FRI BRIEFINGS

Destruction of H5N1 Avian Influenza Virus in Meat and Poultry Products

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INTRODUCTION

Highly pathogenic avian influenza (HPAI) H5N1 virus was first detected in 1996 in domestic geese in southeast China but only became widely recognized the next year as it spread through live-bird markets in Hong Kong infecting many birds and eighteen humans, killing six. Office International des Épizooties (OIE) reports that HPAI H5N1 virus has now spread to domesticated birds in 44 countries and to wild birds in an additional 16 countries in Asia, Europe, and Africa. The largest numbers of outbreaks in poultry have been reported in Vietnam, Thailand, Egypt, Indonesia, and Turkey (*45*). As of 25 July 2007, 319 confirmed cases and 192 deaths among exposed people in 12 countries have been reported to the World Health Organization (*86*).

HPAI viruses cause systemic infections and have been detected in blood, bone, and breast and thigh meat of chickens (*68*). These viruses spread

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rapidly among chickens and turkeys and typically cause death within 48 hours. HPAI H5N1 viruses also infect ducks, but ducks may remain healthy even when virus is present in muscles and internal organs (*7*). This virus has also been detected in over 40 species of wild birds, and there is concern that migrating birds may facilitate the spread of this virulent avian flu strain. Despite attempts to control the spread of this virus, it may eventually reach the U.S. either in wild birds, illegally imported animals, meat from ducks or other preclinically infected poultry, or infected humans. Imported poultry products containing the H5N1 virus are believed to be the source of the 2007 outbreak in turkeys in England (*17*). Duck meat exported from China to Japan in 2003 (*41*) and from China to Korea in 2001 (*76*) was found to contain infective H5N1 virus. Poultry meat smuggled into the U.S. from China was destroyed by USDA in 2006. The smuggled meat was not tested for H5N1 but came from an area in China where the disease exists (*56*).

A major outbreak or even a few hundred cases of this strain of avian flu among poultry in the United States would present significant challenges to the poultry industry in terms of animal husbandry and ensuring the safety of meat and eggs as well as in responding to concerns of consumers. If the H5N1 virus were found to be transmissible to humans from meat and eggs of infected animals, then it will be important to identify effective methods for destroying this virus in foods. This White Paper reviews information on avian flu viruses and the extent of the ongoing world-wide outbreak and then focus on available information on destruction of the virus in meat and meat products. This information will enable AMI Foundation to provide timely information to its members and the public and to determine future research needs.

AVIAN INFLUENZA VIRUSES

Avian influenza (AI) viruses primarily infect birds and occasionally cause illness in humans in close contact with infected birds and in predatory animals, including eagles, tigers, dogs, and cats that consume sick or dead birds. Interspecies barriers to transmission of influenza viruses are generally strong, but some avian and human viruses have infected pigs and an equine virus has infected dogs fed horse meat. AI viruses contain RNA as their genetic material and are covered with an envelope containing lipids. These viruses are classified according to the structures of their hemagglutinin (H) protein and their neuraminidase (N) protein. Currently there are 16 types of H proteins and 9 types of N proteins. The H protein is cleaved by a host cell protease as a necessary part of the infection process. Some viruses cause severe illness and high mortality in chickens and turkeys and are referred to as high pathogenicity strains (usually H5 or H7) while others induce milder symptoms and are called low pathogenicity strains (H1–H16).

Influenza viruses that caused three important human pandemics in the past century, in 1957, 1968, and the infamous 1918 pandemic that caused about 40 million deaths, were derived from avian influenza viruses. The H1N1 strain causing the 1918 pandemic is believed to have been a high pathogenicity strain while the AI viruses involved in development of the other two pandemics originated from low pathogenicity strains. Several recent excellent reviews provide extensive information on AI viruses and their potential for causing human illness (*49;74;80*).

