

## AFRICAN SWINE FEVER

*(Peste porcine Africaine, fiebre porcina Africana, maladie de Montgomery)*

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**Definition** [top](#)

African swine fever (ASF) is a tickborne and contagious, febrile, systemic viral disease of swine .

## Etiology [top](#)

The ASF virus is a large (about 200 nm) lipoprotein-enveloped, icosahedral, double-stranded DNA virus. For many years the agent was classified as an iridovirus (3), but in recent years it was found to have many characteristics of poxvirus; thus, researchers have suggested establishment of a new family for ASF virus (ASFV) (19).

This virus is quite stable and will survive over a wide range of pH. In serum-free medium, ASFV is inactivated at pH 3.9 or lower and at pH 11.5 or higher. In the presence of 25 percent serum, ASFV will remain viable for 7 days at pH 13.4 (17). The virus will survive for 15 weeks in putrefied blood, 3 hours at 50° C, 70 days in blood on wooden boards, 11 days in feces held at room temperature, 18 months in pig blood held at 4° C, 150 days in boned meat held at 39° F, and 140 days in salted dried hams (8A).

Over the years, ASFV isolates with lower virulence have emerged — particularly in the Iberian peninsula. Virulence of isolates varies from highly virulent (essentially 10 percent mortality in 7-10 days after exposure), to moderately virulent (acute illness in which a high percentage of the pigs survive) , to low virulence (only seroconversion occurs).

## Host Range [top](#)

Initially, domestic and wild pigs (Africa: warthog, bush pig, and giant forest hog; Europe: feral pig) were thought to be the only hosts of ASFV (1,16). In 1963, Spanish workers isolated ASFV from the soft tick *Ornithodoros erraticus* collected from ASF-infected farms (13). Subsequently, researchers showed that ASFV replicates in the tick and that there is transstadial, transovarial, and sexual transmission in *Ornithodoros* ticks. *O. moubata* collected from warthog burrows in Africa were shown to be infected with ASFV (5). African swine fever in wild pigs in Africa is now believed to cycle between soft ticks living in warthog burrows and newborn warthogs (18). *Ornithodoros* ticks collected from Haiti, the Dominican Republic, and southern California have been shown to be capable vectors of ASFV (4,5), but in contrast to the African ticks, many of the ticks from California died after being infected with ASFV. Many researchers believe that ASFV is really a tick virus and the pig is an accidental host (11).

Because ASFV-infected ticks can infect pigs, ASFV is the only DNA virus that can qualify as an arbovirus.

## Geographic Distribution [top](#)

African swine fever is present in several African countries and on the island of Sardinia.

## Transmission [top](#)

Even though the soft tick has been shown to be a vector (and in Africa probably the reservoir of ASFV), the primary method of spread from country to country has been through the feeding of uncooked garbage containing ASFV-infected pork scraps to pigs. Once a pig becomes infected, ASFV spreads by direct contact, and contaminated people, equipment, vehicles, and feed. The role of carrier pigs has been difficult to prove experimentally, but circumstantial evidence from the field incriminates carrier pigs. An outbreak of ASF in a contained swine operation in Africa was traced to workers feeding the entrails of guinea fowl to pigs. It was shown that the guinea fowl feed on soft ticks; thus, ASFV was present in the guinea fowl intestines fed to the pigs.

The amount of ASFV needed to infect a pig depends on the route of exposure. Experimentally, a pig can be infected by intramuscular or intravenous inoculation with a 0.13 hemadsorbing dose ( $HAD_{50}$ ); intranasal-oral inoculation required 18,200  $HAD_{50}$ .

In an ASF endemic area where there are soft ticks, ticks can be the source of infection. However, in these areas in Africa, pigs can be very successfully raised in confinement with double fencing, proper isolation, and sanitary procedures. In Africa, the production system with the highest risk of ASF is the village pig, for these pigs roam. The owners do not practice isolation procedures when the pigs are confined.

In other areas, the disease has to be introduced by infected live pigs or by feeding uncooked garbage containing ASFV-infected pork. Once the disease is introduced into a herd, it spreads by direct and indirect contact with secretions and excretions from infected pigs. Aerosol transmission is not important in the spread of ASF. Because ASFV does not replicate in epithelial cells, the amount of virus shed by an ASF-infected pig is much less than the amount of virus shed by a hog-cholera-infected pig. The blood of a recently infected pig contains a very high ASFV titer:  $10^{5.3}$  to  $10^{9.3}$   $HAD_{50}$  per milliliter (7). Therefore, if pigs fight, an infected pig develops bloody diarrhea, or an infected pig is necropsied, blood is shed, and there is massive environmental contamination.

