



Meeting Report

Consultation on potential risks of pandemic influenza A (H1N1) 2009 virus at the human-animal interface

Scientific teleconference

3 June 2009

Background

The global transmission of the pandemic influenza A (H1N1) 2009 virus¹ continues to occur through person-to-person contact. Joint statements have been made by the World Health Organization (WHO), the Food and Agriculture Organization (FAO) and the World Organisation for Animal Health (OIE) about the safety of pork and pork products. Some questions may remain, however, about the potential risk to human health through contact with pigs potentially infected with this virus and their products from primary production to consumer. At the time of this Consultation, the pandemic (H1N1) 2009 virus had been confirmed in one swine herd in Canada.² While food safety issues are not normally raised on a global level when human infections with what appears to be a swine influenza virus occur, given the current public health context it is essential to base ongoing decisions on the most current and accurate science available.

Objectives

The purpose of the Scientific Consultation was to answer questions using the existing science on influenza viruses infecting pigs and identify knowledge gaps associated with the risk of exposure (and subsequent clinical illness) to the pandemic (H1N1) 2009 influenza virus at the human-animal interface. Answers to a list of questions on risks from direct contact, the environment and along the food-chain, as well as the risk of humans transmitting virus to pigs, were sought. Questions were answered using available data on pandemic (H1N1) 2009 virus, extrapolation from data on other influenza viruses that affect swine and expert opinion. In addition, the experts identified data/research needs that are most critical to the above issues and that need to be addressed as a matter of priority.

Areas of discussion

I) Risks from direct contact

What is the nature and level of risk to people working with live pigs?

Swine influenza viruses, including H1N1 and certain other influenza subtypes, can circulate endemically in swine herds. Sporadic human infections, with or without clinical signs, have been reported from some countries with occasional virological confirmation, but also clear serologic evidence of human exposure to these viruses¹. Influenza surveillance in humans, even when extensive, only captures a small proportion of all influenza infections with a minimal amount at the human-animal interface.

According to a number of published studies of occupational exposure, evidence of human infection with swine influenza viruses among those working with pigs in the United States of America (USA) is not uncommon[1-8]. Limited data are available from other countries. Among workers in the USA with direct exposure to pigs, one study found seroprevalence was highest among farmers, followed by veterinarians and then slaughterhouse workers [3]. Within these studies, a number of them [1,3,5] found an increased seroprevalence to circulating swine influenza viruses in farmers compared to urban controls. A serological study of spouses of swine workers in the USA, without reported direct exposure to pigs themselves, also showed evidence of possible viral transmission from the workers to the spouses. The specific exposure remained unclear, but may have been direct human-to-human transmission or fomite transmission [5].

Evidence shows that influenza virus infections in pigs are respiratory in nature and not systemic. It was reported that virus shedding occurs via nasal secretions and coughing during the time when the animal is acutely ill with fever and lethargy. The approximate time frame for shedding has been reported as 2 days after infection, continuing for 4 to 7 days [9, 10]. Virus has not been demonstrated to be shed through the faeces. It was discussed that comorbidities and other conditions, which may exist more frequently in the field, may exacerbate the clinical picture in infected animals.

Virus can circulate among pigs throughout the year. Although there is a seasonal pattern to influenza in pigs, the disease is not restricted to the cold seasons for pigs living in closed systems.

¹ Termed 'pandemic (H1N1) 2009' at the time of this writing

² This was the case at the time of the Consultation. Since, however, there have been other instances where swine herds have been affected in other countries presumably from viral transmission between humans and livestock, including poultry.

Based on an outbreak on a swine farm in Canada where the pandemic (H1N1) 2009 virus has been detected [11] and from recent studies³, the clinical picture for infection with pandemic (H1N1) 2009 virus in pigs is similar to that for other swine influenza viruses.

What is the nature and level of risk to those involved in slaughtering and butchering/processing?

Slaughterhouse workers may be at lower risk than farmers and veterinarians, according to an occupational study conducted in the USA [3]. Since viraemia in pigs is presumed to be very rare and they are not thought to shed virus in faeces, risk of human exposure is believed to come from handling the respiratory tissues only, not the meat or blood. It is always recommended that only healthy animals are allowed into the food-chain after appropriate ante mortem and post mortem inspections.