Low Pathogenicity Strains (LPAI)

Low pathogenicity avian influenza viruses can be detected in respiratory and gastrointestinal tissues of infected birds but scientific evidence indicates that they are not present in blood, muscle, or other tissues (*68;89*). The maturation of AI viruses requires the proteolytic processing of the H protein. The H protein of LPAI viruses contains one arginine at its cleavage site, and this is recognized only by proteases found in cells lining the respiratory and gastrointestinal tracts of birds (*43*).

Sporadic outbreaks of LPAI in domestic poultry occur in many countries, including the U.S., and even though this is a less severe form of the disease, nearly 5 million birds in infected and contact flocks were depopulated to control some outbreaks (*50*). Wild ducks, farm ducks, geese, gulls and other birds are commonly infected with LPAI strains, with 18% of black ducks in one study testing positive (*9;46;62*). An LPAI H5N1 avian influenza strain has been present in wild birds in the U.S. for at least 30 years. Generally

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these birds exhibit few symptoms although high concentrations of virus may be present in feces. LPAI has been isolated from untreated lake water where large numbers of water birds are living and water is an efficient vehicle for spreading infection among flocks (*80*).

Human infections with LPAIs have occurred almost exclusively among persons who have had close contact with domestic poultry during outbreaks. Mild to moderate symptoms have been reported, primarily conjunctivitis and flu-like illness (*2;29*). There is limited evidence that humans have become ill after contact with HPAI-infected wild birds (*26*) and some immunological evidence indicates that waterfowl hunters and wildlife professionals have been exposed to LPAI viruses without any serious effects (*25*).

High Pathogenicity Strains (HPAI)

Highly pathogenic AI viruses contain several basic amino acids at their hemagglutinin cleavage sites, and these are recognized by ubiquitous proteases found in many tissues throughout the body. Such viruses cause rapid and often severe or fatal infections in chickens and turkeys, and infective virus particles can be isolated from many tissues. Although HPAI typically causes death within a few days in an individual bird, it may take up to two weeks to become aware of an outbreak at a large poultry farm after a single introduction. If the detection rule is 50 dead birds on two consecutive days in a 10,000 chicken flock (current Dutch monitoring rule for notification), it was estimated that 12 days would elapse following introduction of one sick bird before an AI outbreak was recognized (*4*).

Prior to the recent HPAI H5N1 outbreak, only 24 outbreaks of HPAI have been reported worldwide in domestic birds since 1959 and most of these were limited geographically. A 2003 outbreak in Europe caused by H7N7 resulted in death or culling of nearly 31 million poultry and sickened at least 89 people. The most frequent human symptom was conjunctivitis rather than typical influenza-like illness (*51;80*). HPAI H7N3 outbreaks occurred in domestic poultry in Canada in 2004 and in Chile in 2002 (*57*). Other recent outbreaks of HPAI in Europe, Asia, Africa, and Australasia have been reviewed (*1*).

Three outbreaks of highly pathogenic avian influenza (not H5N1 strains) have been reported in poultry in the U. S. in: 1924 (H7 in east coast live bird markets); 1983–1984 (H5N2 in chickens, turkeys and guinea fowl in PA and VA); and 2004 (H5N2 in broiler chickens in Texas) (*50*). In all of the outbreaks, the virus was eradicated by euthanizing affected birds and their contacts and disinfection of premises. There was no apparent transmission to humans during these outbreaks (*78*).

In January 2007, an outbreak of HPAI H5N1 occurred on a turkey farm in Suffolk, England. Thousands of birds died within a few days and many more were euthanized to ensure the infection was stamped out. The H5N1 strain isolated from the turkeys was nearly identical to a strain that caused outbreaks in geese in Hungary during the same month. Epidemiological investigations could not pinpoint the exact sequence of events that resulted in transmission of the virus from Hungary but the one possible scenario involved importation of turkey meat from preclinically infected birds from Hungary to the processing plant adjacent to the affected turkey farm in England and transfer of some infected meat from the plant's waste bins to a turkey house by rodents and/or gulls. A few turkeys in this house consumed some scraps of infected meat, became ill, and passed the infection to other birds (*17*).