Piglets born of ASF-convalescent dams are free of ASFV and ASF antibody at birth but seroconvert after ingesting colostrum (14,15). When piglets from noninfected (control) and ASF-convalescent dams were challenge-inoculated when 7 weeks old, the control piglets developed an average viremia of  $10^{7.6}$  and died, whereas the piglets from convalescent gilts developed an average viremia of  $10^{4.9}$  and survived. However, because of persistent infection by ASFV, reestablishing a herd using pigs from convalescent animals will not result in an ASFV-free herd. When farmers in Cameroon repopulated their herds using ASF-convalescent animals, the herds experienced recurring periods of high mortality due to ASF.

### **Incubation Period** [top](#)

After intranasal-oral exposure, pigs usually develop fever and leukopenia in 48 to 72 hours.

### **Clinical Signs** [top](#)

#### **Highly and Moderately Virulent ASF Isolates** [top](#)

The clinical signs of ASF are influenced by the virulence of the virus and the physiological state (age and pregnancy) of the pig. After inoculation of feeder pigs with either a highly virulent or moderately virulent isolate, the clinical course for both isolates is similar for the first 4-6 days post infection. About 2 DPI, the pigs will develop a fever of 105-107° F (40.5-41.7° C) and white pigs will have a reddened skin, moderate anorexia, and leukopenia. When disturbed the pigs will get up and move about but if left alone will after a short time lie down.

After 4-6 DPI, a difference between the pigs inoculated with the different isolates will become apparent.

#### **Highly Virulent Isolate** [top](#)

The pigs become progressively sicker (eat and move less), and most die between 7 and 10 DPI. It is not unusual to see a pig walking and a short time later to find it dead.

#### **Moderately Virulent Isolate** [top](#)

Pigs infected by moderately virulent ASFV usually have a high fever for 10-12 DPI. Some mortality usually occurs at this time. After 12-14 DPI, temperatures and leukocyte counts start to return to normal levels. It is not unusual to have one or

more pigs die as early as 7-8 DPI, but when these pigs are necropsied, the cause of death is frequently hemorrhage into the stomach; the underlying mechanism of death was that ASFV infection caused a thrombocytopenia, resulting in a prolonged bleeding time and hemorrhage from a preexisting gastric ulcer (2). Very young pigs may have a high mortality and have lesions similar to infection by highly virulent virus.

Pigs affected with either isolate, in addition to the reddened skin, may develop dark red to purple discoloration of the skin on the ears (Fig. 13), tail, extremities of the legs, or skin on the hams. This is a nonspecific sign also seen in other diseases. Some groups of pigs will develop diarrhea; this is probably due to disturbed gut physiology and flora rather than a direct effect of the virus because the virus does not replicate in epithelium. In contrast to hog cholera, ASFV-infected pigs do not develop a conjunctivitis or encephalitis, and, despite the high fever, the ASFV-infected pigs stay in good condition, whereas hog cholera-infected pigs quickly lose much weight.

Pregnant animals infected with a high-, moderate-, or low-virulence ASF isolate abort.

### **Low Virulence Isolates** [top](#)

Nonpregnant animals infected by certain low-virulence ASFV may only seroconvert; pregnant animals will abort.

Other low-virulence ASFV isolates will cause a low fever for 2-3 weeks and then reddened areas 1 cm<sup>2</sup> to many centimeters in size may develop in the skin. These areas then become raised and necrotic. These pigs may also have painless enlargements of joints—particularly the carpal and tarsal joints. This form is referred to as chronic ASF (10). Many of these pigs will have recurring episodes of a more acute disease and eventually die during an acute episode.

### **Gross Lesions** [top](#)

### **Highly Virulent ASFV Infection** [top](#)

Pigs that die peracutely from an infection with a highly virulent ASFV may have poorly developed lesions. Animals that die 7 or more DPI have more classic lesions. Three lesions most consistently found and highly suggestive of ASF infection are as follows:

Greatly enlarged dark red to black friable spleen (Fig. 14)

Very enlarged hemorrhagic gastrohepatic lymph nodes (Fig. 15)  
 Very enlarged hemorrhagic renal lymph nodes (Fig. 16).