What is the effect of vaccination on these risks?

The evidence would suggest that appropriately-vaccinated pigs are less likely to become clinically ill and also less likely to shed viruses. The same is believed to be true for maternal immunity, which lasts approximately 10 weeks.

It was reported that some commercial swine vaccine have not had good efficacy and, therefore, autogenous vaccines are frequently used in the USA.

Consensus statement on risks from direct contact:

Humans in direct contact with pigs infected with swine influenza viruses can become infected and can develop influenza-like illness (ILI). Since the virus is shed through nasal secretions of clinically ill pigs, exposure is commonly through aerosols or droplets but is negligible through contact with faeces. The true frequency of human infections resulting from contact with swine is not known. Appropriately-vaccinated swine herds are thought to pose less of a public health risk than unvaccinated herds.

To date, there is no information available to suggest that the pandemic (H1N1) 2009 virus is currently circulating in pigs.

II) Risks in the environment

What scientific evidence is available regarding the presence and persistence of viable influenza viruses especially influenza viruses that infect swine, in manure, the farm environment and on surfaces/fomites?

There has been little specific work done on persistence of swine influenza viruses in the environment, however it would be expected to be similar to that of other influenza viruses. It was agreed that influenza viruses generally persist longer in cold areas. It has been reported under experimental conditions that virus survives in small particle aerosols [12].

It was suggested that due to the negligible faecal shedding of swine influenza viruses, minimal risk was posed by manure from infected herds. The difference between conditions in confinement operations and those in backyard or village pig raising situations, in terms of the ability to clean the housing units, was noted. However, given that faecal shedding is not considered to be a major factor, it is believed that swine influenza viruses may be maintained in herds by naive pigs being introduced to the population. It is assumed that the pandemic (H1N1) 2009 virus will enter the pig and it will circulate among them like other swine influenza viruses.

Consensus statement regarding risk in the environment

The risk of exposure to pandemic (H1N1) 2009 virus from environmental sources, such as contaminated fomites and manure, is probably minimal, especially from pigs raised under confinement conditions.

³ Personal communication, 3 June 2009 Teleconference

III) Risks along the food-chain

What evidence is available regarding the survival of influenza viruses on meat surfaces?

The risk of cross contamination from respiratory secretions or from contact with respiratory organs/tissues to the meat, during slaughter and processing is very low. If it occurred, virus would be present in low concentrations. Since the highest concentration of virus would be in the lungs and respiratory tissue, and not in the intestinal tract, contamination of the meat surface would be less likely. It was noted that there are no data on concentrations or survival of swine influenza viruses on meat.

If evidence were to support the presence of influenza virus in raw meat, what evidence is available regarding the presence and concentrations of influenza viruses in raw meat or other by-products of swine infected with influenza viruses?

There is very little evidence of the virus being present in raw meat. If this did occur, the virus titres would probably be very low. Previous studies have found only a very minimal amount of virus in muscle [13, 14] and that would be readily destroyed by cooking, provided the temperature reaches 70°C. An Australian assessment found the risk of importing swine influenza infection in meat was low [15].

What evidence is available regarding the survival of influenza viruses in cured/dried/otherwise preserved pork or pork products?

It was noted that it is difficult to make general statements regarding cured/dried/otherwise preserved pork or pork products due to the many variations in food preparation and processing techniques. However, many of these products are tested and it has been shown that, in general, most processing methods can inactivate a variety of pathogens, many of which are less labile than influenza.

What evidence is available regarding potential human infection through ingestion of influenza virus (dose-response)?

There are no documented cases of human infection with swine influenza virus via ingestion. It was mentioned that, if ingestion were a viable route of influenza transmission, cases of human infection with highly pathogenic avian influenza (HPAI) H5N1 associated with consumption would have been expected to be more routinely reported, especially as poultry develop systemic infections, virus is found in the meat, and the birds are often slaughtered in home settings.

