

# Aujeszky's Disease

*Pseudorabies, Mad Itch*

**Last Updated:** January 2017

## Importance

Aujeszky's disease (pseudorabies) is a highly contagious, economically significant disease of pigs. This viral infection tends to cause central nervous system (CNS) signs in young animals, respiratory illness in older pigs, and reproductive losses in sows. Mortality rates in very young piglets can be high, although older animals typically recover. Recovered swine can carry the virus latently, and may resume shedding it at a later time. Other species can be infected when they contact infected pigs or eat raw porcine tissues, resulting in neurological signs that are usually fatal within a few days. Serious outbreaks were seen in cattle exposed to infected swine in the past, and thousands of farmed mink and foxes in China recently died after being fed contaminated pig liver. Why animals other than pigs do not typically survive this infection is not clear.

Aujeszky's disease can result in trade restrictions, as well as economic losses, in countries where it is endemic. It remains a significant problem among domesticated pigs in some parts of the world. Variants that recently caused outbreaks among vaccinated pigs in China may be a particular concern. Eradication programs have eliminated this disease from domesticated swine in many nations, including the U.S. The disease has never been reported in Canada. However, viruses are often still maintained in feral pigs and wild boar, and could be reintroduced to domesticated pigs from this source. Viruses from wild suids have also sporadically caused Aujeszky's disease in other animals, particularly hunting dogs.

## Etiology

Aujeszky's disease results from infection by suid herpesvirus 1 (SuHV-1), which is also known informally as Aujeszky's disease virus (ADV) or pseudorabies virus. This virus is a member of the genus *Varicellovirus* and family Herpesviridae (subfamily Alphaherpesvirinae). Viral variants that emerged in China in 2011 have significant genetic differences from other SuHV-1 strains that circulate worldwide.

Diverse SuHV-1 strains are maintained in wild boar and feral swine. These viruses may differ from those circulating among domesticated suids in the same region. At least some of the SuHV-1 strains found in wild suids in the United States and Europe appear to be relatively attenuated for pigs, though not necessarily for other species.

## Species Affected

Members of the family Suidae are the natural hosts for SuHV-1. Animals known to maintain this virus include domesticated pigs (*Sus scrofa*), wild boar (various subspecies of *Sus scrofa*) and feral pigs, which may be either domesticated pigs or crosses between these animals and wild boar. Nearly all other mammals are also thought to be susceptible to infection, but as dead end hosts. Clinical cases have been reported in domesticated mammals including cattle, sheep, goats, cats and dogs (although infections appear to be rare in horses); farmed species including mink and foxes; various captive wild animals (e.g., African wild dogs, *Lycaon pictus*; bears); and uncommonly in wildlife (e.g., a panther, *Puma concolor coryi*, in Florida; red foxes, *Vulpes vulpes*, in Europe; skunks; raccoons, *Procyon lotor*). SuHV-1 does not appear to infect the tailless apes, although some other primates are susceptible.

### Zoonotic potential

Clinical signs do not occur in humans. Seroconversion has been reported, but there is no evidence that the virus replicates significantly or is shed from people.

## Geographic Distribution

SuHV-1 is known to occur in parts of Europe, Asia, Latin America and Africa. This virus is absent from a few countries (e.g., Australia, Greenland), and it has been eradicated from domesticated swine in other countries, including many European nations, the U.S., New Zealand and Japan. However, the virus is still present in wild or feral suids in many of these countries, with published reports documenting its presence in the U.S., Europe and Japan.



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## Transmission

SuHV-1 is usually transmitted between domesticated pigs by the respiratory or oral routes, although venereal transmission is also possible. During acute infections, this virus can occur for more than 2 weeks in tonsillar epithelium, milk, urine, and vaginal and preputial secretions. It is usually spread directly between animals by nose-to-nose transmission; however, it can remain infectious for as long as 7 hours in the air, if the relative humidity is at least 55%. Circumstantial evidence, from outbreaks in Europe, suggests that aerosolized virus might be able to travel a few kilometers under some conditions. Infected porcine tissues can transmit SuHV-1, if they are eaten, and some outbreaks were attributed to eating tissues from other infected animals, particularly rodents. Fetuses can be infected *in utero*.

