AFRICAN SWINE FEVER
STANDARD OPERATING PROCEDURES:
1. OVERVIEW OF ETIOLOGY AND ECOLOGY

FAD PReP
Foreign Animal Disease
Preparedness & Response Plan

United States
Department of Agriculture

DRAFT JUNE 2018
The Foreign Animal Disease Preparedness and Response Plan (FAD PReP) Standard Operating Procedures (SOPs) provide operational guidance for responding to an animal health emergency in the United States.

These draft SOPs are under ongoing review. This document was last updated June 2018. Please send questions or comments to:

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African Swine Fever
Etiology & Ecology Quick Summary

Disease
African swine fever; *Pesti porcine Africaine; Peste porcina Africana; Pestis Africana suum; maladie de Montgomery; warthog disease; Afrikaanse varkpes; Afrikanische Schweinepest.*

Mortality & Morbidity
High morbidity and mortality, often reaching 100 percent mortality for severe forms of the disease.

Susceptible Species
All members of the pig family Suidae and arsagid (soft-bodied) ticks of the *Ornithodoros* species.

Zoonotic Potential?
None.

Reservoir
Wild and feral swine (warthogs and bushpigs) of Africa and *Ornithodoros* species ticks.

Transmission
Direct contact with infective secretions and tissues, primarily through the oronasal route. Indirect contact via fomites. Vector-borne transmission through *Ornithodoros* species ticks.

Persistence in the Environment
Highly stable and temperature resistant (requires heat-inactivation at 56°C/70 minutes or 60°C/20 minutes). Can survive environments where pH levels are between 3.6 and 11.5.

Animal Products and By-Products
Long-lived in blood, feces, and tissues as well as on uncooked pork and pork products.
1.1 Introduction
African swine fever (ASF) is a highly contagious disease of wild and domestic suids with high rates of morbidity and mortality. First described in the 1920s in Kenya, ASF is listed by the World Organization for Animal Health (OIE) as a notifiable disease. At various times throughout the 20th century, ASF has been endemic in Africa, Europe, South America, and the Caribbean. Until the last few years, outbreaks were confined to eastern and southern Africa and Sardinia. However, outbreaks in the Caucasus region and Russia have begun to spread to eastern Europe and pose a great risk for further spread to other European Union countries. ASF does not pose a risk to public health.

1.1.1 Further Information
This document is intended to be an overview, focusing on ASF in domestic swine. Additional resources on ASF, as well as the articles referenced in this standard operating procedure (SOP), are listed in Attachment 1.A and on the APHIS FAD PReP website (http://www.aphis.usda.gov/fadprep).

1.1.2 Goals
As a preparedness goal, APHIS will provide etiology and ecology summaries for ASF, and update the summaries at regular intervals.

As a response goal, the Unified Command and stakeholders will have a common set of etiology and ecology definitions and descriptions, to ensure proper understanding of ASF when establishing or revising goals, objectives, strategies, and procedures.

1.2 Purpose
The purpose of this document is to provide responders and stakeholders with a common understanding of the disease agent.

1.3 Etiology
1.3.1 Name
ASF is also known as Pesti porcine Africaine, Peste porcina Africana, Pestis Africana suum, maladie de Montgomery, warthog disease, Afrikaanse varkpes, and Afrikanische Schweinepest. As its name implies, ASF is a disease endemic in wild and feral African swine.

1.3.2 Virus Characteristics
According to the International Committee on Taxonomy of Viruses,¹ African swine fever virus (ASFV) is categorized as follows:

- Family: Asfarviridae
  - Genus: Asfivirus

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• Genome characteristics: double-stranded deoxyribonucleic acid (dsDNA).

ASFV is unique in that it is the only member of the genus Asfivirus and the only known DNA arthropod-borne virus (arbovirus).

1.3.3 Morphology

ASFV is an enveloped virus, 200 nm in diameter with a linear, double-stranded DNA genome that encodes 160 to 175 genes. It has distinctive morphology, characterized by the dense 80 nm virion core that is composed of the viral genome and an icosahedral capsid, covered by an internal lipoprotein envelope. The outer envelope is derived through the budding process from the cellular membrane of infected cells.2,3

1.4 Ecology

1.4.1 Susceptible Species

All members of the pig family (Suidae) are susceptible to ASFV infection, including

- Domesticated swine (Sus domestica)
- European wild boars (Sus scrofa scrofa)
- Warthogs (Phacochoerus spp.)
- Bush pigs (Potamochoerus porcus)
- Giant forest hogs (Hylochoerus spp.).