Animal studies in ferrets fed meat contaminated with HPAI virus became infected via the respiratory or digestive tracts depending on the virus strain [16]. In other studies and compared to the literature, a three log higher virus dose was required to infect chickens or ferrets via ingestion of infected meat verses inhalation⁴. The oral route is not the natural route of infection.

Consensus statement regarding risk along the food-chain

Available evidence suggests the risk of infection with swine influenza viruses from pork consumption is negligible. Ingestion is not the normal route of infection and the virus is readily destroyed by cooking at 70° C. The combination of multiple risk-reduction variables act together to decrease the risk to be insignificant under most conditions. These variables include: respiratory infection; short-lived infections; short-lived viraemia or organ contamination; the lack of faecal shedding (resulting in low cross contamination potential during slaughter and preparation); cooking pork at proper temperatures; and higher doses of contaminants/viruses required for infection via the gastrointestinal tract.

IV) Risks to swine from humans

What evidence is available regarding the probability of infected humans in contact with swine transmitting the infection to pigs?

In the past, there were several documented examples of influenza viruses, including H1N1 and H3N2, moving from humans into pigs. In some instances the virus has remained stable and spread to other swine herds, while in others the virus seems to have to die out. It was noted that among viruses studied in

⁴ D. Swayne, unpublished data

the USA, the genetic make-up of these viruses provides them with greater ability to adapt and change and they are becoming more promiscuous. With respect to the occurrence of H5N1 avian influenza virus, for the last ten years in China, Hong Kong Special Administrative Region (Hong Kong SAR) and elsewhere surveillance has rarely identified H5N1 in pigs.

Evidence of the pandemic (H1N1) 2009 virus moving to swine is a seemingly rare event since only one occurrence has been reported to date [11].⁵ Since we know this is possible, we need to know when it actually occurs. There is currently very little surveillance that would allow rapid detection of viruses crossing between humans and pigs (or other susceptible animals).

Consensus statement regarding risks to humans from swine

We can expect pandemic (H1N1) 2009 virus to move from humans to pigs. There was consensus that further surveillance to better understand what viruses are circulating in pigs and other animals is needed, but the design, implementation and funding sources for this surveillance are important issues requiring further discussion.

Proposed future actions for consideration:

- Discuss mechanisms, logistics and funding sources for improved surveillance in animals, not just pigs.
- Discuss mechanisms, logistics, funding sources and outline the goals of prospective sampling in swine facilities.
- Surveillance for ILI in pig farm workers.
- Persistence of the virus on fomites.

⁵ See footnote 2. At the time of the Consultation only one occurrence of transmission to pigs was known (Canada). Since we have evidence of the pandemic (H1N1) 2009 in swine in two other countries [17, 18] and in turkeys [19] in a third.

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Annex 1: List of experts

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Annex 2: Background paper on Inactivation of Influenza A viruses by Dr David Swayne, USDA, United States of America

Inactivation of Influenza A Viruses

David E. Swayne

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Influenza A viruses are relatively heat-labile enveloped RNA viruses in the family *Orthomyxoviridae*. All swine, avian, equine and most human influenza viruses are in the genus *Influenzavirus A* (4). They have similar chemical composition of RNA (1%), protein (70%), lipid (20%) and carbohydrates (5-8%). The lipid envelop is derived from the plasma membrane of the host cell which the virus has replicated and thus is susceptible to the same physical and chemical processes that destroy the cell membranes of animal cells. Disinfectants against microbes are licensed in the USA by the Environmental Protection Agency (EPA). Each product is licensed against all influenza A viruses because of the similarity in structure and physiochemical properties (<http://www.epa.gov/oppad001/influenza-disinfectants.html>). The host source (i.e. swine, avian, equine, human, etc.) and the subtype (e.g. H1N1, H5N1) do not change the basic chemical components and structure such that the inactivation properties are altered. This has been currently emphasized by the EPA for the 2009 Emergent H1N1 influenza A virus which would be inactivated by all disinfectants labeled and tested against influenza A virus.