HPAI H5N1

The HPAI strain currently of concern in Asia, Europe, and Africa was first detected in a goose in southern China in 1996 and the following year caused disease and death in poultry and humans in Hong Kong (*19*). The disease was confined to Korea, China and southeast Asia through 2004 (except for Thai birds smuggled into Belgium). In 2005, following a large outbreak in wild birds in the Qinghai Lake area, western China, HPAI H5N1 was detected in several countries in eastern Europe and in quarantined birds in the UK. In 2006, HPAI H5N1 was first reported from Iraq, Nigeria, India, and several European and African countries. Five more countries have discovered this virus in local birds in 2007 including Bangladesh, Togo, Ghana, Kuwait, and Saudi Arabia. As of August 2007, the virus has been detected in a total of 60 countries (*45;49;53*). The role played by migratory birds and trade in poultry in disper-

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sion of this virus is under investigation (*18;21;22;24;32*). Although dissemination of avian viruses by birds appears to be a logical hypothesis, long distance migration is a very taxing endeavor and there is some doubt that wild birds could harbor HPAI viruses and still be capable of flying long distances. Wild birds may, however, spread this virus over shorter distances, as apparently occurred in Europe in 2006 (*82*).

HPAI H5N1 viruses have evolved over time and can now be categorized into three groups or clades. All Middle Eastern, African and European isolates are closely related and differ from some other isolates circulating in China and southeast Asia. This suggests a common origin of the strain of the virus introduced into Africa and Europe, possibly in Russia or at Qinghai Lake in China (*55;83*).

Avian influenza viruses are notorious for mutating frequently, and these mutations can affect host range of the virus, pathogenicity, resistance to drugs and to treatments intended to destroy the virus (*12;59*). For example, early isolates of HPAI H5N1 from chickens in China and Southeast Asia caused no illness or death in domestic ducks although ducks did harbor and shed the virus (*23;83*). However, a strain isolated from frozen duck meat exported from China in 2001 was able to spread systemically in ducks although it did not cause severe illness. Some strains isolated in 2002–2003 did cause mortality in ducks, and in 2005 over 6,000 migratory ducks, geese, and gulls at Qinghai Lake, China died from infection with an H5N1 virus (*8;47*). Ostriches are also susceptible to HPAI H5N1 (*14*). Susceptibility of other wild birds to HPAI H5N1 varies, with pigeons and some North American ducks (pintail, redhead, teal) resistant to these viruses while crows and wood ducks become ill (*5;66*). Some predatory and scavenger birds, including crested hawk eagles from Thailand (*79*) and hooded vultures in Burkina Faso (*20*), have become ill presumably after consuming infected birds.

Cats from several countries have become infected with HPAI H5N1 virus most likely after consuming infected birds (*33;60;87*) and experimental studies have confirmed that cats can indeed become infected after consuming infected meat (*34*). Death results from pneumonia and liver damage and virus was isolated from brain, liver, lung, kidney, spleen and intestines. Infective virus is excreted in feces and sputum (*54*) and cat-to-cat transmission of virus occurs. Horizontal transmission of HPAI H5N1 virus likely occurred among the 147 tigers that died at a Thai zoo. Although the first tigers affected had consumed raw infected chicken, other animals became sick after the feeding of raw poultry ceased (*70*). Subclinical infection with HPAI H5N1 in cats has been reported but these cats did not shed the virus (*35*).

A dog in Thailand died five days after eating duck carcasses infected with HPAI H5N1. Autopsy results demonstrated evidence of severe pneumonia and liver necrosis along with some kidney damage. Infective virus was detected in lungs, liver, kidney and urine (*61*). Experimental infection of dogs via the respiratory tract resulted in rapid seroconversion and shedding of virus from the nose in the absence of any clinical signs of disease (*40*). Ferrets, mice, hamsters, and cynomolgus macaques are also susceptible to HPAI H5N1 virus in experimental trials (*71*). These results indicate that the HPAI H5N1 virus can be transmitted to mammals through food and raise the questions of whether companion animals can spread this disease to humans and whether these animals could provide a host that allows this virus to better adapt to humans. As yet it appears that H5N1 does not spread rapidly among cats and dogs, and there are no reported cases of humans becoming sick after contact with these animals. But this potential mode of transmission to humans should be monitored.

