African Swine Fever: An Overview

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African Swine Fever (AFS) is a devastating viral disease affecting the reticuloendothelial system (RES) of swine. Should ASF be introduced into the United States, it would severely threaten the 9 billion dollar[35] swine industry. Pork producers would lose vast amounts of money and consumers would be faced with higher costs at the meat counter.

ASF is caused by a DNA virus[17,33] of the Iridoviridae[18] virus family. It is highly resistant to inactivation by many disinfectants and environmental factors. The virus may remain viable in soil, blood, bone marrow, and cured or chilled pork products for several months[17,18]. The virus survives in a wide pH range with a notable persistence in highly alkaline environments.[14] This ability to persist is important when one considers transmission of the virus and disinfection of infected premises.

ASF was first reported in East Africa in 1921 by Montgomery[4] who initially thought it was a hyperacute form of hog cholera. Historically ASF was always a peracute, highly fatal, highly contagious disease of swine, but in recent years several antigenic strains of lower virulence have been found.[3,34] Mortality varies from near 100% with the most virulent to less than 20% with the least virulent strains. Lower virulence strains pose the greatest threat to ASF-free countries because they may go undiagnosed for some time and become widespread before they are detected.[20] The greatest economic loss would not be due to acute deaths, but to the poor growth of surviving pigs who also serve as sources of infection for other pigs.

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Incidence & Prevalence

ASF is endemic in parts of Africa where warthogs, bushpigs, giant forest hogs, and *Ornithodoros moubata* ticks act as virus reservoirs.[3,18] The first report of ASF outside of Africa occurred in Portugal in 1957.[3] Subsequent outbreaks have been reported in Spain (1960), France (3 times between 1964-1974), Sardinia, Malta, the Dominican Republic, Brazil (all 1978), Haiti (1979) and Cuba (1971 & 1980).[2,3,17,21,42] The outbreaks in 1978 followed an increased incidence of ASF in Spain and Portugal.[17] The disease was eradicated from France, Italy, Cuba, Malta, Sardinia and the Dominican Republic by slaughter measures.[3,2,9,42] In Malta and the Dominican Republic, the entire swine populations were slaughtered in the eradication efforts. Portugal, Spain, Brazil, and Haiti are continuing eradication efforts.

The source of the outbreaks in the Western Hemisphere is believed to be viruses in uncooked garbage from airliners or seaports which was fed to swine in the countries where the outbreaks occurred.

The virus entering Europe in 1957 was quite capable of persisting in European pigs[3] and was the first indication that lower virulence strains did exist or were emerging. As a result, the disease changed from epizootic to nature in Spain and Portugal. The reason more outbreaks have not occurred in France may be due to the Pyrenees mountain range which forms a natural barrier between France and Spain.

ASF has never been reported in North America, Asia, and Australia.[18,27]

Transmission:

African Swine Fever virus (ASFV) spreads among domestic pigs by direct contact with infected swine, or indirectly by infected ticks

of the genus *Ornithodoros*[3,17,18,41], aerosols[41], infected pens[3], contaminated feed and water[3,18] (including uncooked garbage containing infected swine material), personnel, and contaminated equipment.[18]

Once ASF is established in domestic swine, it usually spreads rapidly due to the short incubation period (5-15 days) and direct transmission.[3] The rate of spread of the disease through a herd is directly proportional to the virulence of the particular strain.[18] The virus is present in high titers in nasopharyngeal secretions at the onset of clinical signs, and with contact transmission the main route of viral invasion is considered to be the upper respiratory tract. Invasion through the lower respiratory and alimentary tracts is also possible.[3,41]