For animal products, the highly pathogenic avian influenza viruses (HPAIV) causes severe viremia and systemic infection, and the virus is uniformly present in internal tissues including meat, and at high titers (9). However, infection of chickens with low pathogenic avian influenza viruses (LPAIV) causes a localized infection in respiratory and alimentary systems, and virus is not present in internal tissues including meat. On rare occasions, LPAIV has been demonstrated in oviducts of turkeys and chickens, but such identifications were of low titer and uncommon (5,13). Similarly, infections with swine influenza viruses are respiratory in nature and presumed to not be systemic (12), but studies have not been conducted to specifically look for virus in meat tissue. In a study looking at holding temperatures below freezing to inactivate influenza A virus in swine meat, the investigators artificially added influenza A virus to meat samples, suggesting that natural infection does not produce virus in meat or if present in meat would be of low titer and sporadic (6). In another study with respiratory infection of pigs with swine influenza virus, investigators identified virus in blood samples and sporadically in meat samples from several inoculated pigs (7), but because of the limited number of pigs and viruses tested and the lack of titer data, the conclusion of systemic infection, especially in meat is tenuous. Brown et al. reported finding swine influenza A virus in some serum samples (2). There have been no reports of influenza A virus isolation from swine meat following natural infection by swine influenza viruses (1,3). Finally, previous risk assessments for swine influenza A virus being transmitted through pig meat was very low (12).

Thermal processing (cooking or pasteurization) is a good method for inactivating influenza A viruses with most studies showing very efficient thermal inactivation HPAIV in meat from infected chickens (8,11). In several studies, thermal inactivation data for several H5N2 LP and HP AIV strains has been produced. Cooking and pasteurization are effective at killing these viruses and have similar inactivation curves. Using the inactivation data, mathematical models predicted complete inactivation of AIV in raw, skinless chicken meat cooked according to current USDA Food Safety and Inspection Service salmonella thermal inactivation guidelines. AIV inactivation at 70°C and 73.9°C were less than 5 seconds and 1 second, respectively, when using a maximal meat infection model (10). Similar results are expected for other influenza viruses.

In general, LPAIV, and in a similar matter swine influenza virus, are not expected to be present in meat from infected animals. However, virus from respiratory secretions or cross contamination from handling respiratory tissues could be a source of carcass surface contamination. This suggests that the amount of virus present on meat from properly eviscerated carcasses is quite low. As with virus titers in meat, the concentration of virus in respiratory secretions or feces is variable and depends on several factors. The volume of fluid that could accidentally contaminate the meat during slaughter or evisceration is also variable. However, the virus concentration in meat contaminated in this manner should be lower than that in meat from chickens infected with HPAIV.

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Annex 3: Background paper on influenza in swine by Dr Cate Dewey, University of Guelph, Canada

Influenza in Swine

Summarized by Cate Dewey, DVM, MSc, PhD
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Influenza A viruses cause clinical problems in pigs. The H1N1, H1N2 and H3N2 subtype viruses circulate widely in pig populations. The H1N1 is referred to as the classical swine influenza virus. H1N1 human influenza viruses are not well maintained in pig populations. However, human H3N2 influenza viruses can be maintained in pig populations. Genetic drift of these viruses in pigs is much less than in humans. It is believed that the introduction of a human H3N2 virus into the North American swine population was critical to the reassorted virus that caused disease in pigs over the past several years. This virus was first introduced into the Ontario swine population in 2005.

Avian H1N1 influenza viruses have been introduced and maintained in pig populations. In the late 1970's the avian H1N1 influenza virus became the dominant virus in the pig populations in Europe. After genetic shift and drift, avian-like H1N1 influenza viruses have spread from pigs to turkeys. Pigs in various parts of the world have antibodies against other non-H1N1, avian influenza viruses.

Pathophysiology

SIV causes an acute infection in pigs. Typically the virus is shed via nasal discharge one day after inoculation in experimental studies and stops by 8 days post inoculation. The virus cannot be recovered from other tissues of the respiratory tract 8 days post inoculation.

