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Swine Influenza, Pork Industry Handbook

Michigan State University

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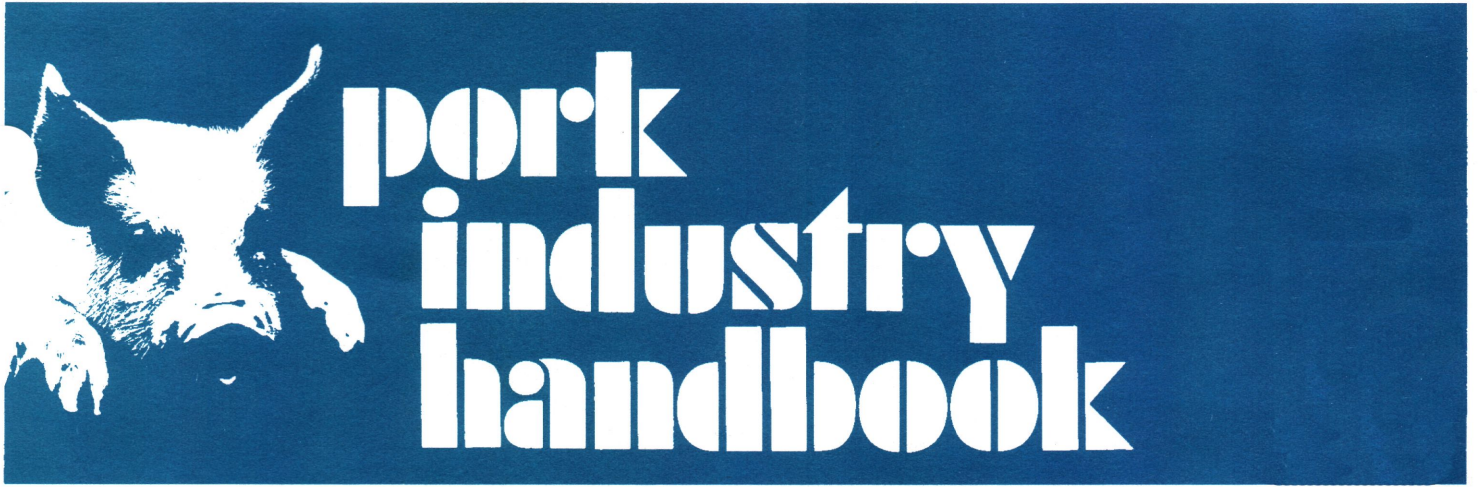
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Swine Influenza

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The first descriptions of influenza in swine appeared in reports of a new disease affecting pigs on farms in western Illinois in 1918. The outbreaks occurred during the time of the great human influenza pandemic (“Spanish flu”) that was responsible for the death of over 20 million people worldwide. Because of the similarity in clinical signs to the disease in humans, the disease in swine was called “hog flu.” But another 12 years passed before researchers proved that the disease is caused by a virus in both swine and humans. The viruses responsible for illness in pigs and people were virtually indistinguishable, and these isolates served as the prototype classical Type A H1N1 influenza viruses. Most researchers believe that the virus, which originally existed in ducks, likely passed from the human to swine populations during the pandemic.

Clinical Signs

The earliest infections were characterized by acute outbreaks of high morbidity (virtually all animals affected) that swept through entire herds in a very short time. Such classic epizootics still commonly occur. Pigs develop high fevers (104°-107° F) and become lethargic and reluctant to move. They may exhibit rapid forced breathing movements at rest, and when forced into activity, many demonstrate a harsh barking cough that is characteristic of the infection. Pigs of all ages may be affected; nursing pigs often show the least severe clinical signs. Pregnant animals frequently abort. Although pigs appear to be quite ill, in the absence of secondary bacterial infections, death loss is minimal. Individual pigs recover within five to seven days; but, because not all pigs become infected at the same time, two to three weeks may pass before the entire group returns to normal.