People in close contact with infected poultry have become infected with HPAI H5N1 viruses with a total of 319 confirmed cases and 192 deaths reported from 12 countries. Subclinical infections may have occurred but this is believed to be rare (*81*). Viral transmission has primarily been from infected birds to humans, probably by inhalation of dust or water droplets containing the virus, but there have been a few cases where person-to-person transmission is believed to have occurred (*31;77*). This virus is apparently not readily transferred from poultry to people even when outbreaks have devastated small flocks in villages (*81*). There have been no confirmed reports of human infection from consuming infected poultry meat but gastrointestinal symptoms such as diarrhea have been observed in

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H5N1-infected patients in southeast Asia (*10;15*). Consumption of fresh duck blood and undercooked poultry has been implicated in some cases of human illness (*28*). So far this virus has not recombined or mutated into a form efficiently transmitted among humans but if it does, there are fears that it might cause a worldwide pandemic.

Infection Control in Birds

Biosecurity measures to prevent exposure of birds to HPAI H5N1 at poultry farms in Asia were described by Mike Robach. These include a broad range of measures to train workers, to isolate birds from potential carriers of infection (cats, dogs, wild birds, people, contaminated equipment and water), to effectively clean and disinfect premises, to destroy contaminants in feed by heating to 80ºC, and to ensure effective communication among supervisors, workers and veterinary personnel. Response plans have been developed for rapidly dealing with any indications of possible contamination or outbreak symptoms.

Another strategy for controlling HPAI in poultry flocks involves vaccination of at-risk birds. However, there is some controversy about this approach because of the concern that vaccinated birds may harbor and shed the virus without becoming clinically ill. It appears that in the field, vaccines do not completely prevent replication of AI viruses in the respiratory and gastrointestinal tract of poultry (*65*). In one set of experiments, treatment of chickens with a commercial vaccine based on an LPAI H5N2 strain prevented illness and death after challenge with HPAI H5N1 but some birds still shed infective virus (*69*). Other researchers have reported that vaccinated birds do not shed AI viruses and that no infective virus was present in meat (*3;42*). Vaccination should be combined with an effective biosecurity program.

HPAI VIRUSES IN FOOD AND METHODS FOR CONTROL

Presence in Meat

HPAI viruses cause systemic infections and are present in skeletal muscles and many internal organs of infected birds. Titers of HPAI H5N1 strains in chicken thigh and breast meat were reported to be $10^{6.8}$–$10^{8.0}$ and $10^{5.5}$–$10^{7.9}$ EID_{50}(median infectious dose)/g, respectively while titers for an HPAI H5N2 strain in chicken thigh and breast meat were reported to be $10^{2.8}$ and $10^{2.3}$ EID_{50}/g, respectively. HPAI H5N1 titers in thigh meat of sick ducks were reported as $10^{4.0}$–$10^{6.0}$ EID_{50}/g and in infected but clinically normal ducks as $10^{2.0}$–$10^{3.4}$ EID_{50}/g (*64;68;72*).

Presence in Eggs

HPAI viruses have been detected on the outside surface of eggs, perhaps as a result of fecal contamination (*67*). HPAI H5N1 viruses and RNA were detected in fluids used to wash duck and goose eggs smuggled from Vietnam into China (*36*). One issue related to surface contamination of shells is the possibility that virus particles may pass through the shell into an egg. Since the avian influenza virus is about 100 nm in diameter and pores in the egg shell are 200–600 nm in diameter, this could occur. A similar size virus, fowl plague virus, is known to penetrate intact eggs (*85*).