Though members of the Suidae family are not native to the Americas, peccaries, or New World pigs (family Tayassuidae), are not believed to become infected.4

1.4.2 Reservoir and Carriers

The method by which ASF remains in circulation in endemic countries varies by geography; in Africa it is maintained by cycling between domestic pigs, wild bushpigs, and warthogs and Ornithodoros species ticks, while in Europe it is typically between domestic pigs and wild boars.5 In sub-Saharan Africa, infected ticks can maintain high titers for many months,6 such that the ticks and warthogs, whose burrows ticks inhabit, provide a sylvatic reservoir. For this reason, scientists have speculated that both wild and domestic swine are “accidental hosts” of ASFV.7

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6 Kleiboeker et al. 2002.
7 Guinat et al., 2016.
1.4.3 Distribution

ASF is endemic in western, eastern, and sub-Saharan Africa, including the island of Madagascar. The sylvatic cycle maintains the virus between warthogs and ticks, which can then transmit it to pigs for several years. In western Africa, free range pig husbandry means that naïve pigs are highly available to the ticks. Outside of Africa, ASF is also endemic in Sardinia, Italy. Since 2005, outbreaks have occurred in the Caucasus (Georgia, Armenia, Azerbaijan), Russia, the Czech Republic, Moldova, Ukraine, Estonia, Latvia, Lithuania, Poland, and Romania. The closest ASF has been to the United States were outbreaks in the Dominican Republic (1978), Haiti (1979) and Cuba (1977–1980).

Figure 1-1. Geographic Distribution of ASF 2007—2017

Source: OIE WAHID 2018.

1.4.4 Introduction and Transmission of ASF

There are three modes of transmission for ASF: direct, indirect, and vector-borne. Direct transmission occurs when infected animals come into contact with healthy animals. Contact with infective saliva, respiratory secretions, urine and feces are effective means of transmission due to the high levels of virus found there. Viral titers and the length of time that swine remain infectious depend on the virulence of the strain causing disease. Direct transmission is the main mode of transmission in suitable habitats for wild pigs; the presence of wild pigs is the most predictive risk estimator of disease spread. Disease can also spread through indirect transmission that occurs via contaminated feed and fomites; this mode of transmission can be particularly important in introducing ASF to disease-free areas. Lastly, argasid ticks (Ornithodoros spp.) serve as a vector for transmission of the disease, passing the virus to swine hosts when taking their blood meal. Infected ticks are also able to transmit ASFV to other ticks (sexual

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transmission),\(^{10}\) to their offspring by passage of the virus to the eggs (transovarial transmission) and from one life cycle stage to another (transstadial transmission). Mechanical transmission via biting flies and mosquitoes has also been mentioned.\(^{11}\)

1.4.5 Incubation Period

The incubation for ASFV varies by route of transmission. For direct contact with ASFV-infected pigs, the incubation period is between 5–21 days. When bitten by an *Ornithodoros* tick, the incubation period can be less than 5 days.\(^{12}\)

1.4.6 Morbidity and Mortality

For all forms of ASF disease (peracute, acute, subacute, and chronic) morbidity rates are very high due to the virus’ extremely contagious nature and the high levels of viral shedding. Mortality is more variable. For the peracute form, infection with a highly virulent strain, mortality can reach 100 percent and occur in the absence of any clinical signs within 7–10 days after exposure. Acute forms of the virus also have mortality rates that approach 100 percent, with death occurring within 6–13 days post-inoculation. The subacute form of ASF is caused by moderately virulent strains and the mortality rate is dependent on the age of the population. Younger pigs have a more severe course of infection with mortality rates ranging between 70 and 80 percent. Older pigs experience mortality rates less than 20 percent. For the chronic form of ASF, where infection is with a moderately or low virulent strain, mortality is typically low.\(^{13}\)

1.4.6.1 Clinical Signs

As with mortality, clinical signs and symptoms vary by virus form (Table 1-1). As previously mentioned, for animals infected with the peracute form of ASF death is often the first indication of disease. Cases infected with the acute form may develop fever (40.5–42°C), anorexia, listlessness, cyanosis, incoordination, increased pulse and respiratory rate, leukopenia and thrombocytopenia (at 48–72 hours), vomiting, diarrhea, and abortion in pregnant sows. Any survivors become carriers for life. Swine infected with subacute forms of ASF present with similar though less intense symptoms as described for the acute form, this includes slight fever, reduced appetite, and depression. Abortion in pregnant sows is also possible and can be an early sign.\(^{14}\)

Cases infected with the chronic form of the virus exhibit weight loss, irregular temperature spikes, respiratory symptoms, necrosis of skin, chronic skin ulcer, arthritis, pericarditis and swelling of the joints. Pigs with chronic ASF will experience recurring episodes of acute disease, which could eventually lead to death.\(^{15,16}\)


\(^{12}\) CFSPH, 2015.


\(^{14}\) CFSPH, 2015

\(^{15}\) USAHA, 2008.