In some herds, swine influenza virus infections are enzootic, i.e., the virus contributes to chronic ongoing disease problems

in the herd without the appearance of an acute outbreak. These situations occur when pigs from different sources with different levels of immunity are mixed after weaning. The virus is carried into the new incoming group with pigs from one of the sources, or introduced from older pigs already on site. The virus moves slowly through the population, causing disease in those pigs with insufficient immunity and only subclinical or no infection in immune pigs. The presence of swine influenza virus in these situations may not be recognized clinically. Such problems are observed in nursery, grower, or finisher pigs. Many are nursery pig problems in large continuous-flow operations. In these cases, swine influenza virus often is found in combination with other swine respiratory pathogens such as porcine reproductive and respiratory disease virus, *Mycoplasma hyopneumoniae*, *Pasteurella multocida*, and *Streptococcus suis*.

Death loss and the number of chronically ill, poor-doing pigs that result may be quite high. These high morbidity, high mortality respiratory disease problems cost producers significant economic loss, not only through loss of pigs but also through decreased feed efficiency and rate of gain because of prolonged recovery time from secondary bacterial infections, as well as through increased medication costs.

In many herds, swine influenza appears predictably once a year, usually in the fall or early winter. Data from diagnostic laboratories suggest there is a definite “flu season” during which the number of diagnoses of swine influenza increases, usually in October through December (see Figure 1). But the disease occurs year-round, and a second smaller peak consistently appears in the spring. Both epizootic and enzootic forms of the disease can be found at any time of year. The virus is nearly ubiquitous; serologic surveys indicate an 80 percent to 90 percent herd prevalence for swine influenza virus in the north central U.S.

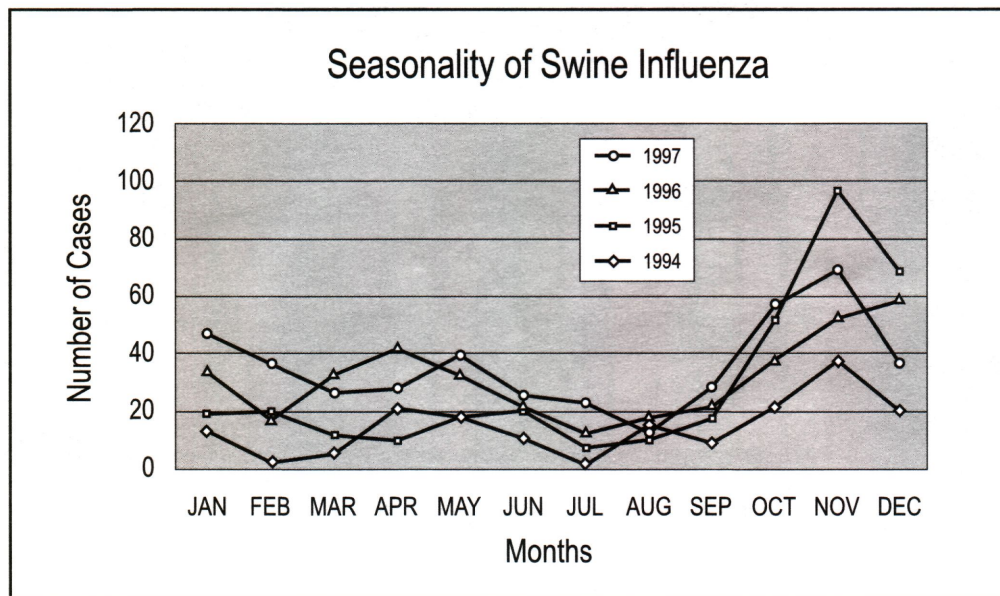


Figure 1. Seasonal distribution of swine influenza diagnoses in cases submitted to the Iowa State University Veterinary Diagnostic Laboratory (1994-1997) which demonstrate the annually consistent rise in influenza cases every fall and a lesser rise each spring.

Pathogenesis of Disease and Lesions

Swine influenza is a highly contagious disease with a very short incubation period. Pigs inoculated intranasally will develop fever and begin shedding virus in nasal secretions within 24 hours. The virus infects epithelial cells lining the nasal passages, trachea, bronchi, and bronchioles in the lungs. Necrosis and sloughing of these epithelial cells and the consequent irritation to the airways of the respiratory tract results in the harsh, barking cough characteristic of the disease (see Figure 2). In the absence of significant secondary bacterial

infections, lungs may return to normal within two weeks of initial infection.