During an HPAI outbreak in northeastern U.S. in 1983–1984, infective virus was detected in albumen and yolk samples as well as on egg shell surfaces of up to 57% of eggs laid within 18 days after appearance of clinical signs (*67*). In experimental studies, HPAI H7N1 was detectable in eggs from broiler chickens within 3 days after experimental nasal infection (*63*) and HPAI H5N2 was present in 85–100% of chicken eggs laid 3–4 days post-infection with titers as high as $10^{4.9}EID_{50}$ /mL (*67*). H5N1 was also present in eggs and oviducts of naturally infected quail, with titers of $10^{4.6}$–$10^{6.2}$ EID_{50} /mL (*52*). In contrast, LPAI viruses have not been detected in eggs (*67*).

Survival of an LPAI H5N2 virus experimentally introduced into egg yolk and albumen and onto egg shells has been measured at storage temperatures of 4–20ºC. Survival was inversely related to temperature and yolk was the best medium for survival with virus detectable in yolk at all temperatures for up to 17 days. After 3–4 hours of drying on the egg shell surface, this virus was not detectable at any temperature (*16*).

Cooking and Heat Treatment

Meat. A reproducible microassay using small pieces of meat was developed to measure thermal inactivation of HPAI H5N1 in naturally infected poultry. No infective virus was isolated from the

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meat after it changed from pink-tan to white color and was exposed to 70ºC for 1 sec (*64*). Using this assay, survival curves were constructed for H5N1 virus in chicken meat exposed to 57–61ºC. From experimental data, conservative D values (dose required to reduce virus titers by one log) at temperatures of 57, 58, 59, 60, 61, and 70ºC were estimated as 321.1, 195.4, 118.9, 72.4, 44.0, and 0.5 sec, respectively (*72*). A 4 log reduction in HPAI H7N7 titer in cell culture occurred after exposure to 63ºC for 90 sec (*30*).

Eggs. Cooking eggs to a temperature of 160ºF (71ºC) is reported to kill avian influenza viruses (*85*). In experimental studies, LPAI viruses in eggs appear to be more susceptible to heat than HPAI viruses. Pasteurization of liquid whole eggs, liquid egg whites and 10% salted yolk to industry standards of time/temperature inactivates HPAI H5N2 viruses. However, virus remained infective after the standard protocol of 54.4ºC for 7–10 days for dried egg white. Treatment at 67ºC for 15 days would inactivate HPAI virus and preserve quality of egg products (*67*).

Irradiation

The small size and low moisture content of viruses ensures that they are generally more resistant to irradiation than bacteria, including spores. Poultry in the U.S. may be irradiated to a dose of 3 kGy (kiloGrays). There is not yet any published data on the sensitivity of AI viruses in meat or eggs to irradiation. Data on other foodborne viruses (hepatitis, polio, rotavirus) tested in fish, shellfish or beef indicate that D values range from 2 to 10 kGy (*48*). Tests with Newcastle Disease virus (similar in structure and size to AI viruses) in "egg fluid" reported a D value of 2 kGy (*73*) and exposure of two influenza viruses (H3N2 and H1N1) in culture fluid to irradiation also yielded D values of 2 kGy (*37*). Therefore, complete inactivation of AI viruses in poultry meat (reported to be in the range of about 10^6–$10^{8.0}$ EID_{50}/g) and eggs would not be accomplished under approved radiation levels.

High Pressure

Inactivation of viruses by pressure depends on the quantity of pressure (MPa, megaPascals), temperature, and other solutes in the suspending medium. In tests with an HPAI H7N7 virus suspended in chicken meat, 25 seconds at 15ºC and 500 MPa induced a 5 log reduction in virus titers. A 1.5 log reduction was caused in one minute by exposure to 400 MPa at 15ºC or at 300 MPa at 30ºC (*30*).

Results for other foodborne viruses indicate a range in susceptibility: (a) a seven log reduction for hepatitis A virus following 5 min exposure to 450 MPa at 22ºC; (b) an 8 log reduction in rotavirus following exposure to 300 MPa for 2 min at 25ºC; and (c) <1 log reduction of poliovirus following 60 min exposure to 600 MPa at 20ºC (*27*).