ASFV is an arbovirus[10]; therefore a major factor in persistence in an area is its ability to survive in arthropods.[10] In Africa, transmission to domestic pigs is primarily by *Ornithodoros moubata*[3,17,18], an argasid tick. It appears that warthogs are the source for the ticks, which in turn, infect domestic swine[18]. Other work has shown that viremias in warthogs are usually too low to infect ticks[26], in which case maintenance within the tick population becomes more important. Sexual (via seminal fluid) and transovarial tick-to-tick transmission as well as transstadial maintenance have been shown to occur.[13,18,26] Some ticks retain the virus and are able to transmit it to swine for at least 8 years.[18] In Spain and Portuga,[1] *O. erraticus* is the species which has become the viral reservoir.[17]

Research has been done attempting to ascertain whether external parasites indigenous to the United States are able to transmit and maintain ASFV.[10] *Amblyoma* spp., ixodid ticks which were studied because they are common livestock pests, ingest large amounts of blood, but maintain the virus only a short time, therefore no transmission to swine was observed. *Ornithodoros coriaceus* ticks maintain the virus transstadially and transmit the virus to swine experimentally. Eight other *Ornithodoros* spp. parasitic to mammals exist in the US, therefore a number of potential ASFV vectors may be present in this country. *Hematopinus suis,* the hog louse, harbors the virus, but does not transmit it experimentally. However, the fact that it is harbored would lead one to suspect that the virus could potentially be transmitted.

Airborne transmission of ASFV has been demonstrated over distances up to 2.3 meters,[41] so this type of transmission may occur only in intensive swine-raising facilities where swine are closely confined. Pig movement and splashing of excreta are ways in which infective aerosols are generated.

Swine which survive the initial infection period remain infected, and they or the pork products produced from them are sources of virus for other swine.

Clinical Description

With virulent strains of ASFV, often the first indication the disease is present is finding a pig dead.[18] More commonly a high fever (105°F)[18] appears abruptly following the short incubation period and persists for about 4 days. Pigs are usually depressed, weak, and have reduced appetites[18], although some pigs reportedly continue to eat and drink and remain quite alert until they are near death.[17] Extreme weakness of the hindquarters with difficulty in walking is an early and characteristic sign.[3] Coordination is retained in the front legs and affected pigs may walk on them, dragging themselves around. Hyperemia and cyanotic blotching of the skin over the snout, ears, hindquarters, fetlocks, and under the belly is common.[17] Pigs may huddle, but not as severely as they do with hog cholera.[17] The respiratory rate increases along with the temperature and respiration appears to become painful after 2-4 days. Coughing and dyspnea are common.[17,18] Rapid pulse rates and serous to mucopurulent ocular and nasal discharges are seen.[3] Diarrhea, dysentery, and vomiting occur in some cases. The diarrhea initially is mucoid, but often becomes bloody. Once bloody diarrhea is observed, the prognosis is poor.[18] Ninety percent of pregnant sows will abort regardless of the stage of gestation.[17] Petechial and ecchymotic hemorrhages may be seen on membranes and skin of the aborted fetuses.[18]

Virulent strains of ASFV usually cause death within a day or two after the appearance of obvious clinical signs and are often preceded by convulsions.[3].

Temperatures of pigs surviving low virulence strains return to normal in 2-3 weeks,[22] but are usually intermittently febrile due to being chronically infected. Chronically infected pigs become emaciated, develop soft edematous swellings over limb joints and

under the mandible, and often have a chronic cough.[3,18]

No clinical signs are seen in the reservoir species, but they remain carriers for life.

Clinical pathology studies show a 40-50% decrease in the total WBC count by the fourth day of fever,[3] but it usually returns to normal levels a few days later if the pigs survive.[22] A pronounced lymphopenia is the primary cause of the leucopenia.[3,40] Controversy exists over whether B cell or T cell lymphocyte numbers are reduced the most.[28,39] The reason for this discrepancy is not known, but possibly different strains of the virus have varying effects on different lymphocyte populations. Despite this quantitative reduction of lymphocytes, the response to mitogens is unaffected in the remaining lymphocytes.[39] It is unusual that these responses apparently remain unaffected with a virus which affect the RES.

