the diagnosis of African swine fever. *Am. J. vet. Res.,* **27**: 485.

4. DE KOCK G., ROBINSON E.M., KEPPEL J.J.G., 1940. Swine fever in South African Onderstepoort. *J. vet. Sci. Anim. Ind.,* **14**: 31.

5. DETRAY D.E., SCOTT G.R., 1957. Blood changes in swine with African swine fever. *J. Am. vet. Med. Assoc.,* **126**: 131-314.

6. FAO, 2000. African swine fever. *Empress-Transboundary Anim. Dis. Bull.,* No 14/2: 15-17.

7. GONZAGUE M., ROGER F., BASTOS A., BURGER C., RANDRIAPARANY T., SMONDACK S., CRUCIERE C., 2001. Isolation of a non-haemadsorbing, non cytopathic strain of African swine fever virus in Madagascar. *Epidemiol. Infect.,* **126**: 453-459.

8. GREIG A., 1972. Pathogenesis of African swine fever in pigs naturally exposed to the disease. *J. comp. Pathol.,* **82**: 73-79.

9. GREIG A., PLOWRIGHT W., 1970. The excretion of two virulent strains of African swine fever virus by domestic pigs. *J. Hyg.* (Camb.), **68**: 673-682.

10. HESS W.R., 1981. African swine fever: a reassessment. *Adv. vet. Sci. comp. Med.,* **25**: 39-69.

11. HESS W.R., PAN I.C., 1977. The immune response in African swine fever. In: Proc. Agric. research semin. Hog cholera/classical swine fever and African swine fever, Tierarztliche Hochschule, Hanova, Germany, 6-11 Sept. 1976. Luxembourg, Directorate-General XIII, EUR 5904, p. 602-611.

12. HORST H.S., DIJKHUIZEN A.A., HUIRNE R.B.M, LEEUW P.W., 1998. Introduction of contagious animal diseases into the Netherlands: elicitation of expert opinions. *Livest. Prod. Sci.,* **53**: 253-264.

13. KORN G., 1969. Epidemiology, diagnosis and control of African swine fever and hog cholera in Spain, France and Italy. *Tierrnerztl. Umsch.,* **24**: 124-126.

14 LEMAN A.D., GLOCK ROBERT D., MENGELING WILLIAM L., PENNY R.H., SCHOLL E., STRAW B., 1981. Diseases of swine, 5th Edn. Ames, IA, USA, Iowa State University Press, 832 p.

15. MALMQUIST W.A., HAY D., 1960. Haemadsorption and cytopathic effect produced by African swine fever virus in swine bone marrow and buffy coat culture. *Am. J. vet. Res.*, **21**: 104.

16. MANNELLI A., SOTGIA S., PATTA C., OGGIANO A., CARBONI A., COSSU P., LADDOMADA A., 1998. Temporal and spatial patterns of African swine fever in Sardinia. *Prev. vet. Med.*, **35**: 297-306.

17. MAURER F.D., GRIESEMER R.A., JONES T.C., 1958. The pathology of African swine fever. A comparison with hog cholera. *Am. J. vet. Res.,* **19**: 517.

18. MCDANIEL H.A., 1979. African swine fever. In: Proc. 21st annual meeting. *Am. Assoc. vet. Lab. Diagn.*, **21**: 391-397.

19. MCDANIEL H.A., 1986. African swine fever. In: Leman Glock A.D., Mengeling R.D.W.L., Penny R.H.C., Scholl E., Straw B. Eds, Diseases of swine, 6th Edn. Ames, IA, USA, Iowa State University Press, p. 237- 245.

20. MILLER HAYS S., 1996. African swine fever poses risk to U.S. hogs. *Agric. Res.,* **44**: 14-17.

21. MONTGOMERY R.E., 1921. On a form of swine fever occurring in British East Africa (Kenya Colony). *J. comp. Pathol.*, **34**: 154, 242.

22. MURPHY F.A., GIBBS E.P.J., HORZINEK M.C., STUDDERT M.J., 1999. Veterinary virology. New York, NY, USA, Academic Press, 629 p.

23. ODEMUYIWA S.O., ADEBAYO I.A., AMMER LAAN W., AJUWAPE A.T.P., ALAKA O.O., OYEDELE O.I., SOYELU K.O., OLALEYE D.O., OTESILE E.B., MULLER C.P., 2000. An outbreak of African swine fever in Nigeria. Virus isolation and molecular characterization of the VP72 gene of a first isolate from West Africa. *Virus-Genes,* **20**: 139-142.

24. OIE, 1990. Recommended diagnostic techniques and requirements for biologic products, Vol. II. Paris, France, OIE.

25. OIE, 1995. An outbreak of African swine fever in the southern region of Malawi. *Revue sci. tech. Off. int. Epizoot.*, **14**: 655-666.

