

Swine Influenza

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Minor updates: June 2024

Importance

Swine influenza is an acute respiratory disease caused by influenza A viruses that circulate among pigs. The morbidity rate is usually high, and mortality low, but more severe outbreaks are possible, and reduced growth rates in young pigs can cause economic losses. Swine influenza viruses occasionally infect other species, including humans. In people, clinical cases tend to resemble human influenza and are often mild, though there have been a few deaths. Most cases involve a single person who had contact with pigs, but limited spread is possible, and in rare instances, swine influenza viruses can become established in human populations. The 2009-2010 human pandemic was caused by a virus that appears to have resulted from genetic reassortment between North American and Eurasian swine influenza viruses. This virus now circulates worldwide as a seasonal human influenza virus. People have transmitted it back to pigs, where it has reassorted with various swine influenza viruses. These events and other changes in circulating swine influenza viruses have increased viral diversity and complicated vaccination programs in pigs. The number of swine influenza cases reported in humans also appears to have increased recently, particularly in the U.S., where many infections were acquired from pigs at agricultural fairs. Whether this is due to the changes in swine influenza viruses, increased awareness and surveillance for zoonotic influenza viruses in humans, or a combination of factors is still uncertain.

Etiology

Swine influenza viruses belong to the species *influenza A virus* (genus *Alphainfluenzavirus*, family Orthomyxoviridae), a large group of highly variable viruses that are adapted to circulate in particular hosts, but can occasionally infect other species. Most influenza A viruses are maintained in birds (avian influenza viruses), but a few circulate in mammals. In addition to pigs, mammalian reservoir hosts include people (human influenza A viruses), horses (equine influenza viruses) and dogs (canine influenza viruses). (Additional viruses circulate in bats, but do not seem to be transmitted to other species.) On rare occasions, influenza viruses can adapt to a new host species, either “whole” or after recombining with another influenza virus.

Influenza A viruses are classified into subtypes (e.g., H3N2) based on two variable surface proteins, the hemagglutinin and neuraminidase. There are currently 18 recognized hemagglutinins (H1 to H18) and 11 neuraminidases (N1 to N11). These two proteins are major targets for the immune response, and there is ordinarily little or no cross-protection between different HA or NA types. Mutations cause gradual changes in a virus’s HA and NA genes, a process called ‘antigenic drift.’⁴ If the hemagglutinin and neuraminidase proteins change enough, a host’s existing immune responses against that virus may no longer be protective.

Genetic reassortment, which results from “re-shuffling” the 8 viral gene segments when two different viruses infect a single cell, can result in more rapid changes. Viruses can reassort whether they are adapted to the same host species or originally came from different hosts (e.g., an avian influenza virus reassorting with a swine influenza virus). Reassortment can result in large or small changes in viruses, but an important aspect is that it can generate viruses containing either a new HA, a new NA, or both. Such abrupt changes, called ‘antigenic shifts,’ may be sufficient for the novel virus to completely evade existing immunity in its reservoir host. The high variability in influenza viruses also means that two viruses with the same subtype (e.g., an H3N2 avian influenza virus and an H3N2 swine influenza virus) may be only distantly related.

Subtypes and diversity in swine influenza viruses

Knowledge about the influenza viruses circulating among pigs is incomplete, but surveillance has identified a number of unrelated H1N1, H3N2 and H1N2 swine influenza viruses, as well as multiple variants and reassortants of these viruses. Several naming systems have been introduced to classify these viruses into lineages and clusters/ clades of related viruses, for instance the H1- α , H1- β , and H1- γ clades, or an alphanumeric global naming system for H1 HA genes (e.g., 1A.1, 1A.1.1; 1A.2, 1A.3.3.3). Virus diversity seems to be particularly high in North America, though the reasons for this are not entirely clear.



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One swine influenza virus, known as the classical H1N1 virus, seems to have circulated in pigs since the 1918 human flu pandemic. Some evidence, based on concurrent outbreaks in humans and pigs on their farms, suggests that pigs may have acquired this virus from people, after which time the viruses in pigs and people diverged. The classical H1N1 swine influenza virus was the major influenza virus of pigs in North America for approximately 70 years. While it was also found sometimes in pigs in Europe, the predominant H1N1 virus in Europe in the last few decades was an avian-origin virus that jumped “whole” from birds into pigs in the 1970s. It is known as the Eurasian H1N1 swine influenza-virus or the avian-like H1N1 virus. Both the classical and Eurasian H1N1 viruses became established in pigs in parts of Asia, and they have also recombined with other viruses. The H1N1 virus that caused the 2009 human pandemic has also been transmitted repeatedly from people to pigs, and subsequent recombined with various swine influenza viruses.