Because of the airborne route of entry and the initial infection of the airways, the gross lesions induced by swine influenza virus resemble bronchopneumonia of bacterial origin. Irregular coalescing areas of consolidation are observed in the cranioventral and hilar areas of the lung (Figure 3). In severe infections, involvement of nearly the entire lung, with spread of the virus throughout all levels of the airways and into the alveoli, may result in a diffusely firm and edematous lung.

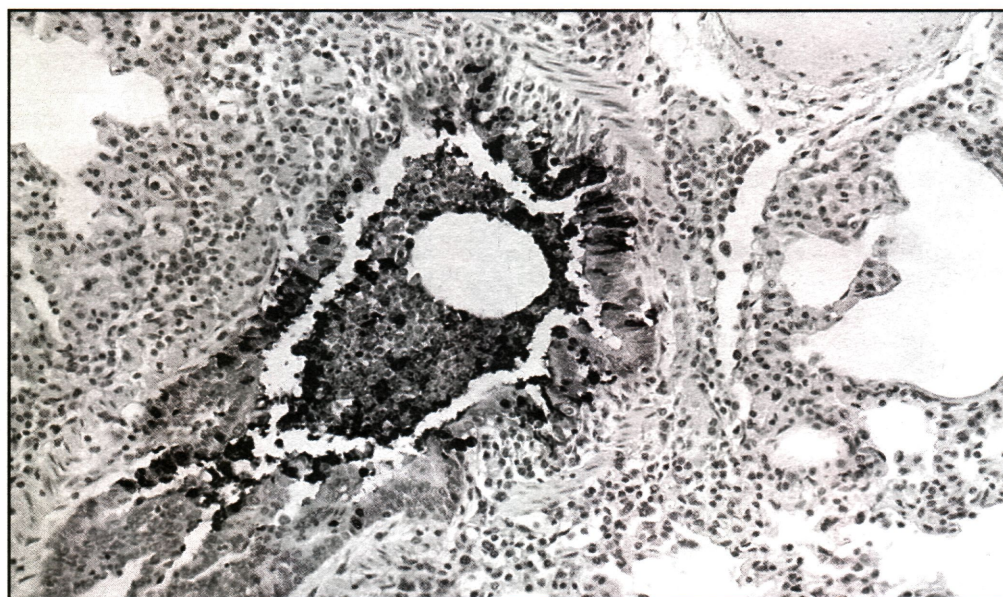


Figure 2. Photomicrograph of lung from a nursery pig infected with swine influenza virus approximately 24 to 48 hours after initial infection. Virus-infected bronchiolar epithelial cells (as demonstrated by immunohistochemical technique) are beginning to slough into the lumen of the bronchiole.