SIV specifically targets the bronchiolar epithelium. Early during the infection, most bronchi and bronchioles and many alveolar epithelial cells will contain virus before the pig clears the virus. A gram of lung tissue may contain 10^9 egg infectious dose (EID₅₀) of virus. However, by 2-3 days post inoculation, these lung virus numbers have declined. SIV replicates in the epithelial cells of the lungs, trachea, nasal mucosa, tonsils and tracheobronchial lymph nodes. Researchers have been largely unsuccessful in demonstrating extrarespiratory replication of the virus. The virus can be isolated from the nasal secretions of pigs while the pig is febrile. These can be collected using a nasal swab. In young pigs, the preferred sample is a pharyngeal swab because of the size of the nasal passage. In pigs that die or are euthanized during the acute phase of the illness, virus can be isolated from trachea and lung tissue.

The clinical signs of SIV in an individual pig include hyperthermia, anorexia, depression and reluctance to stand. These signs are due to the production of cytokines interferon- α , interleukin-1 and interleukin-6. The cytokines induce lung dysfunction and inflammation.

The severity of clinical signs is partially determined by the amount of virus that reaches the deep lung tissue. Infections in the upper respiratory tract result in mild or no clinical signs.

Diagnosis

Clinical signs of disease in a naive herd are strongly suggestive of SIV because of the rapid spread, the severity of clinical signs, the rapid spread of the disease, the fact that all age groups are affected and the low mortality.

Virus can be isolated from the nasal passages (using a nasal swab) when a pig is febrile.

Immunity

SIV invokes production of antibodies and a cell mediated immunity. The antibodies can be detected as early as 3 days post infection but can typically be measured 7 – 10 days after infection. They decline 8 – 10 weeks post infection. The pig completely eliminates the virus within one week post infection. Pigs will be protected against reinfection with the same or similar strain of the virus. Maternally acquired passive immunity lasts 4 – 14 weeks.

Experimental infections

Pigs can be experimentally infected via intratracheal (IT), aerosol and intranasal routes. However, only the IT route, using high doses of the virus ($>10^{7.5}$ EID₅₀) will reliably result in clinical signs and pathology due to SIV.

Epidemiology

SIV has historically been considered a disease of late fall and early winter. However, SIV does circulate in swine barns in all seasons of the year. This shift is likely due to the shift of pigs to indoor, confinement production.

The infection typically causes a herd outbreak of disease due to the rapid spread the virus within the barn. SIV typically causes interstitial pneumonia. Clinical evidence of disease occurs after an incubation period of only 1-3 days. Disease occurs in most animals and in all ages within the herd. Morbidity may reach 100% but mortality is less than 1%. The pigs will recover after 5 – 7 days with little or no long-term illness unless there are secondary respiratory infections.

The movement of pigs from one farm to another is responsible for the introduction of SIV into a naive herd. Infected pigs will shed 10^7 infectious particles per ml of nasal secretion at 2-5 days post exposure. Spread from one pig to another requires pig-to-pig contact. Pigs are infected via the nasopharyngeal route. In a naive herd, the virus will spread to all parts of the herd very rapidly.

If the herd is managed on a continuous flow basis (meaning that the barn is never empty), new, naive pigs will become infected over time. This enables the virus to circulate at low levels in the farm. Typically in this latter example, there will not be obvious clinical signs of the virus in the herd. The breeding herd and their offspring will maintain immunity against the virus. However, the virus may be maintained in the herd because of the purchase of breeding stock animals or the production of farm-born naive pigs. These will be either breeding stock gilts that are introduced to the herd or pigs that are born in the herd who lose maternal immunity at about 10 weeks of age. There is no carrier state for this virus in pigs. In these herds where the virus is maintained, there are little or no obvious clinical signs of disease. If nursery age pigs develop mild symptoms, they are hard to distinguish from a variety of other causes of respiratory illness in pigs. Testing finisher pigs, shortly before they are sent to market, show that many herds have some pigs that are serologically positive for influenza although the producer states that the herd had no outbreak of disease. It is therefore believed that the virus circulates in the herds without causing illness.

Prevention and Control

Vaccination and biosecurity are key components of a prevention program. Primary vaccination is given twice, two weeks apart and then sows are revaccinated biannually. Commercial vaccines contain both H1N1 and H3N2 subtypes.