SuHV-1 can be transmitted on fomites and in carcasses. This virus can survive for several days in contaminated bedding, soil, feed, manure, grass and water, with some reports of environmental survival for up to 2 weeks, under certain conditions, at 20-24°C. It may remain viable longer when temperatures are very cold.

The primary method(s) of transmission in wild and feral suids are debated, although some evidence suggests that most viruses may be spread venereally in these animals. Some wild suids may become infected by sniffing tree trunks marked with wild boar saliva during the mating season. Other authors have proposed that direct oral and respiratory transmission between animals might be significant. The risk of aerosol transmission from wild suids to domesticated pigs is thought to be low, as the concentration of animals shedding viruses is low and many of the strains seem to be attenuated.

Infected domesticated and wild pigs can become latent carriers of SUHV-1. The inactive virus is carried in nerve ganglia near the site of virus entry. The trigeminal ganglia seem to be the primary site in domesticated swine, but some studies suggest that the sacral ganglia may be more important in wild suids (although virus can also be found in the trigeminal ganglia). Latent virus can become reactivated after stressors including transport, crowding, corticosteroid injections or farrowing.

Other species usually become infected by close contact with infected pigs, or by ingesting contaminated raw tissues (e.g., meat, liver, and lung) from pigs or other animals. Some animals might become infected through breaks in the skin. Although animals other than pigs may occasionally shed some virus in nasal and oral secretions, they die very soon after infection, and do not usually transmit the virus further. Nevertheless, rare lateral transmission has been reported in sheep and cattle.

## Disinfection

SuHV-1 is susceptible to quaternary ammonium compounds, phenolics, 2% sodium hydroxide, sodium hypochlorite, chlorhexidine, ethanol, iodine and some other disinfectants. Although this virus is only stable between pH

5 and 9, inactivation by acids is reported to be variable. SuHV-1 can be inactivated by sunlight, drying and high temperatures, but how long it remains viable is likely to be influenced by the specific conditions and presence of organic matter.

## Incubation Period

The incubation period is usually 2-6 days in pigs. It is thought to be less than 9 days in cattle and sheep. Reported incubation periods in dogs and cats range from 2 to 10 days, but most cases probably become apparent in 2-4 days.

## Clinical Signs

### Suids

In pigs, the clinical signs vary with the age of the animal. In piglets less than a week of age, fever, listlessness and anorexia are quickly followed by tremors, seizures or other signs of CNS involvement. Some piglets with hindleg paralysis may sit on their haunches in a "dog-like" position. Others may become recumbent and paddle, or walk in circles. Mortality in this age group is very high; once neurological signs develop, the animal usually dies within 24 to 36 hours. Sudden death may also be seen. Similar signs occur in slightly older piglets, but the mortality rate is lower. Vomiting and respiratory signs have also been reported in older age groups.

In weaned pigs, Aujeszky's disease is mainly a respiratory illness, with clinical signs that commonly include fever, anorexia, weight loss, coughing, sneezing, conjunctivitis and dyspnea. Respiratory disease may be complicated by secondary bacterial infections. CNS signs are occasionally seen. Weaned pigs tend to recover after 5-10 days. In adults, the infection is usually mild or inapparent, with respiratory signs predominating. However, some adult pigs may develop more severe respiratory signs that can progress to pneumonia. Neurological signs ranging in severity from mild muscle tremors to convulsions have been reported occasionally. Pregnant sows may reabsorb infected fetuses, abort, or give birth to weak, trembling neonates; affected litters can contain a mixture of normal piglets, stillborn piglets and weak piglets.

Infections in feral swine and wild boar tend to be asymptomatic or mild in many cases. Mild respiratory signs are the most common syndrome observed in the wild; however, CNS signs were documented during an outbreak among wild boar in Spain, as well as in two animals in Germany. Many of the viruses circulating among wild suids seem to be of low virulence, and cause little or no illness even in domesticated pigs, although very young animals may develop respiratory and neurological signs. The effects of these viruses on reproductive function have not yet been evaluated. Whether wild suids have a high degree of resistance to virulent SuHV-1 is not clear. One study found that wild boar inoculated with virulent SuHV-1 from domesticated pigs did not become ill, but two other studies reported moderate to severe clinical signs in feral swine or wild boar.