Despite the severe lymphopenia, the immune system of chronically infected pigs apparently remains intact.[24] Proof of an intact humoral immune system comes from the fact that concurrent inoculation with ASFV and Foot-and-mouth disease virus results in antibody production against FMDV.[39] An intact cell-mediated immune system is demonstrated by the development of delayed hypersensitivity to Mycobacterial antigen (a CMI response) in ASFV-infected pigs.[30]

A neutrophilia occurs due to an increase in juvenile neutrophils, indicating a severe inflammatory reaction.[3,40] Neutrophil function also apparently remains normal.[28]

In pigs surviving more than 6-7 days, serum antibody levels drop, but later rise again. This may be due to IgM production initially with the second rise due to IgG production.[38] Chronically infected pigs show a hypergammaglobulinemia.[18] There appears to be a high degree of similarity in the clinical pictures seen with the various strains of ASF, the differences being a matter of degree of signs, with the most virulent strains causing the most marked signs.[18]

Pathogenesis

The pathogenesis of ASF is complex and is not yet fully understood.

Following contact infection, the main route of viral invasion is considered to be the upper respiratory tract.[3,41] The virus replicates in the regional lymph nodes prior to generalized viremia which occurs 48-72 hours post-infection.[3] Pigs also begin shedding the virus at this time.

Following injection by ticks, the virus quickly infects macrophages and reticular cells of the lymphatic tissues, multiplying to high titers in these cells before they undergo lysis.[18]

After 1-3 days of infection, high virus titers exist in tissues containing a large RE component, such as the tonsils, lymph nodes, spleen, liver, and bone marrow.[4] The most visibly affected cells are the vascular endothelial cells which results in hemorrhage, serious exudate, infarction, and edema throughout the body.[3]

Swine macrophages derived from blood monocytes are apparently the primary target cells for ASFV replication.[9,13] The virus produces no detectable effect on macrophages from humans, rabbits, guinea pigs, and rats. The reasons for resistance in these species is not known.[6,9]

Pigs show a poor but unique immunological repsonse to ASFV.[40] The response is poorly understood and presents several anomalies, one of which is the failure to demonstrate neutralizing antibody even though complement-fixing, precipitating, and hemagglutination-inhibition antibodies are readily detectable.[39] Apparently pigs are able to produce antibodies against some parts of the virus, but not against the virulence factor. Although here, too, conflicting evidence exists since a parallel is observed between gammaglobulin and antibody levels and the resolution or recurrence of fever, suggesting that some neutralizing antibody must have been formed to stop further viral replication.[6] Also, swine that survive ASF infection or have been infected with attenuated virus often resist challenge with the homologous strains.[4,34]

Available evidence indicates that true recovery from ASF does not occur.[25] Resistance of surviving pigs to subsequent exposure may depend on a persisting infection rather than the persistence of circulating antibodies. This condition of infection-immunity is known as premunition.[7] The virus persists in the macrophage in the long-term carrier state,[37] hence, this may be where the virus takes refuge when and if neutralizing antibodies are present. The reason ASF- immune pigs are unble to inactivate and eliminate the virus is unknown. It has been sug-

gested that the complex nature of the virus may have something to do with the inability of pigs to produce neutralizing antibodies.[4,13]

Delayed hypersensitivity reaction to ASFV antigen is believed to be the pathogenesis of most of the chronic signs of ASF.[39] The chronic penumonia is believed to be due to the formation of insoluble antigen-antibody complexes in the alveoli.[23] A similar mechanism might be responsible for the subcutaneous swellings over leg joints, fibrinous pericarditis, and ulceration of the skin observed in chronically infected pigs.

