African Swine Fever Backgrounder

(December 10, 2006)

Causative agent

African Swine Fever (ASF) is caused by a virus that is the only member of the Asfarviridae family. The virus is a double-stranded DNA virus, 200 to 300 nanometers (nm) in diameter, enveloped, and icosahedral in shape. To date, the ASF virus is the only DNA virus known to replicate in an insect vector; therefore, the virus qualifies as an arbovirus. ASF virus is very resistant to pH and temperature changes.

Natural distribution

ASF was first identified in Kenya in 1921. Domestic pigs, European wild boars, and American wild pigs are clinically affected. Warthogs, bush pigs, and giant forest hogs can be infected by the virus without becoming ill. Other species, including humans, appear resistant to infection.

ASF is endemic in sub-Saharan Africa, and outbreaks of the disease have occurred in Brazil, Cuba, the Dominican Republic, Haiti, Italy, Malta, Portugal, and Spain. A severe outbreak, resulting in the death or destruction of almost one-half of the country's pig population, occurred in Cameroon in 1982. Marked losses have also occurred in southern Mozambique, several West African countries, Madagascar, and Kenya since 1994.

The World Organization for Animal Health (OIE) classifies ASF as a notifiable disease because of its potential for rapid spread and substantial impact on the international trade of animals and animal products. In accord with the Public Health Security and Bioterrorism Preparedness Response Act of 2002, USDA has recognized ASF virus as a select agent that could pose a severe threat to animal health and/or animal products in the United States.

Transmission

Although wart hogs are carriers of the ASF virus, they cannot directly infect domestic swine. *Ornithodoros* ticks, however, feed on viremic wart hogs and are capable of transmitting the infection if the tick then feeds on a susceptible domestic pig. In areas without wart hogs, such as Malawi, Zambia, Mozambique, and Angola, the virus may adapt to create a carrier state in domestic pigs.

Following a blood meal from an infected pig, the tick becomes infected with the virus, and the virus replicates (reproduces) in the tick. In addition, the infection can be maintained in the tick as it matures through its life stages (transstadial transmission), and can be passed to the next generation through the eggs (transovarial transmission). Sexual transmission of the virus also occurs in *Ornithodoros* ticks. Tick vectors remain infected for long periods and play an important role in persistence of the virus. When a tick ingests a blood meal from a susceptible pig, the virus can then be transmitted from the tick to the animal.

Once infected, domestic pigs are capable of transmitting ASF to susceptible pigs by direct and indirect contact. The oronasal route is the principle means of infection. Infected pigs develop a high degree of viremia (virus particles circulating in the bloodstream) and virus shedding, and are capable of infecting other pigs for at least 2 days before developing clinical signs of disease. Contaminated equipment, vehicles, clothing, and footwear can mechanically transmit the virus to susceptible animals. Aerosolization does not appear to play a role in the transmission of ASF virus.

The incubation period ranges from 2 to 15 days. If a pig recovers from ASF, it is capable of shedding the virus for long periods; therefore, recovered animals are important potential sources of infection.

Uncooked pork products, uncooked garbage containing infected meat, and the feeding of uncooked waste food from airplanes and ships can also spread the disease. The virus can persist for 140 days in salted, dried hams and for 150 days in boned meat stored at 39°F (4°C).

ASF virus can persist for 70 days in blood on wooden boards, 15 weeks in putrefied blood, 11 days in feces at room temperature, and 18 months in pig blood stored at 39°F [4°C]. The presence of serum increases resistance of the ASF virus to pH variation.

For these reasons, ASF is a potentially devastating disease from production, economic, control, and epidemiologic standpoints.

Clinical Signs

ASF exists in acute, subacute, and chronic forms. The acute form results from infection with a highly virulent ASF virus. Some pigs will die suddenly, without prior clinical signs of disease. Others will develop a high fever (105 to 107.6°F [40.5 to 42°C]), anorexia, dullness, coughing, thick nasal and ocular discharge, and be lethargic. Affected pigs will often huddle together. Pigs develop reddened skin, especially on their ears, snout, abdomen, and lower limbs; this is more noticeable in white-skinned animals. Pigs may develop hind limb weakness, a staggering gait, and convulsions. Vomiting, constipation, mucus-covered feces, or bloody diarrhea may be observed. Affected pigs may also exhibit labored breathing and bleeding under the skin. Abortions, stillbirths, and weak litters are frequent manifestations. Death is often within a few days of onset of clinical signs, but can occur as long as 20 days later.