Diverse H3N2 viruses likewise circulate in pigs. A reassortant that contains human-origin H3 and N2 and internal gene segments from the avian-origin H1N1 virus is currently the dominant H3N2 swine influenza virus in much of Europe, though other human-origin H3N2 viruses have also been found in some regions. In North America, the predominant H3 viruses are the triple reassortant H3N2 viruses, which emerged in the 1990s. These viruses, which were the first widespread H3 swine influenza viruses in this region, contain HA and NA genes from human influenza viruses, and internal protein genes from the classical swine influenza virus, an avian influenza virus and a human influenza virus. The combination of internal protein genes carried by these viruses is known as the triple reassortant internal gene (TRIG) cassette. TRIG-containing viruses have become very diverse, recombining with other viruses to acquire various H1, H3, N1 or N2 proteins from human influenza viruses and H1 and N1 proteins from the classical swine influenza virus. North American triple reassortant H3N2 viruses also became established in parts of Asia, where they have undergone additional recombination with the viruses circulating there. One of these reassortants, an H1N1 virus with the TRIG cassette, is reported to be common among pigs in China.

H1N2 viruses are also common in some areas. Surveillance from the U.S. found roughly equivalent numbers of H1N2, H1N1 and H3N2 viruses in 2010-2016. One Asian H1N2 virus caused a major outbreak in Japan in 1989-1990 before becoming established in pigs, and has spread to some other countries. It is a reassortant between the classical H1N1 virus and early human-like H3N2 viruses. There are occasional reports of other subtypes, such as H2N3 and H3N1, though they seem to be uncommon.

Other influenza viruses in pigs

Avian influenza viruses are found sporadically in pigs, feral swine and wild boar. Diverse subtypes (e.g., various

H4, H5, H6, H7, H9, H10 and H11 viruses) have been isolated from infected herds. Serology also provides evidence of exposure to a number of different viruses. While a few avian viruses became adapted to pigs or contributed some of their gene segments to swine influenza viruses, most do not persist. Viruses from other hosts seem to be rare, though one outbreak in China was caused by an H3N8 equine influenza virus, and an H1N7 virus found in pigs in Europe was apparently a reassortant between swine and equine influenza viruses. As of 2022, canine influenza viruses have not been detected in pigs.

Serological and virological evidence indicates that influenza B and C viruses of humans and influenza D viruses, most likely from cattle, can also infect pigs occasionally. Serological evidence suggested that one outbreak in pigs might have been caused by influenza B viruses acquired from humans, though experimentally infected pigs had mild or no clinical signs. Further information on these viruses is available in the influenza D, avian influenza, equine influenza and influenza factsheets.

Species Affected

Swine influenza viruses affect domestic pigs. Evidence for circulating viruses has also been reported in feral swine and wild boar, but the susceptibility of some wild suids, such as peccaries, does not seem to have been examined. Some viruses have caused disease in turkeys, ferrets and mink. Once a virus enters turkey flocks, it can be maintained in this species. There are also sporadic reports of swine influenza viruses in other animals. Two H3N2 viruses isolated from pet dogs in China had high homology to human-like H3N2 swine influenza viruses. Experimental infections have been reported in calves and sheep, and a few studies have suggested that some antibodies found in cattle might have been caused by exposure to swine influenza viruses, although definitive identification of the virus source was not possible. An H1N1 swine influenza virus, which did not cause disease in either poultry or pigs, was isolated from a duck in Hong Kong, and experimental infections have been reported in ducks. There are no published reports of naturally infected chickens, and swine influenza viruses do not seem to replicate efficiently in experimentally infected chickens.

Zoonotic potential

Infections with various H1N1, H3N2 and H1N2 swine influenza viruses are reported sporadically in humans. While there are occasional reports of limited person-to-person transmission, the only virus known to have adapted to humans is the 2009 pandemic H1N1 virus. This virus, which seems to be a reassortant between North American H1N2 and Eurasian H1N1 swine influenza viruses; subsequently became established as a seasonal human influenza virus and is currently the predominant H1N1 virus circulating in people. It contains a hemagglutinin that is most closely related to swine influenza viruses in North America, a neuraminidase related to swine influenza viruses in Eurasia,

and internal proteins from two or more swine influenza viruses including the North American triple reassortant H3N2 viruses and a Eurasian virus.