26. OIE, 1996. International disease statistics. *Bull. Off. int. Epizoot.*, **108**: 651-712.

27. OIE, 1997. International disease statistics. *Bull. Off. int. Epizoot.*, **109**: 95-117.

28. OIE, 2000. World animal health in 1999. Part 1: reports on the animal health status and disease control methods and tables on incidence of list A diseases. Paris, France, OIE, 339 p.

29. OIE, 2000. African swine fever in Mozambique: south of the Save River. *Dis. Inf. Off. int. Epizoot.*, **13**: 65-67.

30. OIE, 2001. African swine fever in Senegal. *Dis. Inf. Off. int. Epizoot.*, **14**: 25-30.

31. OIE, 2001. African swine fever in the Democratic Republic of Congo. *Dis. Inf. Off. int. Epizoot.*, **14**: 81-88.

32. OIE, 2001. African swine fever in the Democratic Republic of Congo. *Dis. Inf. Off. int. Epizoot.*, **14**: 93-100.

33. OIE, 2001. African swine fever in Benin. *Dis. Inf. Off. int. Epizoot.*, **14**: 123-129.

34. OIE, 2001. African swine fever in Togo. *Dis. Inf. Off. int. Epizoot.*, **14**: 143-148.

35. OIE, 2001. African swine fever in South Africa: in the African swine fever controlled area. *Dis. Inf. Off. int. Epizoot.*, **14**: 191-192.

36. OIE, 2001. African swine fever in Zambia. *Dis. Inf. Off. int. Epizoot.*, **14**: 213-221.

37. O.I.E, 2001. African swine fever in Kenya. *Dis. Inf. Off. int. Epizoot.*, **14**: 229-232.

38. PAN I.C., DE BOER C.J., HEUSCHELE W.F., 1970. Hypergammaglobulinemia in swine infected with African swine fever virus. *Proc. Soc. exp. Biol. Med.*, **134**: 376-371.

39. PAN I.C., DE BOER C.J., HESS W.R., 1972. African swine fever: application of immunoelectroosmophoresis for the detection of antibody. *Can. J. comp. Med.,* **36**: 309-316.

40. PAN I.C., MOULTON J., HESS W.R., DE BOER C.J., TESSLER J., 1973. African swine fever. Immunologically-mediated necrotizing pneumonia. *Fed. Proc.,* **32**: 1038 (cited by McDaniel, 1986).

41. PASTOR M.J., LAVIADA M.D., SANCHEZ-VISCAINO J.M., ESCRIBANO J.M., 1989. Determination of African swine fever virus antibodies by immunoblotting assay. *Can. J. vet. Res.*, **53**: 105-107.

42. PENRITH M.-L., 1998. An introduction and overview of African swine fever (ASF) in Africa. Rome, Italy, FAO, p. 1-4.

43. PEREZ J., FERNANDEZ A.I., SIERRA M.A., HERRAEZ P., FERNANDEZ A., DE LAS MULAS J.M., 1998. Serological and immunohistochemical study of African swine fever in wild boar in Spain. *Vet. Rec.*, **143**: 136-139.

44. PLOWRIGHT W., 1977. Panel discussion on the eradication of African swine fever in Europe. In: Proc. Agric. research semin. Hog cholera/classical swine fever and African swine fever, Tiererztliche Hochschule, Hanova, Germany, 6-11 Sept. 1976. Luxembourg, Directorate-General XIII, EUR 5904, p. 772-781.

45. PLOWRIGHT W., PERRY C.T., GREIG A., 1974. Sexual transmission of African swine fever in the tick, *Ornithodoros moubata porcinus* Walton. *Res. vet. Sci.,* **17**: 106-113.

46. PLOWRIGHT W., THOMSON G.R., NESER J.A., 1994. African swine fever. In: Coetzer J., Thomson G., Tustin R. Eds, Infectious diseases of livestock. Oxford, UK, Oxford University Press, p. 568-599.

47. POLO JOVER F., SANCHEZ BOTIJA C., 1961. Informe sobre la peste porcina africana en España. Enero-abril 1961 (evolución, diagnóstico y profilaxis). *Bull. Off. int. Epizoot.,* **56**: 388.

48. REICHARD R.E., 1978. African swine fever in the Americas. In: Proc. 82nd annual meeting US Animal Health Association, p. 226-231.

49. RENNIEL L., WILKINSON P.J., MELLOR P.S., 2001. Transovarial transmission of African swine fever virus in the argasid tick *Ornithodoros moubata*. *Med. vet. Entomol.,* **15**: 14d-14b.

50. RIBEIRO J.M., AZEVEDO R.J., TEIXEIRO M.T.O., BRACO FORTE M.C., RODRIGUES R., RIBEIRO A.M., OLIVEIRO E., NORONHA F., GRAVE PEREIRA C., DIAS VIGARIO J., 1958. Peste porcine provoquée par une souche différente (Souchel) de la souche classique. *Bull. Off. int. Epizoot.,* **50**: 516.