## Other species

In susceptible animals other than pigs, Aujeszky's disease typically presents with neurological signs, often accompanied by localized pruritus near the site of virus entry. These cases are almost always fatal within a few days.

In cattle and sheep, the initial sign is usually intense pruritus, concentrated in a patch of skin, which presents as; severe licking, rubbing or gnawing. Self-mutilation is common. Affected animals become progressively weaker, and eventually recumbent, before death. Convulsions, bellowing, teeth grinding, cardiac irregularities and rapid, shallow breathing are also common.

Commonly reported clinical signs in dogs include depression, anorexia, dyspnea, tachypnea, and vomiting, excessive salivation, spasms of the laryngeal and pharyngeal muscles, ataxia, and muscle stiffness, spasms or rigidity. Many dogs have facial pruritus, although this is not invariably present. Restlessness or vocalizations are common, and some dogs become aggressive. Bloody diarrhea and/or hematemesis were reported in a few cases. Some dogs die very rapidly with few or no clinical signs; others typically die 1-2 days after becoming ill. Fewer cases have been described in cats, but the reported signs have been similar, with anorexia, signs of pruritus in some cases, excessive salivation, and neurological signs (e.g., incoordination, paralysis). Aggressiveness is reported to be rare in cats. Farmed foxes developed fever, anorexia, vomiting and dyspnea, followed by intense pruritus, frequent snarling and signs of discomfort (repeated lying down and standing).

The clinical signs in mink can include neurological signs (e.g., abnormal behavior such as cage biting), anorexia, diarrhea, dyspnea and vomiting. While some sources state that mink do not usually develop pruritus, animals were reported to scratch at the abdominal and facial skin during an outbreak in China. Whether this seemed to be caused by pruritus or CNS disease was not stated. The pathogenesis of Aujeszky's disease is reported to be different in mink than in other species, and can include systemic hemorrhages. However, there are currently no reports of hemorrhagic signs in live mink.

## Post Mortem Lesions [Click to view images](#)

### Suids

Gross lesions are often subtle, absent or difficult to find in pigs. Many animals have serous or fibrinonecrotic rhinitis, but this may be visible only if the head is split and the nasal cavity opened. Pulmonary edema, congestion or consolidation is sometimes present, and secondary bacterial pneumonia can result in more obvious gross lesions. The lymph nodes may be congested and contain small hemorrhages. Affected pigs may also have necrotic tonsillitis or pharyngitis, congested meninges or necrotic placentitis. Necrotic foci are possible in the liver and spleen, especially in very young piglets or fetuses.

In animals with CNS signs, microscopic examination of the white and gray matter typically reveals nonsuppurative meningoencephalitis. Additional microscopic findings may include necrotic tonsillitis, bronchitis, bronchiolitis and alveolitis. Focal necrosis is common in the liver, spleen, adrenal glands and lymph nodes of affected fetuses. Unusually, experimental infection of older (60-day-old) pigs with one of the newly emerged strains in China caused obvious necrosis in multiple tissues outside the brain (e.g., tonsil, lung, spleen, liver), on histopathological examination.

## Other species

In cattle, the only gross lesions may be areas of edema, congestion and hemorrhage in the spinal cord. These lesions are usually found in the portion of the spinal cord that innervates the area of pruritus. Microscopic lesions of cellular infiltration and neuronal degeneration may be found in the affected portions of the spinal cord and CNS.

Gross lesions are often minimal or absent in dogs, although facial alopecia and edema (due to pruritus), mild to diffuse acute pulmonary alveolar emphysema and edema, endocardial and epicardial petechiae and ecchymoses, and bloody intestinal contents have been reported in some animals. However, dogs with Aujeszky's disease in China often had hemorrhagic lesions in the heart (petechiae and ecchymoses in endocardium and epicardium, valvular hemorrhage, cardiac thrombi), gastric and thymic hemorrhages and focal pulmonary hemorrhage and/or congestion. Most of these dogs had numerous dark-red to black, raised, soft, blood-filled areas in the spleen, and a few had renal or pleural hemorrhage. The typical microscopic finding in dogs with Aujeszky's disease is nonsuppurative encephalitis in the brainstem, but inflammation is also common in nerve ganglia (e.g., the trigeminal ganglia), and there are reports of microscopic lesions affecting the heart and intestinal myenteric ganglia, and small areas of hepatic necrosis. Cases from China had microscopic evidence of hemorrhages and congestion in multiple tissues, and necrosis or exudation in the myocardium, in addition to the lesions reported from dogs in other countries.