HPAI VIRUSES IN THE ENVIRONMENT AND METHODS FOR CONTROL

A recent comprehensive review on inactivation of avian influenza viruses presents information on the efficacy of a variety of chemical agents and physical conditions (*13*). Much of the experimental work has not used HPAI viruses for testing because of the stringent requirements for working with these pathogens. Viral strains vary in their susceptibility to different treatments and so results presented for LPAI strains are approximations of what would be expected for HPAI strains. In addition, it is important to remember that the efficacy of a particular treatment will depend on other factors in the environment that may make the virus more or less sensitive to heat, pH, irradiation, chlorine, etc.

Survival in / on Environmental Samples

Water. Ducks shed high levels of LPAI viruses in feces into surface waters, and it appears that infection cycles are maintained in wild aquatic birds by long-term persistence of these viruses in water and ingestion by other ducks during feeding (*62*). Recent experiments with two HPAI H5N1 strains and eight LPAI viruses demonstrated that persistence in distilled water was significantly related to salinity and temperature. Under most conditions the H5N1 strains did not remain infective for as long as the LPAI strains, but estimated survival was as long as 158–182 days at 17ºC and 28 days at 28ºC (*6*). Shorter survival times of an LPAI H5N2 virus were observed in other experiments (*39*). Natural waters would also contain organic matter and

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microbes which may also affect survival. Some data on survival of AI viruses in surface waters collected in Bulgaria indicate that AI viruses do not persist as long in water containing microorganisms (*88*).

Chicken manure. Chicken manure is used as fertilizer for some crops in Asia and there has been concern that virus could survive in manure and contaminate some vegetables, for example, onions. Experiments conducted in Thailand demonstrated that H5N1 virus mixed with dry manure was inactivated within one day at 25ºC and more rapidly at higher temperatures (*11*). Survival of an LPAI H7N2 strain was tested in chicken manure at several temperatures. Virus mixed with manure was inactivated in 15 min at 56ºC, 23 days at 15–20ºC and survived even longer at refrigeration temperatures (*38*). Survival appears to be longer in moist environments but it may be that, as demonstrated in experiments in water above, the HPAI N5N1 virus is not as well adapted to survive in the environment as some LPAI viruses.

Surfaces. An LPAI virus (H13N7) survived at least three days at room temperature on nine surfaces tested (steel, tiles, gum boot, tire, egg shell, and plastic) but was not detectable on egg trays (cardboard) or cotton or polyester fabric at two days or on wood at three days. Virus remained infective on feathers and latex for at least six days. Lower survival on porous surfaces may be due in part to the difficulty in recovering and enumerating viruses that lodged in pores of the material (*75*).

Culture media. LPAI viruses were reported to survive more than 15 days in culture media at room temperature (*75*) and more than eight months at refrigeration temperatures (*38*).

Inactivation / Decontamination Methods

AI viruses are of intermediate size and are covered with a lipid envelope. They are relatively unstable in the environment, being susceptible to heat, pH extremes, and dryness. Cool moist conditions and organic matter can extend viability of these viruses.

Chlorine and other sanitizers. Chlorine and other sanitizers can inactivate viruses as well as bacteria but their effectiveness depends on temperature, viral concentration, and removal of organic matter and dirt that bind these disinfectants, reducing their effect on pathogens. Inactivation of LPAI H5N2 was found to be variable depending on the medium but did occur at final free chlorine values of >8 mg.min/L (*39*). Some disinfectants and 70% ethanol inactivated LPAI viruses within 15 minutes of exposure (*38*).

Soaps and detergents have not been specifically tested against AI viruses but are expected to be effective because they would disrupt the lipid envelope (*13*).

Acid or base treatment. AI viruses are apparently more sensitive to acids than bases: Five minutes of exposure to pH 2 completely inactivated H7N2 while exposure to pH 10 or 12 for fifteen minutes had no effect on infectivity (*38*).