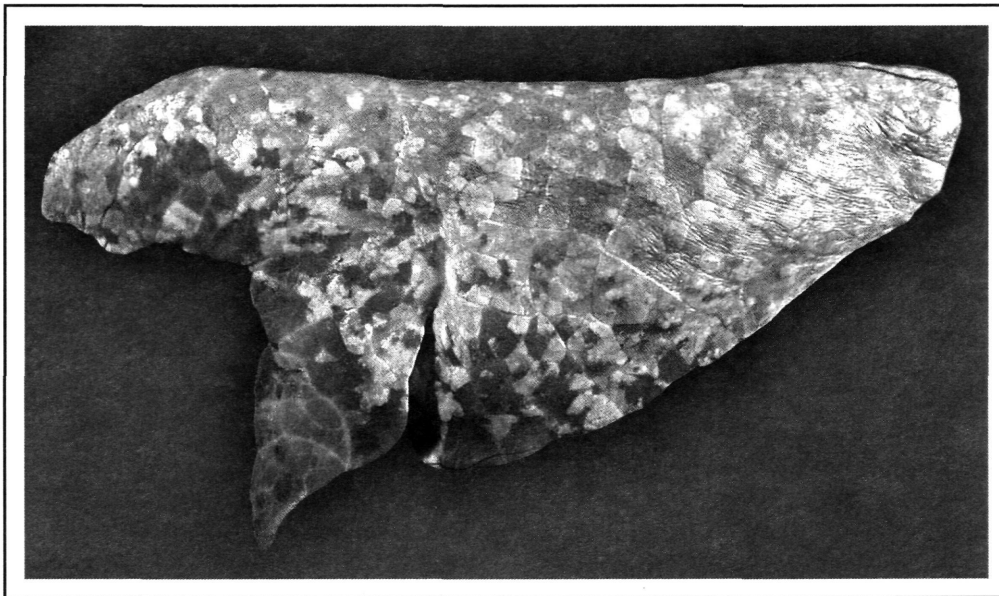


Figure 3. Lung from an 80 lb. pig infected with swine influenza virus. The coalescing dark red foci of lobular consolidation in the cranioventral portions of the lung are characteristic gross lesions of SIV infection.

Influenza Viruses

Swine influenza virus, unlike most other viruses affecting swine, is related to a much broader family of similar viruses that infect a wide range of host species. Influenza is a significant disease problem in humans, pigs, horses, and domestic poultry; but subclinical, sporadic, or experimental infections have been reported in many other species including waterfowl and other birds, sheep, goats, alpacas, fallow deer, dogs, cats, bears, bats, ferrets, mice, primates, harbor seals, and even whales.

Influenza viruses are enveloped single-stranded RNA viruses called orthomyxoviruses. The virus has 10 genes. Eight of the genes code for internal proteins. These proteins are highly conserved, meaning very little variation is found in these proteins regardless of the source of the virus. They include type-specific antigens which are used to identify virus isolates as influenza viruses and to classify the influenza viruses as Type A, B, or C. Influenza viruses that infect animals are almost exclusively Type A viruses. Most of the influenza strains of significance in humans are Type A strains, but Type B and C influenza viruses are also human pathogens. The internal proteins are not considered major immunogens against which a host will raise a humoral immune response that protects against reinfection. However, some of these proteins may be important in stimulating the lymphocytes involved in cell-mediated immunity which facilitates recovery from infection.

The two remaining genes code for the proteins most important in characterization, the hemagglutinin (H) and neuraminidase (N), external proteins which project from the surface of the viral envelope. The hemagglutinin proteins contain the receptor binding site responsible for attachment of virus to host cell receptors, fusion of viral envelope with host cell membrane, and internalization of the viral genome into the host cell. This protein also contains the antigens most important in eliciting a protective host immune response. The neuraminidase proteins assist in penetration of the virus through the mucus layer that lines the respiratory tract, in release of progeny virus from host cells, and in prevention of self-aggregation of virus

particles. The neuraminidase also stimulates an immune response in the host, but this response is less protective than that raised against the hemagglutinin. More extensive variation is found in the external H and N proteins than in the internal proteins, and these external proteins have been used to classify influenza viruses into subtypes. Fifteen different H subtypes and nine different N subtypes have been identified among Type A viruses.

Only influenza viruses with H1 and H3 proteins and N1, N2, and N7 proteins have been found in swine. Worldwide, the two most common subtypes of influenza virus that cause significant disease in swine are H1N1 and H3N2. The original H1N1 swine influenza virus was found only in the U.S. until about 1976 when it apparently was carried to Italy with pigs imported from the U.S. From that point, the virus spread to the rest of Europe and to the British Isles. Subsequently, other strains of H1N1 and H3N2 influenza viruses were found in swine in these countries. Currently, swine populations in Europe and Great Britain are infected with both H1N1 and H3N2 strains, and more recently, H1N2 strains.

For reasons unknown, only H1N1 strains appeared to be responsible for influenza in swine in the U.S. for over 75 years. Serological surveys revealed a low level of infection (<5%) by H3N2 strains, probably originating from humans with little clinical effect, but no new subtypes were isolated from the swine population during that time. Not all H1N1 strains were similar.