The pathology of Aujeszky's disease in mink is reported to differ from other species, with the most prevalent lesions being hemorrhages and ischemia associated with a small-vessel, systemic vasculopathy, and a minimal or even absent inflammatory reaction in the CNS. Gross lesions reported during outbreaks among farmed mink in China included splenic lesions (numerous soft, raised, dark-red-to-black, blood-filled areas), diffuse intestinal erythema, bloating of the stomach and intestines, epicardial petechiae and ecchymoses, focal pulmonary hemorrhage and/or congestion, and renal hemorrhage.

## Diagnostic Tests

### Suids

SuHV-1, its nucleic acids and antigens may be found in nasal swabs, oropharyngeal fluid, and swabs or biopsies of the tonsils in live pigs. The brain, spleen and lung are the preferred organs for virus isolation at necropsy, although virus may also be found in other tissues. Latently infected pigs are most likely to be identified by examining the trigeminal ganglion in domesticated pigs and the sacral and/or trigeminal ganglia in wild suids, but live virus is not usually recovered.

SuHV-1 can be isolated on a number of cell lines or primary cell cultures; porcine kidney (PK-15) cells are most often used. Isolated viruses can be identified with immunofluorescence or immunochemical staining, virus neutralization or polymerase chain reaction (PCR) assays. PCR can also identify viral nucleic acids directly in secretions or tissue samples. Most PCR tests can detect the recently emerged viral variants in China, but new tests that can distinguish these strains from classical SuHV-1 isolates have also been published. Immunofluorescence can detect viral antigens in tissue samples and nasal swabs.

Serological tests for Aujeszky's disease include virus neutralization, latex agglutination and enzyme-linked immunosorbent assays (ELISAs). Other assays, such as a rapid immunochromatographic strip, have been published, and may be licensed in some areas. Some ELISAs and latex agglutination tests can distinguish vaccinated from infected pigs, if gene-deleted vaccines are used. Paired serum samples should be collected in regions where SuHV-1 circulates, to distinguish recently infected animals. Some serological tests can also be used with whole blood, milk or muscle exudates (meat juice).

### Other species

Virus isolation, PCR and antigen detection tests can diagnose Aujeszky's disease in animals other than pigs. Brainstem tissues (e.g., mesencephalon, medulla oblongata and cerebellum) have been used in clinical cases in dogs, cats, foxes, mink and other species. The section of the spinal cord that innervates the pruritic area may need to be collected in cattle. Other recommended tissues in cattle include the pruritic area of skin and subcutaneous tissues. Viral antigens were also found in the cervical spinal cord, trigeminal ganglia, other peripheral ganglia, heart and stomach of some dogs, and the spinal cord, trigeminal ganglia and oropharyngeal mucosa of mink. Other tissues that may be useful include haired skin from pruritic areas, salivary gland, lung, pharyngeal mucosa and adrenal gland.

Serology is not helpful in species other than pigs; these animals usually die before mounting an antibody response.

## Treatment

There is no specific treatment for Aujeszky's disease, except supportive care and treatment for secondary infections.

## Control

### Disease reporting

A quick response is vital for containing outbreaks in disease-free regions. Veterinarians who encounter or suspect Aujeszky's disease should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

### Prevention

Preventive measures in an endemic region include isolation and testing of new animals before they are added to a herd, and biosecurity measures to prevent entry on contaminated fomites, people and roaming animals including rodents and birds. Infected herds may be quarantined to prevent them from transmitting the virus to other animals.