Infection with a moderately virulent ASF virus results in the subacute form of disease. Clinical signs are similar but less severe. Affected animals are usually ill for 5 to 30 days, and death is observed in 15 to 45 days. Pregnant sows will abort. Very young pigs infected with moderately virulent ASF may develop clinical signs that more closely resemble infection with a virulent strain, and mortality is greater.

The chronic form of ASF results from infection with an ASF virus of low virulence. Observed clinical signs are variable, and include weight loss, irregular temperature fluctuations, respiratory problems, necrosis and sloughing of skin, chronic skin ulcers, and arthritis. Affected animals may develop pericarditis (inflammation of the membrane that encases the heart), lung adhesions, and swelling over the joints. As the name implies, this form has a slower onset, and develops over 2 to 15 months. Pregnant sows will abort, but nonpregnant sows may seroconvert without showing clinical signs of illness.

The acute, febrile, hemorrhagic syndrome associated with ASF closely resembles that of classical swine fever (CSF, or hog cholera), and the two diseases may be indistinguishable in many outbreak situations. Minor differences have been observed, and may facilitate a presumptive diagnosis. Unlike CSF, ASF does not produce conjunctivitis (inflammation of the tissues around the eyes) or encephalitis, and pigs infected with the ASF virus do not appear to lose condition as rapidly as those affected with the CSF virus. Differential diagnoses include erysipelas, salmonellosis, pasteurellosis, and septicemia.

Diagnosis

Definitive diagnosis of ASF requires viral isolation and identification. Appropriate diagnostic samples include uncoagulated blood (in heparin [10 IU/ml] or EDTA [0.5%]) collected during the early portion of the febrile stage, as well as sections of the spleen, kidney, lungs, and lymph nodes obtained at necropsy. Aborted fetuses do not usually harbor the virus, so it is necessary to obtain samples from the sow.

Serologic tests available for diagnosis of ASF include enzyme-linked immunosorbent assay (ELISA), complement fixation (CF), and indirect immunofluorescence. Immunoblotting is considered a confirmatory test, and counter immunoelectrophoresis is used for screening groups of animals. A polymerase chain reaction (PCR) assay can detect the presence of virus in tissues.

Specimens for serologic examination or virus isolation should be shipped refrigerated (4°C [39°F]). Organ sections for pathologic evaluation should be submitted in neutral-buffered 10% formalin.

Treatment

African swine fever is a reportable disease. State or federal animal health officials should be immediately notified when ASF is observed or suspected. There is no treatment for AHS.

Morbidity and Mortality

The case fatality rate (the number of clinically affected animals that die from the disease) of the acute form of ASF is 95 to 100% in susceptible populations. The case fatality rate associated with the subacute form of disease ranges from 30 to 70%. The chronic form of ASF has a low case fatality rate.

Prevention and Control

The importation of only ASF-free pigs is important to prevention of disease in non-endemic countries. In endemic areas, control and eradication of ASF involves slaughter and disposal of all acutely infected and seropositive animals, and good isolation and sanitation practices.

Use of pig-proof fencing to prevent exposure to warthogs or other suspected carriers is recommended. Feeding uncooked remains of any warthog, bush pig, wild pig, or domestic pig carcasses to swine should be avoided. Proper hygiene should be observed and pigs quarantined before they are introduced into herds. Control of tick vectors is also important to prevention of ASF.

There is no vaccine for ASF. The virus is inactivated by sodium hydroxide, hypochlorites, formalin, orthophenylphenol, and iodine compounds.

Strict quarantine and slaughter are employed to control outbreaks. Once AHS is confirmed, infected, recovered, and AHS-susceptible animals that have contacted affected animals are slaughtered; carcasses, bedding, and all animal products in the affected area are destroyed