Geographic Distribution

Swine influenza viruses are thought to be enzootic in most areas that have dense populations of pigs. Outbreaks has been reported from North and South America, Europe, and parts of Asia and Africa. However, these viruses might also circulate inapparently in areas where outbreaks have not been identified, as infected herds can be asymptomatic or have only mild clinical signs. While each continent generally has the same subtypes, the viruses themselves can vary between regions as well as continents.

Transmission

Influenza viruses are transmitted in droplets and aerosols created by coughing and sneezing, and by contact with nasal discharges, either directly or on fomites. Most viruses are thought to enter the body through the respiratory tract, but the eye might act as an additional entry point. Close contact and closed environments favor transmission. The importance of aerosol transmission in pigs is debated, though it has been demonstrated within densely populated pig barns, and has been proposed to occur over longer distances in swine-dense regions. One group found no evidence for transplacental transmission.

How swine influenza viruses are transmitted to farmed mink and ferrets is not entirely clear, but some viruses were thought to have originated in pig or turkey tissues in feed. Other possible sources of exposure, such as nearby pig farms, were also identified in some outbreaks. Humans mainly seem to be infected by contact with pigs or their environments, though a few people were apparently infected via fomites or from other people. Person-to-person transmission does not seem to be common, but it has occasionally been reported among family members or other close contacts, and a limited outbreak occurred on a military base in the 1970s.

Environmental survival of influenza viruses can differ with the type of surface, ambient conditions and presence of organic matter (e.g., feces). Low temperatures and protection from sunlight enhance survival. Swine influenza viruses were inactivated in untreated pig slurry in 1-2.5 hours at 50-55°C (122-131°F), 2 weeks at 20°C (68°F), and 9 weeks at 5°C (41°F). Their persistence on fomites or in water is likely to be similar to other mammalian influenza viruses. Human influenza A viruses remain viable for less than 24-48 hours on most surfaces, and often seem to be infectious for a few minutes to hours in many environments. Nevertheless, they might survive longer under some conditions. Some laboratory experiments suggest that avian influenza viruses and human influenza A viruses may persist for weeks or months in some types of water (e.g., distilled); however, they might be inactivated much faster in aquatic environments that contain normal microbial flora.

Disinfection

Influenza A viruses are susceptible to a wide variety of disinfectants, such as sodium hypochlorite, 60-95% ethanol, quaternary ammonium compounds, aldehydes (glutaraldehyde, formaldehyde), phenols, acids and povidone-iodine. Common household agents, including 1% bleach, 10% malt vinegar and 0.01-0.1% dishwashing liquid (washing up liquid), were found to destroy the viability of human influenza viruses, although hot water (55°C; 131°F) alone was ineffective in rapidly eliminating these viruses. Influenza A viruses can also be inactivated by heat of 56-60°C (133-140°F) for a minimum of 60 minutes (or higher temperatures for shorter periods), as well as by ionizing radiation or extremes of pH (pH 1-3 or pH 10-14).

Infections in Animals

Incubation Period

Clinical signs usually appear within 1-3 days in pigs.

Clinical Signs

Swine influenza viruses in pigs

Swine influenza is an acute upper respiratory disease with clinical signs that may include fever, lethargy, anorexia, weight loss, coughing, sneezing, nasal and ocular discharge, conjunctivitis and labored breathing (expiratory dyspnea or “thumping”). The cough usually develops after a few days, at which time the fever has often started to diminish. Abortions may be seen in some herds. Secondary or concurrent bacterial or viral infections, other illnesses and stressors such as transport can exacerbate the clinical signs. Severe, potentially fatal bronchopneumonia is seen occasionally. Swine influenza viruses can also circulate among pigs with few or no clinical signs, and some herds may have clinical cases only in certain age groups, such as weanling pigs with waning immunity, while other animals remain asymptomatic.