Interferon apparently does not play a role in preventing the initial infection or maintaining persistence.[37]

The fact that the macrophage is the target cell for ASFV along with the immunological repsonse of chronically infected swine classes ASFV with a series of viruses (lymphocytic choriomeningitis, Aleutian disease, equine infectious anemia, and lactate dehydrogenase virus) which produce persistent infections, preferentially replicate in macrophages, and in chronically infected animals produce non-neutralizing antibodies.[9]

Necropsy Findings

The damage done to the endothelial cells lining the blood vascular system is responsible for most of the leisons observed at necropsy.[8] In general, the severity of the lesions is directly proportional to the amount of damage done to the circulatory system. In acute, severe ASF, lesions are marked, whereas in subacute, chronic or inapparent forms, lesions may be minor to absent.[18]

Ascites, hemoperitoneum, hydrothorax, and hydropericardium are often noted upon opening the carcass.[18,19]

Severe necrosis of lymphoid tissue is a prominent early feature of ASF.[40] Lymph nodes, especially the hepatogastric node,[22] are the most frequent sites for hemorrhage and edema. Most affected nodes are 2-4 times normal size. They may have subcapsular hemorrhages, infarcts, or may be hemorrhagic to the point of resembling hematomas.[18] The spleen, which contains the highest concentration of cells containing viral antigen,[4] is enlarged in 50% of ASF cases and may be up to 4 times its normal size.[17,22] Splenic infarcts seen on the surface are indistinguishable from those seen in hog cholera.[17]

Petechial and ecchymotic hemorrhages are evident on the pleural surfaces of the lung.[18] Interlobular septae are prominent, and the lungs do not collapse when the thorax is opened. Focal blanched lobules are seen on the surface due to internal pressure forcing blood from them. Occasionally fibrous adhesions form between the lungs and thoracic wall.[18]

The pericardial sac may become thickened and opaque. Petechial and ecchymotic hemorrhages occur on the epicardium and endocardium.[18]

Petechial and ecchymotic hemorrhages may occur on the diaphragm.[18]

The liver is congested and swollen with a mottled or diffusely darkened surface.[18]

Edema may be marked in the wall of the gallbladder, and its blood vessels may be severely engorged. It often contains a mixture of blood and bile containing enough fibrin to cause clotting of the mixture.[18,22]

The kidneys are usually congested and hemorrhagic. Petechiae on the serosal and cut surfaces are most common, but they may resemble large hematomas in the sublumbar region. In these cases, a layer of clotted blood about 6mm thick appears to encircle the kidney either inside or outside of the renal capsule.[18] Petechiae are often seen in the urinary bladder with virulent strains.

Since pigs often continue to eat until late in the course of the disease, they usually have ingesta in their stomachs. Some cases show a gastritis manifested by a bright red fundus.

Segments of the small intestine are occasionally hemorrhagic, but it is usually normal except for occasional petechial and eccymotic hemorrhages in the mucosa and around blood vessels. Peyer's patches may be edematous and necrotic.[17] Congestion and petechial to ecchymatic hemorrhages are more common in the large intestine than the small intestine. Ulceration of the large intestine may be seen with the more virulent strains. In 10-20% of cases, the colon contains large amounts of blood mixed with ingesta. These pigs usually have bloody diarrhea before death.[18]

Brain lesions are found in cases caused by highly virulent strains. Lymphoid infiltration, hemorrhage, and degenerative changes are seen in the meninges and blood vessels going into the CNS tissue.[18]

Microscopic lesions, which are most pronounced in the spleen, lymph nodes, lungs,

and liver, consist of pyknosis, karyorrhexis, karyolysis, edema, and hemorrhage.[18]

Lesions of chronic ASF include pleuritis, pericarditis, pneumonia (which may resemble the caseous lesions of tuberculosis), and hyperplasia of the lymph nodes.[4,13,18,22]

Diagnosis

Due to the potentially rapid spread of ASF in a virgin area, accurate and rapid diagnoses must be made on suspected cases. Clinical signs and necropsy findings should lead one to suspect ASF.

