African Swine Fever

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Abstract

Originally endemic to sub-Saharan Africa, African swine fever virus (ASFV) has spread to many countries around the world, with severe consequences for swine health and commercial swine production. There have been no recorded outbreaks of ASFV in the United States; however, knowledge of ASFV-related diseases and their diagnosis is integral to surveillance and prevention. ASFV has many genotypes, and the clinical signs vary tremendously. Depending on the course of disease, the efficacy and usefulness of available diagnostic tests will vary. Because the virus is transmitted by soft ticks, control has proven challenging.

Introduction

African swine fever virus (ASFV) is an enveloped, double-stranded DNA virus in the family *Asfarviridae* and genus *Asfivirus*. *Asfivirus* is the only genus in the *Asfaviridae* family, and over 20 genotypes of varying virulence have been recorded. ASFV is the cause of African swine fever and related diseases in porcine species. Disease is endemic to areas of sub-Saharan Africa and the island of Madagascar, but has been introduced to many non-endemic areas in across the world, including the Mediterranean, the Middle East, the Caucasus region, and even the Caribbean. Recent outbreaks have affected swine populations in Russia, Latvia, Lithuania, Estonia, and Poland. There is currently no vaccine available for ASFV. The eradication of ASFV is costly and difficult, and may take several decades. This devastating disease is a threat to the domestic swine population of the United States. Intensive surveillance and diagnostic capabilities are necessary to prevent its entry into the United States.

Transmission

Modes of transmission of ASFV include direct and indirect contact. Direct contact includes contact with infected animals. ASFV can survive relatively well in the environment, making indirect contact a major concern. The virus can be spread by fomites, including farm equipment and vehicles. ASFV also survives in the carcasses of diseased animals, including in uncooked meat. One of the most important factors that hampers control and eradication is the vector-borne mode of transmission. Soft ticks in the genus *Ornithodoros* can serve as vectors and reservoirs of ASFV, and can transmit the virus to swine and to other ticks in the same genus. This characteristic makes ASFV the only arthropod-borne DNA virus. *Ornithodoros* species are ubiquitous, and competent vectors exist in North America.

Pathogenesis

ASFV can enter the body through the palatine tonsils, and from there migrate to retropharyngeal lymph nodes, where it may replicate in target cells. ASFV has an affinity for mononuclear leukocytes, particularly monocytes and macrophages. ASFV enters mononuclear cells via endocytosis. However, depending on the genotype, ASFV can replicate in other blood cell types, including erythrocytes, lymphocytes, and neutrophils. The destruction of macrophages leads to the release of injurious agents such as interleukin 6, tumor necrosis factor-α, complement factors, and arachidonic acid. The release of these components compromises hemostasis, which leads to systemic hemorrhage. These intracellular components also lead to inflammation and apoptosis of endothelial cells. According to the World Organization for Animal Health (OIE), the incubation period is 3-19 days, depending on the virulence of the strain. However, within 30 hours of infection, ASFV may be detected in almost all organs. Important secondary sites of viral replication include the liver, kidneys, and spleen.

Clinical Signs and Lesions

ASFV can lead to a wide range of clinical signs in swine, depending both on the host species and the virulence of the ASFV genotype in question. The clinical manifestations of ASFV infection are divided into several categories: peracute, acute, subacute and chronic. The peracute form will result in sudden death. The acute form may result in high fever (104.9°F - 107.6°F), reddened skin (especially the ventral abdomen, the tips of the ears, and extremities), tachypnea, tachycardia, anorexia, depression, vomiting, diarrhea, and abortions. Death usually occurs in 6 to 13 days, and the mortality rate may approach 100%. The subacute form of disease causes the same clinical signs as the acute form, but with decreased severity. The course of disease can last up to 45 days; the mortality rate varies from 30% to 70%. The chronic form of disease leads to a wide range of non-specific clinical signs, including variable fever, anorexia, depression, arthritis, swollen joints, and chronic skin ulcers. The mortality rate for the chronic form is low. Infected animals that recover from clinical disease will remain carriers of ASFV and can contribute to the propagation and spread of the virus.

Wild porcine species that are native to Africa, such as the giant forest hog (*Hylochoerus meinertzhageni*) and the warthog (*Phacochoerus aethiopicus*) usually experience subclinical infections. Porcine species not native to Africa, such as the domestic pig (*Sus domestica*) and the European wild boar, usually experience clinical disease. The one exception is the peccary (*Tayassu tajacu*), which does not seem to be susceptible to the virus.

The classic lesions seen of African swine fever include renal petechiae ("turkey egg kidneys"), widespread petechiation and ecchymosis of mucous membranes, diffuse gastrointestinal edema, congestive splenomegaly, and multiple areas of cutaneous necrosis and ulceration. The top differential diagnosis for ASFV is classical swine fever (CSF), which can be identical in clinical signs and lesions. These two diseases can only be differentiated by diagnostic tests.

Diagnostics

Adequate diagnostic tests are necessary for the detection of ASFV, and are an integral component of disease surveillance and confirming outbreaks. The necessary samples for diagnostic purposes are the spleen, lymph nodes, tonsils, and kidneys. These samples must be kept at 4°C (39.2°F) to be viable. Blood samples (with 0.5% EDTA as an anticoagulant) can be used if collected in the febrile stage.

Several diagnostic tests can be used to detect ASFV. Fluorescent antibody tests can be performed on impression smears of infected tissue and can distinguish between classical swine fever and ASFV. The disadvantage to the fluorescent antibody test is its decreased sensitivity in subacute and chronic cases. An antigen ELISA test is available for ASFV, but is only recommended for acute cases. The advantage to ELISA is that it is relatively inexpensive and quick. The disadvantage is that chronic cases form antigen-antibody complexes, which decreases the sensitivity of the test. PCR is considered the gold standard for detection according to the OIE. Real-time PCR is more sensitive and specific than conventional PCR. There are PCR tests available for many of the known ASFV genotypes. PCR may also be used to detect virus in tick samples. Virus isolation via hemadsorption is a confirmatory test for ASFV. If an animal is positive for ASFV, the erythrocytes will adhere to infected macrophages. This test is highly specific and sensitive. The disadvantage is that it takes at least 6 days to determine a negative result. For this reason, virus isolation is used as a confirmatory test after initial testing.

Conclusion

African swine fever virus is a serious threat to the U.S. swine industry. Given the nature of this disease, its potential to be highly virulent, and its vector-borne transmission, it would be cumbersome and very costly to eradicate. In order to protect swine producers, emphasis must be placed on surveillance and diagnostic testing. In addition to proper biosecurity practices on commercial premises, it is important that veterinary practitioners remain vigilant for evidence of outbreaks of this disease. Knowledge of the clinical signs, the proper diagnostic samples, and proper selection of diagnostic tests in a timely manner is integral to the control of ASFV.

References

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