Other influenza viruses in pigs

Infections with the 2009 pandemic H1N1 virus have resembled swine influenza, and were mild in most cases. Diarrhea was reported in experimentally infected pigs and some infected herds, but not others. The clinical signs in pigs infected with avian influenza viruses vary, but respiratory illnesses and asymptomatic infections have been seen. Experimental infections and reports of infected herds suggest that pigs infected with Asian lineage H5 HPAI viruses do not become severely ill.

Swine influenza viruses in other hosts

Turkeys infected with swine influenza viruses may develop respiratory signs and/or experience disorders of egg laying (decreased egg production and/ or abnormal eggs).

Respiratory signs of varying severity were reported during outbreaks in ferrets and mink. Mink infected with one

H3N2 swine influenza virus sometimes developed pneumonia as well as other respiratory signs, and mortality was elevated on some ranches, especially when other pathogens were also present. Another H3N2 virus caused significant coughing but little mortality in this species. An H1N2 swine influenza virus was found in the lungs of mink during an outbreak of severe respiratory disease with hemorrhagic bronchointerstitial pneumonia. However, these animals were co-infected with hemolytic *E. coli*, and the hemorrhagic pneumonia and high mortality rate were attributed to the secondary bacterial component. An outbreak in ferrets, caused by an H1N1 virus, was characterized by respiratory signs that sometimes included severe dyspnea, with some deaths in severely affected animals.

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

In severe, acute cases in pigs, the lungs may be diffusely edematous and congested, with large amounts of foam in the larger airways including the trachea. More often, the disease appears as a cranioventral bronchopneumonia affecting variable amounts of lung tissue. Affected parts of the lungs are consolidated, with a sharply demarcated, dark red to purplish-red, depressed appearance. A few hemorrhagic, emphysematous bullae may occasionally distend the interlobular spaces. Concurrent bacterial infections, common in naturally infected animals, can result in more extensive lesions. Lymph nodes associated with the respiratory tract may be variably enlarged and congested.

Diagnostic Tests

Swine influenza viruses, their antigens or nucleic acids can be detected in respiratory secretions from live or dead animals or in tissues (e.g., nasal turbinates, tonsil, trachea, lung) postmortem. Nasal or oral swabs are the most common diagnostic samples in live pigs, but snout/ nasal wipes, udder wipes of suckling piglets, tracheal or bronchoalveolar lavage samples, or, in a herd health situation, cotton ropes chewed by the pigs, might also be employed. Virus shedding is usually brief, and respiratory samples should be collected as soon as possible after the onset of clinical signs, ideally within 24-72 hours, for virus isolation. Because many infections are mild or subclinical, samples from apparently healthy pigs may also be diagnostic.

Most clinical cases are diagnosed by RT-PCR and/or antigen detection. Tests for swine influenza virus antigens include ELISAs immunohistochemistry and immunofluorescence. Virus isolation may be employed occasionally, particularly in the characterization of new influenza viruses, though it is not used routinely for diagnosis. Swine influenza viruses can be isolated in embryonated chicken eggs or cell cultures (e.g., Madin-Darby canine kidney cells). Isolated viruses can be subtyped with hemagglutination inhibition and neuraminidase inhibition tests or RT-PCR, as well as by sequence analysis of the viral HA and NA genes.

Swine influenza can be diagnosed retrospectively by a rising antibody titer in paired serum samples. The hemagglutination inhibition test, which is subtype specific, is used most often. ELISAs are also available. Other serological assays (e.g., virus neutralization, indirect fluorescent antibody test, agar gel immunodiffusion) have been described in swine, but are not commonly employed in diagnosis. Cross-reactivity with other influenza viruses can sometimes be an issue when using serology.

Treatment

Influenza is usually treated with supportive care, and good management may help reduce the severity of the illness. Antibiotics to control secondary infections may also be appropriate. Antiviral drugs used to treat human influenza are not generally administered to swine.

Control

Disease reporting

Although swine influenza viruses are common and widespread among pigs, and are not usually reportable, veterinarians should remain aware of any reporting requirements in their area. State authorities should be consulted for requirements in the U.S.

Prevention

Management measures such as all-in/all-out production can help prevent the introduction of viruses. Isolating and testing newly acquired pigs, or animals returning to a facility, is also helpful. Other sources of infection to consider in biosecurity plans include contact with wild and feral pigs, wild birds (especially waterfowl and other birds from aquatic habitats), poultry, people and possibly other species such as horses; and fomites including unsafe water sources that may contain viruses. Good management can help decrease the severity of disease in persistently infected herds, and isolating sick animals early might help reduce transmission within the facility. Infected swine herds can be cleared of influenza viruses by depopulation. Certain management programs may also be successful.