Other lesions described for ASF are more variable and are as follows:

Dark red to purple areas of skin on ears, feet, and tail  
 Petechial hemorrhages on serosal surfaces (Fig. 17)  
 Petechial to ecchymotic hemorrhages in the renal cortex  
 Perirenal edema  
 Edema of the gall bladder (Fig. 18)  
 Swollen liver  
 Edema of the lung.

In pigs infected orally, the submandibular lymph node may be enlarged and have some hemorrhage. Other peripheral lymph nodes may have only edema.

### **Moderately Virulent Virus [top](#)**

The gross lesions 8-12 DPI in pigs infected with a moderately virulent ASFV are similar to those infected by a highly virulent ASFV. The main difference in the lesions between these two types of isolates is that in infections by a moderately virulent ASFV, the spleen although enlarged, has a more normal color and is not friable.

### **Low Virulent Virus [top](#)**

The most common lesions in chronic ASF are necrotic skin lesions (Fig. 21, 22), consolidated lobules in the lung (Fig. 19), generalized lymphadenopathy (Fig. [20](#)), swollen joints, and pericarditis.

Aborted fetuses may be anasarcaous, and there may be petechial hemorrhages in the placenta, skin, and myocardium, and a mottled liver.

### **Morbidity and Mortality [top](#)**

The warthog and bush pig develop a viremia but have a very mild or subclinical disease, whereas ASF infection in domestic pigs and European feral pigs can cause a high mortality.

Morbidity in a previously unexposed herd will usually be 100 percent in pigs that have contact with each other. Mortality varies with the virulence of the isolate.

Highly virulent isolates will cause about a 100 percent mortality. Infection by lesser virulent isolates can cause mortality that varies from a low percentage to 60-70 percent . Factors that can increase mortality in infections by the lesser virulent isolates are concurrent disease, a young age, and pregnancy.

## Diagnosis [top](#)

### Field Diagnosis

The highly virulent form of ASF will be easiest to diagnose because essentially 100 percent of the pigs will die. African swine fever caused by the lesser virulent isolates will be more difficult to diagnose but should always be suspected when there are febrile pigs and necropsy findings include the following:

- Greatly enlarged dark red to black spleen
- Very enlarged hemorrhagic gastrohepatic lymph nodes
- Very enlarged hemorrhagic renal lymph nodes.

African swine fever has frequently been misdiagnosed as hog cholera. In contrast to hog cholera, ASFV-infected pigs do not develop a conjunctivitis or encephalitis, and despite the high fever, the ASFV-infected pigs stay in good condition. In contrast, hog cholera-infected pigs are severely depressed and quickly lose much weight; moreover, they usually have a foul smelling diarrhea.

## Specimens for the Laboratory [top](#)

The ASFV is present in the blood starting about 2 DPI. In infections by lesser virulent isolates, ASFV can usually be isolated from the blood for 25 or more DPI. Specimens for laboratory diagnosis are as follows:

- Heparinized blood
- Clotted blood or serum
- Submandibular lymph node
- Inguinal lymph node
- Tonsil
- Spleen
- Gastrohepatic lymph node
- Lung
- Liver
- Kidney.

Bone marrow should be submitted if there are considerable postmortem changes.

The specimens should be shipped refrigerated or frozen. Pieces of the preceding tissues, the brain, and any other gross lesion should be submitted in 10 percent buffered formalin.

Aborted fetuses are usually free of virus; therefore, it is necessary to submit a blood sample from the dam.

### **Laboratory Diagnosis** [top](#)

The initial diagnosis of ASF in a free area requires isolation and identification of the virus. After the initial diagnosis, confirmation of a diagnosis can be made by demonstrating ASF antigen in tissue or ASF antibody.

### **Differential Diagnosis** [top](#)

Differential diagnoses for ASF should include hog cholera, erysipelas, salmonellosis, and eperythrozoonosis.

### **Vaccination** [top](#)

There is no vaccine.

### **Control and Eradication** [top](#)

#### **Prevention** [top](#)

Introduction of the disease into free areas can be prevented by cooking all garbage fed to pigs (this applies to commercial and backyard pigs and pets [potbellied pigs]) and importing only ASF-disease free pigs.

#### **Eradication** [top](#)

Control and eradication of ASF in developed countries can be accomplished by slaughter and disposal of all acutely infected pigs, widespread testing and elimination of all seropositive animals, and good herd isolation and sanitary practices.

Today (1996), ASF is not as great a threat to the United States as it was several years ago. The major pork-exporting countries have eradicated the disease in domestic pigs.

**Public Health** [top](#)

Human beings are not susceptible to ASFV infection.

**GUIDE TO THE LITERATURE** [top](#)

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