In 1988, several outbreaks of respiratory disease in swine in Canada were described as "atypical swine influenza" because of the prolonged course of disease in the affected herds, the unusual microscopic appearance of infected lungs, and because the virus, although still an H1N1 strain, exhibited a much greater degree of antigenic and genomic variation from previously described strains. Reports of similar SIV infections in the north central U.S. followed; and in 1992, an additional strain of "atypical" SIV, distinct from that found in Canada, was isolated from pigs in Nebraska.

A more dramatic change occurred in the U.S. in 1998. H3N2 influenza viruses were recovered for the first time from swine with severe respiratory disease. Many herds reported abortion and death in adult swine, and respiratory disease in pigs of all ages. The new subtype became widespread in a very short time. Serologic studies conducted in the midwest at the end of June, 1999, indicated that over 90 percent of swine herds had been infected with the new H3N2 virus. Over 2/3 of the herds had been exposed to both viruses.

Epidemiology

Most influenza infections in swine originate from other swine. Individual pigs shed the virus in oral and nasal secretions for 5 to 7 days and do not harbor the virus for prolonged periods. Exposure of naive (non-immune) pigs to infected pigs results in rapid infection of additional pigs and more shedding of virus. In this way, the virus moves through an entire group of pigs in a short time. In populations of pigs containing both naive and immune pigs, virus spread through the group may be slower. The source of virus in herds that repeatedly develop acute outbreaks every fall is uncertain. Because the outbreaks occur even in closed herds into which no new swine have been introduced, the virus must remain in the herd between outbreaks. No long-term carrier state has been identified. The virus likely continues to circulate within the herd at a low subclinical level until enough naïve pigs are present to support a new epizootic.

A much debated and researched aspect of influenza viruses that impacts epidemiology is the potential ability of these viruses to cross species boundaries and infect species other than the host from which they originated. As mentioned earlier, influenza viruses are found in humans and in a wide variety of animal species in addition to swine. Waterfowl appear to be the natural reservoir for this virus. Populations of wild ducks, geese, and wading shorebirds throughout the world are almost universally infected with influenza viruses. Every subtype of influenza virus that has ever been recovered from humans, other mammals, and birds has been found present in these populations, as well as some subtypes that are unique to the waterfowl. The infections in waterfowl are asymptomatic intestinal infections. The birds shed the virus in feces and thus can easily contaminate the environment in which they live, exposing each other as well as other species with which they come in contact. In contrast, influenza in other species is a respiratory tract infection, and the virus is spread among individuals via nasal secretions by direct contact or by aerosolization through coughing.

Researchers believe that influenza in other species originated from the viruses in the waterfowl reservoir. Infrequently, and under circumstances not fully understood, a strain of avian influenza virus infects a mammalian host and becomes adapted to that species. Some of the viruses responsible for human influenza pandemics appear originally to have been avian strains. Some of the viruses recovered from swine in Europe and Great Britain also appeared to have been avian strains. Influenza viruses also appear to have the ability to cross between humans and swine and between domestic poultry and swine. Although the potential for this cross-species infection is real and specific cases have been documented, the event does not occur frequently. In most cases, such infection appears to be short-lived with minimal clinical effect.

Another aspect of influenza viruses that relates to cross-species infection and disease is the fact that the genes are distributed among eight separate genome segments. This gives the virus a greater capacity for forming new strains by reassortment. If a host is infected with more than one strain of influenza virus at the same time, during multiplication within the cells of the host, some of the new virus particles produced may contain some genes from each virus, resulting in the formation of a new influenza strain. This process is called antigenic shift and may result in the appearance of a new virus with dramatic disease consequences. Reassortment may contribute to the appearance of new epidemics of human influenza that occur every 10 to 20 years.

The pig has been described by some researchers as the likely host in which reassortment occurs (the "mixing vessel"). The normal body temperature of the pig lies between the higher temperatures of birds and the lower temperatures found in humans. The respiratory tract of the pig also contains both types of cell receptors to which avian and human influenza viruses can attach. Southern China and other parts of Asia may serve as the center from which many new strains of human influenza virus originate because the area has some of the highest concentrations of humans, pigs, and domestic ducks in the world. Because of agricultural practices in the area, humans, pigs, and ducks are in close contact.