Vaccines, which are often targeted at certain age groups (e.g., sows, growing/finishing pigs), are used to help control clinical signs in some countries. A number of commercial and autogenous swine influenza vaccines are produced, but the hemagglutinins of the vaccine strain and the virus must be well-matched; thus, the diversity of viruses can complicate vaccine use and selection. Furthermore, some combinations of swine influenza vaccines and poorly matched challenge viruses were reported to exacerbate disease in pigs, at least in a laboratory setting. An additional concern is that selection pressures from increased vaccination might encourage more rapid changes in virus diversity.

Morbidity and Mortality

Swine influenza viruses

Swine influenza viruses are common in pigs, and many farms worldwide are infected with at least one virus. Seroprevalence rates of about 20-60% are frequently reported in surveys, though some studies find higher or lower values. Higher herd and pig densities are associated with higher prevalence. Studies of feral swine in North America and wild boars in Europe and Asia have found antibodies in 0% to $\geq 40\%$ of the animals tested, though fewer animals seem to be infected than in domestic herds.

The severity of an outbreak can be influenced by management factors, co-infections with other pathogens and other stressors. However, mortality rates are generally low in uncomplicated cases, with case fatality rates typically ranging from $< 1\%$ to 4%, and most animals recovering within 3-7 days. The main economic impact is usually from reduced weight gain and a longer time to reach market weight. Infections with the 2009 pandemic H1N1 virus and many avian influenza viruses tend to resemble swine influenza. The 2009 pandemic H1N1 virus often causes only mild disease in pigs, with reported morbidity rates from $< 1\%$ to 90%, but little or no mortality.

Outbreak patterns are influenced by pre-existing immunity and the type of production system. If a virus infects a naive population, it may cause an epizootic with rapid transmission in pigs of all ages, and in the classical picture of influenza, up to 100% of the animals in a naive herd may become ill. Under current production systems in endemic areas, however, it is more common for viruses to persist in a herd and cause recurrent outbreaks in nursery piglets and/or older fattening pigs. Annual outbreaks are mainly seen during the colder months in traditional production systems in temperate climates, but they can occur year-round in tropical and subtropical regions. Outbreaks can occur at any time of the year under intensive (confinement) farming, but are more common when there are fluctuating temperatures and decreased ventilation, such as autumn.

Swine influenza viruses in mink and ferrets

Morbidity and mortality rates reported in influenza outbreaks among mink vary greatly, and appear to be influenced by factors such as co-infections with other pathogens. In an outbreak caused by a triple reassortant H1N1 swine influenza virus in ferrets, the morbidity rate was 8% and the mortality rate was 0.6%.

Infections in Humans

Incubation Period

Swine influenza in humans seems to become apparent within a few days.

Clinical Signs

Swine influenza in people is generally indistinguishable from the illnesses caused by human influenza A viruses (seasonal influenza). Most confirmed cases have been mild, though young children were sometimes hospitalized for dehydration. Upper respiratory signs and nonspecific signs of illness are common, and some patients may have gastrointestinal signs. One study of North American H3N2 viruses found that ocular redness or irritation was more common than with seasonal influenza viruses. There may also be other presentations: acute parotitis was reported in a 6-year-old with H3N2 influenza, and the symptoms in one young patient were limited to fever and vomiting. Pneumonia, serious illnesses and deaths have been reported sporadically, generally, though not always, in people who had underlying health conditions or were immunocompromised by other illnesses or pregnancy. Serological surveillance of occupationally exposed professions supports the existence of mild or asymptomatic infections.

Diagnostic Tests

Diagnosing a swine influenza virus infection is complicated by the clinical similarity between these illnesses and human influenza, the viral proteins shared by some human and swine influenza viruses, and the poor sensitivity of some routine diagnostic tests employed in people (e.g., commercial rapid test kits) for some animal influenza viruses. Many recent swine influenza cases were diagnosed by genetic methods, particularly RT-PCR, though virus isolation can also be used. A number of cases were detected by investigating influenza-like illnesses associated with attendance and contact with pigs at fairs. It may also be possible to diagnose infections retrospectively by serology; however, cross-reactivity with human influenza viruses can be an issue. Testing for novel influenza viruses is generally performed by state, regional or national public health laboratories.