A list of differential diagnoses could include hog cholera, salmonellosis, erysipelas, listeriosis, mycoplasmosis, pasteurellosis, pigweed (*Amaranthus retroflexus*) poisoning, and lamb's quarter (*Chenopodium album*) poisoning.[2,18,19] Hog cholera can be ruled out by inoculation of 10 ml of a hog cholera antiserum-treated 10% suspension of spleen, lymph node or whole blood from the suspected case into known ASF-susceptible pigs.[18,20,21] Salmonellosis and acute erysipelas can be ruled out by repeated negative bacterial cultures and by treating the 10% tissue suspension with antibiotics. Listeriosis, mycoplasmosis, and pasteurellosis may all produce some signs similar to ASF, but can be differentiated from ASF at necropsy. The peri-renal edema and hemorrhage caused by pigweed and lamb's quarter poisoning may resemble that caused by ASF, but the absence of other lesions will rule them out.[19]

If ASF cannot absolutely be ruled out, laboratory confirmation is imperative.[18]

By its nature, culture and assay of the virus on primary pig leucocytes or bone marrow culture will always be the most reliable test available, but by the time tissue cultures are prepared, inoculated, and read, the assay takes 7-10 days.[5,37] A number of serological tests have been developed which provide faster results. Originally all ASFV isolates were hemadsorbing, therefore the hemagglutination inhibition (HAI) test was used as the diagnostic test for ASF. Now many non-hemadsorbing strains have appeared and the HAI test is no longer used extensively.[34] Other serological tests that have been developed include immunoelectroosmophoresis (IEOP),[25,38] reverse radial immunodiffusion (RRID)[25,38] radio-immunoassay (RIA),[5,38] agar gel diffusion precipitation (AGDP),[25,31] complement fixation (CF)[32] indirect immunofluorescence (IFI),[25,32] direct immunofluorescence (IFD)[18] and enzyme-linked immunosorbent assay (ELISA).[1,12,36]

The IEOP, RRID, CF, and AGDP tests all have the disadvantage of lower sensitivity than other available tests. However, RRID is a rapid test (18 hours) and lends itself well to field use.[25]

IFD has a low sensitivity in low virulence forms of ASF due to the relatively low number of virus particles.[18]

RIA has the advantage of early antibody detection (3-4 days post-inoculation), sensitivity, speed (5 hours) and large-scale testing.[5,38]

IFI is sensitive and allows visualization of the distribution of fluorescence within cells which lends support to other serological tests.[13]

The recently developed ELISA test, however, appears to be the test of choice for ASF. It is sensitive, specific, rapid (24 hours), inexpensive, feasible with modest equipment, and lends itself well to large-scale testing. The test can be modified to detect classes of immunoglobulins, therefore detection of large amounts of IgM might indicate a recent infection.[11] A disadvantage of the ELISA test is the occurrence of some false positive tests due to the high sensitivity of the test. For this reason, all positive sera should be retested with another fairly specific test such as IFI.[13] False negative tests are not seen with the ELISA test.[11]

The procedure of using the ELISA test followed by IFI is currently being used in the Dominican Republic with excellent results.[11]

Except for the HAI test, all strains of ASFV apparently cross-react extensively in the serologic tests.[5]

Treatment and Control

Since ASF is a viral disease, no effective therapy exists to cure infected pigs. Good husbandry and supportive therapy may lower the mortality rate,[18] but since surviving pigs usually become chronically infected, they do not perform well and serve as possible sources of infection for other pigs.