51. ROLLE M., MAYR A., 1993. Mikrobiologie, Infektions und Seuchenlehre. Stuttgart, Deutschland, Aufl, Enke, 6 p.

52. SEIFERT H.S.H., 1996. Tropical animal health. Dordrecht, the Netherlands, Kluwer Academic Publishers, 548 p.

53. STEYN D.G., 1928. Preliminary report on a South African virus disease amongst pigs, 13th and 14th reports. Onderstepoort, South Africa, Division of Veterinary Education and Research, p. 415.

54. TUBIASH H.S., 1963. Quantity production of leukocyte cultures for use in haemadsorption tests with African swine fever virus. *Am. J. vet. Res.,* **24**: 381-384.

55. VELHO E.L., 1956. African swine fever in Angola. *Bull. Off. int. Epizoot.*, **46**: 335.

56. VELHO E.L., 1957. La peste porcine africaine. *Bull. Off. int. Epizoot.*, **48**: 395-402.

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Résumé

Ayoade G.O., Adeyemi I.G. Exposé général sur la peste porcine africaine

La peste porcine africaine (PPA) est une maladie virale très grave en extension et menaçant le développement des productions porcines dans le monde entier. Le virus est un déoxyribovirus cytoplasmique de symétrie icosahédrique de la famille des *Asfarviridae*. C'est une arbovirus (transmis par les tiques) et c'est le seul arbovirus contenant de l'ADN. Les suidés sauvages d'Afrique, principalement le phacochère et le potamochère, sont les vertébrés à l'origine de la PPA. Les porcs domestiques sont infectés par les tiques (*Ornithodoros moubata)* ou par contacts directs avec des porcs sauvages. Des transmissions transstadiales et transovariennes ont lieu chez les tiques. Récemment, la PPA a causé une morbidité et une mortalité élevées chez les porcs domestiques au Nigeria et dans d'autres pays de l'Afrique de l'Ouest, considérés auparavant comme étant indemnes de la maladie. Aucune voie de protection vaccinale n'a été mise au point à ce jour. La protection réside dans des mesures sanitaires avec abattage et des mesures d'accompagnement, quarantaine stricte des porcs et des produits issus des élevages aux frontières, moyens nécessaires pour limiter les foyers de PPA en Afrique, notamment en Afrique de l'Ouest. Une meilleure connaissance de la maladie, la cuisson des eaux grasses dans l'alimentation des porcs, ainsi que des mesures de contrôle des porcs en divagation sont devenues les priorités dans l'éradication de la PPA. Les recherches sur un vaccin doivent être intensifiées. Les services gouvernementaux doivent améliorer les voies de diagnostic de terrain et de laboratoire, ainsi que les méthodes et réseaux d'informations, la PPA étant une maladie à déclaration obligatoire.

Mots-clés : Porcin – Virus peste porcine – Epizootologie – Diagnostic – Surveillance – Méthode de lutte – Contrôle de maladies – Afrique.

Resumen

Ayoade G.O., Adeyemi I.G. La peste porcina africana: una síntesis

La peste porcina africana (PPA) es una devastadora enfermedad viral en expansión, que amenaza actualmente la industria porcina mundial. El virus es un desoxiribovirus citoplásmico icosaedral (ICDV), perteneciente a la familia *Asfarviridae*. Es un arbovirus (transmitido por garrapatas) y es el único arbovirus que contiene ADN. Los suinos salvajes de Africa, principalmente el jabalí y el cerdo de bosque, son los huéspedes vertebrados originales de PPA. Los cerdos domésticos son infectados por garrapatas (*Ornithodoros moubata*) o por contacto directo con suinos salvajes. Las infecciones trans estadio y trans ovariana pueden presentarse en garrapatas. Recientemente, PPA provocó una alta morbilidad y mortalidad en los cerdos domésticos en Nigeria y otros países de Africa del oeste, previamente conocidos como libres de la enfermedad. No se ha descubierto una vacuna protectora, por lo tanto, la política de sacrificio con compensación adecuada, cuarentena estricta de los cerdos y sus productos en las fronteras son necesarias para parar las epidemias de PPA en curso en Africa, particularmente en Africa del oeste. Para erradicar la PPA es ahora urgente la toma de conciencia de PPA, cocinar bien la carne de cerdo antes de servirla y reforzar la regulaciones contra los cerdos deambulantes. La investigación para la producción de una vacuna debe intensificarse. Los gobiernos deben mejorar la clínica veterinaria ambulatoria y los servicios de diagnóstico, así como la red de información, ya que PPA es una enfermedad notable.

Palabras clave: Cerdo – Virus peste porcina – Epizootología – Diagnóstico – Vigilancia – Método de control – Control de enfermedades – Africa.