In most Aujeszky's disease-free areas, domestic swine herds must still be protected from contact with feral or wild suids and their tissues. Prevention of direct contact (e.g., a double fence system), together with strict sanitation, is thought to be the most important measure. Some analyses conclude that the risk of aerosol transmission from wild suids appears low. Because infections transmitted to domesticated pigs from wild suids can be inapparent, it may be necessary to periodically monitor higher risk herds with laboratory tests.

Vaccination can aid disease control, but needs to be part of a comprehensive disease control program. Routine vaccination of domesticated herds is generally prohibited in countries that are officially free of Aujeszky's disease. The currently available vaccines protect pigs from clinical signs and decrease virus shedding, but do not provide sterile immunity or prevent latent infections. Attenuated, inactivated and gene-deleted marker vaccines are available; the gene-deleted vaccines allow vaccinated pigs to be distinguished from pigs infected with field viruses. Some of the currently available vaccines are reportedly ineffective against variant viruses that have emerged in China since 2011. Vaccines against these strains are in development. The feasibility of using oral vaccines in feral pigs and wild boar is being investigated.

Strategies to eradicate Aujeszky's disease from a herd include test and removal, offspring segregation, and depopulation. In the test and removal strategy, the breeding herd is tested monthly, with the removal of animals that test positive. This technique works best when there is a relatively low prevalence of infection in the herd. It can also be combined with vaccination. One difficulty with the test and removal strategy is that it may be difficult to detect latently infected animals. In the offspring segregation technique, the breeding herd is vaccinated, and young weaned piglets are removed and raised to adulthood at another site. These pigs are tested periodically, and any positive animals are removed. The original herd is eventually depopulated and replaced with Aujeszky's disease-free animals. Depopulation and repopulation is the most drastic technique. The premises are cleaned, disinfected and left empty of pigs for 30 days.

Preventive measures in other species include avoidance of contact with potentially infected swine, including wild suids, and their tissues or carcasses. Raw tissues from suids should not be fed to carnivores. Vaccines are not currently available for species other than pigs, and the attenuated viruses in swine vaccines are reported to cause Aujeszky's disease in some species.

## Morbidity and Mortality

Aujeszky's disease is especially prominent in areas with dense pig populations. Nearly all of the pigs in some herds may become infected. The mortality rate decreases with increasing age; it may be as low as 1 to 2% in grower and finisher pigs, 5-10% in weaner pigs, up to 50% (or higher) in nursery pigs, and as high as 100% in animals < 2 weeks of age. Approximately 20% or fewer sows abort. However, strains differ in virulence. Some may cause deaths even among adults, while others tend to cause mild or no signs, except perhaps in very young animals. Strains that emerged among vaccinated pigs in China in 2011 are reported to be more virulent than some classical strains. Piglets born to immune sows may be protected by maternal antibodies up to 4 months of age.

SuHV-1 has become an increasing issue in wild suids, primarily as a risk for transmission to domesticated animals. Wild boar and/or feral pig populations are expanding and spreading to new regions. Reported seroprevalence rates in these animals vary widely, from <1% to 61-66%, frequently with areas of both low and high prevalence on the same continent (e.g., North America, Europe). The seroprevalence can fluctuate over time. Many of the SUHV-1 strains found in wild suids in North America and Europe appear to be attenuated for adult pigs, although they may still cause disease in very young piglets. Severe illness has been reported in older suids in the wild, though rarely.

In animals other than suids, Aujeszky's disease is almost always fatal. In the past, severe losses sometimes occurred in cattle exposed to asymptotically infected pigs. Recently, outbreaks among farmed mink and foxes fed contaminated pork livers in China affected approximately 8,000 mink on seven farms, and about 1,200 foxes. Mortality in mink can be high; the mortality rate in this outbreak was 80-90%. In the foxes, the morbidity rate was approximately 80% and the case fatality rate was 100%. In recent years, there have been a number of reports of fatal cases in hunting dogs, which had apparently acquired the virus from feral swine or wild boar in North America, Europe and China.

## Internet Resources

[The Merck Veterinary Manual](#)

[United States Department of Agriculture \(USDA\) Pseudorabies](#)

[World Organization for Animal Health \(WOAH\)](#)

[WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals](#)

[WOAH Terrestrial Animal Health Code](#)

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\* Link is defunct