Treatment

Illnesses caused by swine influenza viruses are treated similarly to human influenza, with supportive care (e.g., fluids and rest) in uncomplicated cases, antibiotics as needed for secondary bacterial pneumonia, and hospital care if necessary for more severe illnesses. Two groups of antiviral drugs inhibit influenza A viruses: the adamantanes (amantadine, rimantadine), and neuraminidase inhibitors (zanamivir, oseltamivir, peramivir and laninamivir). These drugs are most effective if started within the first 48 after the onset of clinical signs. There is limited knowledge about their efficacy for the swine influenza viruses, but amantadine-resistant isolates were found to be common in viral lineages found in pigs in the U.S.

Prevention

Protective measures for zoonotic influenza viruses include sanitation and hand hygiene (i.e., frequent hand washing), and the use of personal protective equipment (PPE), when appropriate. Detailed recommendations, including recommendations for people and exhibitors attending agricultural fairs, have been published by some public health agencies. Generally such recommendations focus on hand hygiene, avoidance of close contact with pigs, and precautions to avoid contamination of mucous membranes, such as prohibition of eating and drinking in swine barns. They also advise people at risk for more serious illnesses from human seasonal influenza viruses, including young children, to stay away from pigs, as well as pig barns at fairs. While live swine influenza viruses are not likely to be present in retail meat, any viruses that survived long enough to reach consumers would be inactivated by cooking the meat, while viruses on fomites can be neutralized with ordinary food safety precautions used when handling raw meat products.

When visiting a physician for an illness that began soon after contact with animals, the potential for zoonotic exposure should be mentioned.

Morbidity and Mortality

The overall prevalence of swine influenza virus infections in humans is uncertain, as most infections resemble human influenza and are likely to be overlooked. More than 350 serologically or virologically confirmed clinical cases caused by H1N1, H1N2 and H3N2 viruses have been reported sporadically since the 1950s, including one localized outbreak at the Fort Dix military base in 1976. Clinical cases have been documented more frequently in recent years, possibly due to factors such as changes in the circulating viruses, susceptibility of young people to viruses previously maintained in humans and now circulating in pigs, and increased awareness of zoonotic influenza, as well as new reporting requirements in the U.S.

While the interpretation of serological studies is complicated by cross-reactivity with past and present human influenza viruses, such studies suggest that mild or asymptomatic swine influenza virus infections might be relatively common among people who work with pigs. Clinical cases are also seen in people who have more casual contact with pigs, such as visitors at agricultural fairs or livestock shows, and there are a few reports where there was no apparent swine contact, i.e., infections probably acquired via fomites or from another person.

Although adults have also been affected, many cases, including most of the recent H3N2 cases in the U.S., were in children. Most illnesses have been mild and resembled human influenza. However, people at elevated risk of severe illness from human influenza viruses (e.g., young children, the elderly, those who are immunosuppressed or who have chronic respiratory or cardiovascular conditions) are also

expected to be at greater risk from swine influenza viruses. In rare instances, both human and swine influenza viruses can cause severe or fatal cases even in young, healthy people. A review of swine influenza cases between 1950 and 2014 found nine illnesses (< 3%) that were fatal. Three patients were previously healthy, including two young patients and one 37-year-old; one was pregnant; three were immunosuppressed (two cancer patients, one 3-year-old child on long-term steroids for chronic kidney disease); and one was elderly, with congestive heart failure and diabetes. The health status of one person was not known. There have also been infrequent reports of serious cases since that time, such as two recent descriptions of severe illnesses in an obese but immunocompetent adult in his 40s and an apparently healthy school-aged child. Both survived with intensive care.

Internet Resources

[European Centre for Disease Prevention and Control \(ECDC\). Swine Influenza](#)

[Public Health Agency of Canada \(PHAC\). Human Influenza A with Swine Origin](#)

[The Merck Manual](#)

[The Merck Veterinary Manual](#)

[United States Centers for Disease Control and Prevention \(CDC\). Information on Swine/Variant Influenza](#)

[U.S. Department of Agriculture \(USDA\) Animal and Plant Health Inspection Service \(APHIS\).](#)

[World Health Organization. Influenza. Avian and Other Zoonotic](#)

[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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