Ultraviolet light. Some reports indicate that UV light cannot efficiently inactivate AI viruses requiring 45 min of exposure to destroy an HPAI H7N3 virus in peptone water (*13*). However when suspended in phosphate buffer or wastewater, LPAI H5N2 could not survive a UV fluence of >10 mJ/cm^2 (which is significantly less than the exposure used in wastewater treatment plants that utilize UV disinfection) (*39*). UV light cannot effectively destroy viruses that are mixed into manure because the solid particles shield many viruses (*11*).

Copper. Copper and copper alloys have been shown to have antibacterial properties. *E. coli* O157:H7 cells spread on a copper or alloy metal surface (initial load 5 x 10^7 cfu) were completely inactivated within six hours at 4–20ºC. When inoculated on to stainless steel, these bacteria can survive more than four weeks (*84*). Copper was also found to rapidly inactivate a human influenza virus when the virus was spread on a copper surface (initial titer 10^7 particles) and incubated at 22ºC, 50–60% humidity (*44*).

Anaerobic digestion in a wastewater treatment plant inactivates AI viruses within 3 days (*39*).

Composting of poultry carcasses infected with HPAI H5N2 with oat straw and goat manure was found to inactivate AI viruses within 10 days (*58*).

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Summary

HPAI viruses can infect a variety of birds but generally cause the most devastating symptoms in chickens, turkeys, and other gallinaceous birds (quail, partridge, pheasant). These birds often die within a few days of exposure. Although HPAI viruses are widespread in the tissues and probably in eggs of sick birds, it is unlikely that infected chicken or turkey meat or eggs will be offered for sale commercially on a large scale both because of biosecurity measures instituted by farmers and producers and the fact that these birds rapidly succumb to infection. Nevertheless, the possibility that some birds may have a subclinical infection cannot be completely discounted. There is the puzzling outbreak among turkeys in England in January 2007, which is thought to be caused by importation of turkey meat from subclinically infected birds from Hungary.

Ducks appeared to be resistant to early strains of HPAI H5N1 but some more recently evolved stains are more pathogenic. Clinically normal, infected ducks contain virus in their skeletal muscles although reported virus titers are lower than those found in infected, symptomatic ducks. Duck meat exported from China to Japan and Korea has been found to contain infective H5N1 virus so infected duck meat has already entered the human food chain.

It is thought that most human infections have resulted from inhalation of virus while raising or slaughtering infected poultry. However in a number of cases in southeast Asia, patients have experienced gastrointestinal symptoms, suggesting that they may have been infected orally. Certainly, several predator mammals (dogs, cats, martens, tigers) have acquired HPAI by consuming raw infected birds. There are also a few human cases where person–person spread is believed to have occurred.

Considering the unlikely possibility that infected birds would be slaughtered for meat or that eggs from infected birds would be collected, is there a food safety issue? Standard conditions for cooking eggs and poultry meat have been shown to destroy this virus with the apparent exception of production of dried eggs. High pressure and heat can also inactivate this virus but irradiation is not likely to be effective at approved doses. Different strains of these viruses vary somewhat in sensitivity to these control methods, but any cooking method that destroys *Salmonella* or *Campylobacter* should also inactivate HPAI H5N1.

Although HPAI H5N1 virus is not as hardy as some other viruses, it has been shown to remain infective for several days on surfaces at ambient temperatures and for weeks or months in water. Survival is even longer in the refrigerator. AI viruses have a lipid coat that makes them susceptible to detergents and various sanitizers including chlorinated compounds. UV light may be effective in inactivating the viruses under some conditions and the viruses are also neutralized under acidic conditions. Precautions to inactivate virus on cutting boards and equipment and in chilling tanks will prevent cross-contamination from infected birds.

HPAI viruses should not be a threat to food safety if: (a) meat and eggs are properly cooked and (b) persons involved in food preparation do not cross-contaminate vegetables, fruit, or other ready-to-eat foods (that will not be cooked) with infected meat.

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