Table 1-1. Clinical Signs and Symptoms Caused by the Different Forms of ASF

<table>
<thead>
<tr>
<th></th>
<th>Peracute</th>
<th>Acute</th>
<th>Subacute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Virulence of strain</td>
<td>High</td>
<td>High</td>
<td>Moderate to low</td>
<td>Low</td>
</tr>
<tr>
<td>Immune status</td>
<td>Death before seroconversion</td>
<td>Many die before seroconversion</td>
<td>Seropositive</td>
<td>Seropositive</td>
</tr>
<tr>
<td>Clinical signs</td>
<td>Often found moribund or dead</td>
<td>Febrile (40.5°C–41.5°C), leukopenia, anorexia, blood in feces, reluctant to move, erythemic skin progressing to cyanosis near death</td>
<td>Variable but typically similar to, though less severe than, acute ASF</td>
<td>Mild fever for 2–3 weeks; pregnant sows may abort; reddened then dark, raised, dry, and necrotic skin lesions, especially over pressure points</td>
</tr>
<tr>
<td>Gross lesions</td>
<td>Death occurs before distinct lesions form</td>
<td>Spleen enlarged (up to 3 times normal), dark and friable; multiple hemorrhages of internal organs, especially kidneys and heart; hemorrhagic lymph nodes; edema of gall bladder and lungs; congestion of meninges and choroid plexus</td>
<td>Lesions are similar but milder than acute ASF; spleen may be 1.5 times normal size; lymph nodes enlarges but only mildly hemorrhagic; few petechial on kidneys</td>
<td>Fibrinous pleuritis, pleural adhesions, caseous pneumonia, hyperplastic lymphoreticular tissues, nonseptic fibrous pericarditis, necrotic skin lesions</td>
</tr>
</tbody>
</table>


Finally, ASF gets its classification as a viral hemorrhagic disease because of the lesions that form with the acute, subacute, and chronic cases. Lesions or hemorrhages occur throughout the body, such as on the lymph nodes, the kidneys, larynx, bladder, colon, and gall bladder.17

### 1.5 Environmental Persistence of ASF

ASF virus is highly stable and temperature resistant and can persist in the environment for a long time. Table 1-2 gives an overview of ASFV susceptibility and resistance characteristics.

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17 Kleiboecker, 2002.
Table 1-2. Resistance of ASF virus to Physical and Chemical Action

<table>
<thead>
<tr>
<th>Action</th>
<th>Resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature</td>
<td>Highly resistant to low temperatures. Heat inactivated by 56°C/70 minutes; 60°C/20 minutes.</td>
</tr>
<tr>
<td>pH</td>
<td>Inactivated by pH &lt;3.9 or &gt;11.5 in serum-free medium. Serum increases the resistance of the virus, e.g. at pH 13.4—resistance lasts up to 21 hours without serum, and 7 days with serum.</td>
</tr>
<tr>
<td>Chemicals/Disinfectants</td>
<td>Susceptible to ether and chloroform. Inactivated by 8/1000 sodium hydroxide (30 minutes), hypochlorites—2.3% chlorine (3 minutes), 3/1000 formalin (30 minutes), 3% ortho-phenylphenol (30 minutes) and iodine compounds.</td>
</tr>
<tr>
<td>Survival</td>
<td>Remains viable for long periods in blood, feces and tissues; especially infected, uncooked or undercooked pork products. Can multiply in vectors (<em>Ornithodoros</em> sp.).</td>
</tr>
</tbody>
</table>


### 1.6 Risk of Introduction to the United States

ASF has never been detected in the United States. Protections such as the Swine Health Protection Act, (which requires that food waste consumed by pigs be heat-treated to remove pathogens such as ASFV) and 9 Code of Federal Regulations 94.8 (which prohibits importation of uncooked pork or pork products from countries with recent outbreaks of ASF or where ASF is endemic), guard against outbreaks in the United States; however, due to travel and the illegal importation of pork and pork products, it remains possible that ASF could enter the United States.

Though the natural argasid host is *O. porcinus*, *Ornithodoros* spp. ticks live throughout the world, and many are believed to be competent vectors of the virus.\(^{18}\) Outbreaks in the Caribbean and South America in the 1970s and 1980s demonstrate that vectors and hosts are available and that the Americas are vulnerable to another ASF incursion. Active surveillance is ongoing in the United States for Classical Swine Fever, a disease clinically indistinguishable from ASF, but there are no active surveillance mechanisms for ASF. There are no licensed vaccines for ASF in the United States.

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\(^{18}\) Kleiboeker, 2002.
Attachment 1.A References and Resources


## Attachment 1.B Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>APHIS</td>
<td>Animal and Plant Health Inspection Service</td>
</tr>
<tr>
<td>ASF</td>
<td>African swine fever</td>
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<tr>
<td>ASFV</td>
<td>African swine fever virus</td>
</tr>
<tr>
<td>CEAH</td>
<td>Centers for Epidemiology and Animal Health</td>
</tr>
<tr>
<td>DNA</td>
<td>deoxyribonucleic acid</td>
</tr>
<tr>
<td>dsDNA</td>
<td>double-stranded deoxyribonucleic acid</td>
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<tr>
<td>FAD PReP</td>
<td>Foreign Animal Disease Preparedness and Response Plan</td>
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<tr>
<td>OIE</td>
<td>World Organization for Animal Health</td>
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<td>SOP</td>
<td>standard operating procedure</td>
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<td>USAHA</td>
<td>United States Animal Health Association</td>
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<tr>
<td>USDA</td>
<td>United States Department of Agriculture</td>
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<tr>
<td>VS</td>
<td>Veterinary Services</td>
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