Numerous unsuccessful attempts have been made to produce vaccines against ASF.[4,6,7,16] Killed vaccines confer almost no protection against virulent virus.[7] Attenuated live virus vaccines may show some transitory protection, but many pigs will show post-vac-

cinal reactions including pneumonia, skin ulcers, pericarditis, and disturbances of locomotion.[16] The attenuated virus may revert to live virulent virus[13] as evidenced in Portugal where 500,000 pigs were vaccinated with an attenuated virus, after which 20% of them died with pulmonary lesions from which the virus was isolated.[6]

Efforts are underway to develop temperature sensitive mutant strains for use in vaccines.[1] Genetic engineering may result in viral antigen free of virulent material. However, the development of a subunit vaccine will be more difficult than for other diseases due to the complex enzyme system of ASFV.[13]

Consequently, the only course of action open at this time is slaughter of all infected and exposed swine followed by disinfection of contaminated premises.[3,8] Lipid solvents are effective against the virus due to the lipid envelope surrounding it.[14] The ortho-phenyl phenol compounds such as One-Stroke Environ[a] appear to be the most effective.[13,17] Other disinfectants which have been used with varying success are 2% sodium hydroxide,[8] iodine compounds,[13] and fluorocarbons.[4] Following disinfection, a three-month waiting period must elapse after which susceptible "sentinel" pigs are brought onto the premises.[29] If these pigs remain healthy for three months, the premises is considered free of the virus.

Attention must also be given to preventing virus transmission to other premises in the area. Adequate, systematic cleaning and disinfecting buildings, equipment, vehicles, and clothing must be done to prevent indirect transmission.[15] Veterinarians, feedmen, and other service personnel who visit many farms must be especially careful.[17] Sewage water must be handled properly to guarantee that it will not be a vehicle of contagion to other area swine farms.[15] Destruction of parasites which could potentially transmit or be a reservoir of the virus must be considered. Garbage should not be fed to swine or should be thoroughly cooked to inactivate the virus before it is fed.

Immediate and exact reporting of suspected cases of ASF is essential. Swine owner participation will be enhanced by educating them about the disease and by rapid and fair indemnification for slaughtered animals.[15,21]

The major impediments to controlling ASF are: 1) the lack of a safe and effective vaccine, 2) the emergence of virus strains that produce subacute, chornic, and subclinical infections, and 3) the possible establishment of virus reservoirs in tick populations.[1]

Conclusions

The recent influx of refugees from Cuba and Haiti along with the active Caribbean tourist trade increase the threat of the introduction of ASF into the United States. Fishermen from Caribbean islands are known to carry live pigs and pork products on board. Large amounts of pork products have been confiscated from legal and illegal immigrants and destroyed.[19]

All necessary and possible steps to prevent the entrance of ASF into the US must be taken, because a low virulence form could become widespread before it is diagnosed. The estimated cost of eradicating an outbreak, depending on the size, is between 7.5 million and 558.6 million dollars.[18] If eradication efforts failed, the cost to US consumers would be about 2 billion dollars the first year.[18] The fact that ASF has not become established in more continental European countries is evidence that it can be controlled and eradicated.

The first line of defense against ASF lies at the US borders, and consists of maintaining strict surveillence at seaports, airports, and land border crossings.[29] All pork products (except government imports) and pork-containing garbage should be confiscated and destroyed or thoroughly cooked before being fed to swine. The Office International des Epizooties (OIE) has published a set of zoosanitary rules recommended for international trade of live swine and pork products to reduce the chance of international spread of ASF.[21]

The secondary line of defense lies in other countries of the Western Hemisphere.[29] If ASF can be eradicated from nearby countries, it will lessen the chance of the disease entering the US. For this reason, the US declared states of emergency disease situations in Haiti and the Dominican Republic to allow the US to aid in the ASF eradication programs.[43]

The US Department of Agriculture has an emergency plan which can be pressed into action immediately should ASF enter the country.[29] Other current USDA work includes studying species of American ticks which might act as ASFV reservoirs, attempts to grow

ASFV in cell lines of mosquitoes and ticks, and further work on the immunobiology of ASFV.[1]

Given the speed and ease of travel in today's world, the probability that ASF will be introduced into the US is very high. We cannot talk in terms of "if" ASF arrives, but "when" it arrives, and we must be ready when it does.

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[a]Available from Vestal Laboratories, St. Louis, Mo.