Molecular analysis of the H3N2 swine influenza viruses that appeared in swine in the U.S. in 1998 indicated that the North Carolina isolate was a double reassortant virus with genes for all but one of the internal proteins from the pre-existing H1N1 swine virus and genes for the external H3 and N2 proteins and one internal protein from a human H3N2 virus. The midwest isolates were triple reassortant viruses that contained the same three genes from the human H3N2 virus but also two genes for internal proteins from an avian virus along with the remaining five genes from the original H1N1 swine virus. Ongoing studies are being conducted on viruses and sera from swine to determine when and where reassortment first may have occurred.

Diagnosis

Because of its dramatic appearance, acute outbreaks of swine influenza can be fairly reliably diagnosed on clinical signs. However, in enzootic situations, the presence of the virus cannot be determined by observation alone and submission of samples to a veterinary diagnostic laboratory is necessary. Because of the existence of more than one subtype in swine populations, identification of the specific subtype may be necessary if vaccination is desired. Tests are available for direct detection of the virus or for measuring antibody induced by the virus.

Necropsy of affected pigs may reveal typical gross lesions, but other pathogens can induce similar lesions. Portions of the affected lung can be submitted to diagnostic laboratories for histopathologic evaluation for characteristic microscopic lesions, for fluorescent antibody or immunohistochemical tests that detect the virus in these tissues, or for isolation of the virus. If no animals are available for necropsy, nasal swabs can be taken for direct detection of virus in the nasal secretions by antigen-capture enzyme-linked immunosorbent assay, or by isolation. Nasal swabs or lung tissue should be taken from acutely ill pigs exhibiting high fevers. The virus disappears very rapidly from the pig and is difficult to detect by four to five days after the beginning of infection.

Serum samples can be taken from pigs that have recovered to determine if antibody against the virus is present. Pigs will develop detectable titers by a week after infection. A more thorough method of serologic testing is to take serum samples at the time of illness and again two to four weeks later from the same pigs and compare the titers. Pigs infected with the virus should exhibit a marked rise in titer. Tests have been developed in most laboratories that will allow identification of the specific subtype with which the pigs have been infected.

Occasionally, requests are received at diagnostic laboratories to test aborted fetuses for swine influenza virus. Although pregnant sows with acute influenza may abort as a secondary reaction to high fever, there is minimal evidence that swine influenza virus will cause abortion by direct infection of the fetus and examination of fetal tissues for this virus is unwarranted. Serologic testing of sows that have aborted is a better method of determining if influenza is involved.

Treatment and Prevention

Treatment of herds afflicted with influenza is directed toward antimicrobial therapy to control secondary bacterial infections. If environmental conditions and management factors are good, and bacterial respiratory pathogens are not a problem in the herd, such therapy may not be necessary.

The first step in prevention is to stop exposure of immunologically naive younger pigs to older previously infected pigs, some of which may still be shedding virus. All-in, all-out

movement of pigs into areas with common or nearly adjacent airspaces may be necessary because of the ready spread of the virus by aerosolization.

Vaccination also has been effective in reducing problems due to swine influenza. Vaccination of sows has been used to protect pigs that contact the virus in the nursery through prolongation of passive immunity in these pigs. If the problem occurs in older grower or finisher pigs, vaccination of nursery pigs may be beneficial. In these situations, the sows should not be vaccinated as the prolonged passive immunity may interfere with effective vaccination of the weaned pigs. Most pigs nursing unvaccinated sows with low residual HI titers will have passive titers drop below 1:40 (which the vaccine manufacturer suggests may interfere with response to vaccination) by 6 weeks of age. The majority of pigs nursing vaccinated sows will not have titers that drop below 1:40 until they are about 16 weeks old. Evaluation of the age at which infection usually occurs in the herd and the rate of decline of passive immunity in weaned pigs may be necessary to determine the best vaccination scheme.

Currently, separate vaccines are available for H1N1 and H3N2 swine influenza viruses. Thus, diagnostic efforts may need to be directed toward not only identifying swine influenza as a contributor to respiratory problems in a given herd, but also toward determination of the subtype involved so that the proper vaccine can be used for control of the problem. Combination vaccines will likely be available in the near future.



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