



**HIGHLY PATHOGENIC AVIAN INFLUENZA  
STANDARD OPERATING PROCEDURES:  
1. OVERVIEW OF ETIOLOGY AND ECOLOGY**

**FAD PReP**

**Foreign Animal Disease  
Preparedness & Response Plan**



**United States  
Department of  
Agriculture**

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The Foreign Animal Disease Preparedness and Response Plan (FAD PReP) Standard Operating Procedures (SOPs) provide operational guidance for responding to an animal health emergency in the United States.

These draft SOPs are under ongoing review. This document was last updated in **September 2015**. Please send questions or comments to:

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**FAD PreP**  
 Foreign Animal Disease  
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# Contents

1.1	Introduction .....	1-2
	1.1.1 Further Information.....	1-2
	1.1.2 Goals .....	1-2
1.2	Purpose.....	1-2
1.3	Etiology.....	1-3
	1.3.1 Name .....	1-3
	1.3.2 Virus Characteristics .....	1-3
	1.3.3 Morphology .....	1-3
	1.3.4 Genus Characteristics.....	1-3
	1.3.5 Influenza A Virus Subtypes .....	1-4
	1.3.6 Influenza A Virus Identification.....	1-4
	1.3.7 Currently Circulating HPAI Viruses.....	1-4
1.4	Ecology .....	1-5
	1.4.1 Susceptible Species .....	1-5
	1.4.2 Reservoirs .....	1-5
	1.4.3 Introduction and Transmission of HPAI Virus .....	1-6
	1.4.4 Incubation and Infectious Periods .....	1-6
	1.4.5 Morbidity and Mortality in Birds .....	1-7
	1.4.6 Human and Animal Influenza Viruses.....	1-7
	1.4.7 Antigenic Shift and Antigenic Drift.....	1-7
1.5	Environmental Persistence of AI Viruses .....	1-8
	1.5.1 In Eggs and Egg Products .....	1-9
	1.5.2 In Meat .....	1-10
	1.5.3 In Poultry Byproducts and Waste .....	1-10
	1.5.4 In Carcasses .....	1-11
1.6	Threat of HPAI to the United States.....	1-11
	Attachment 1.A References and Resources.....	1-13
	Attachment 1.B Abbreviations .....	1-18

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## **Highly Pathogenic Avian Influenza (HPAI)**

### **Etiology and Ecology Quick Summary**

#### **Disease**

Highly pathogenic avian influenza (there is also low-pathogenic avian influenza [LPAI]), also known as fowl plague.

#### **Mortality and Morbidity**

HPAI can produce high mortality and morbidity in domestic poultry.

#### **Susceptible Species**

Many susceptible species: chickens, turkeys, ducks, geese, pheasants, dogs, swine, other mammals, and some non-mammalian species.

#### **Zoonotic Potential (yes/no)?**

Yes.

#### **Reservoir**

Migratory waterfowl are a key natural reservoir of LPAI, but there is no evidence of HPAI generation in reservoir hosts to-date.

#### **Transmission**

Direct exposure to infected birds, excrement, or other secretions. Poultry generally become infected thru direct or indirect contact with waterfowl. HPAI occurs following transmission of LPAI strains from reservoirs into poultry followed by virus mutation to high pathogenicity.

#### **Persistence in the Environment**

Viable in cold and humid environments for longer periods.

#### **Animal Products and By-Products**

Virus can persist in poultry meat products; for example, meat needs to be heated to at least 70 °C for 3.5 seconds.

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## 1.1 Introduction

Highly pathogenic avian influenza (HPAI) is a highly infectious viral disease that affects a wide range of bird species. Clinical signs of the disease depend on the strain and subtype of virus and the species of bird infected. Avian influenza (AI) is classified according to disease severity, with two recognized forms: HPAI, also known as fowl plague, and low pathogenicity avian influenza (LPAI). AI viruses that cause HPAI are highly virulent, and mortality rates in infected domestic flocks often approach 90–100 percent. LPAI viruses exhibit variable degrees of pathogenicity in domestic poultry.

In the 2014–2015 HPAI outbreak in the United States, H5N2 was the most common subtype of HPAI followed by H5N8; the outbreak resulted in the loss of nearly 50 million birds. Initially introduced through wild waterfowl, H5N2 adapted and ultimately spread rapidly through domestic poultry flocks in the Midwest.

However, HPAI can affect other mammal species and is zoonotic: HPAI H5N1 has infected humans and caused fatalities. As of July 17, 2015, there have been 449 human deaths, and over 844 human cases, reported as a result of laboratory confirmed HPAI H5N1.<sup>1</sup> While HPAI has a relatively high species-specific transmission barrier, other strains have also infected humans. No HPAI infections in humans (including responders to the incident) were reported during the 2014–2015 HPAI outbreak in the United States.

### 1.1.1 Further Information

In addition to this *HPAI Standard Operating Procedure (SOP): Overview of Etiology and Ecology*, information can also be found in the *HPAI Response Plan: The Red Book*. This document is intended to be an overview, focusing on HPAI in domestic poultry. These documents are available on the APHIS website: [www.aphis.usda.gov/fadprep](http://www.aphis.usda.gov/fadprep). Additional resources on HPAI are listed in [Attachment 1.A](#).

### 1.1.2 Goals

As a preparedness goal, the Animal and Plant Health Inspection Service (APHIS) will provide etiology and ecology summaries for HPAI and update these summaries at regular intervals.

As a response goal, the Unified Command and stakeholders will have a common set of etiology and ecology definitions and descriptions, to ensure proper understanding of HPAI when establishing or revising goals, objectives, strategies, and procedures.

## 1.2 Purpose

The purpose of this document is to provide responders and stakeholders with a common understanding of the disease agent.

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<sup>1</sup> World Health Organization (WHO). “Cumulative Number of Confirmed Cases of Avian Influenza A/(H5N1) Reported to the WHO.” 2015. [http://www.who.int/influenza/human\\_animal\\_interface/EN\\_GIP\\_20150717\\_cumulativeNumberH5N1cases.pdf](http://www.who.int/influenza/human_animal_interface/EN_GIP_20150717_cumulativeNumberH5N1cases.pdf).

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## 1.3 Etiology

### 1.3.1 Name

H5N1 is also referred to as fowl plague and grippe aviaire.<sup>2</sup> It is caused by influenza virus A.

### 1.3.2 Virus Characteristics

According to the International Committee on Taxonomy of Viruses, this disease has the following characteristics:

- Family: *Orthomyxoviridae*
- Genera, five genera contain only one species:
  - Influenza virus A
  - Influenza virus B
  - Influenza virus C
  - Thogotovirus
  - Isavirus
- Baltimore Classification: Group V (–) ssRNA.

Influenza A virus is further classified on the basis of the surface glycoproteins, hemagglutinin (HA or H) and neuraminidase (NA or N). Sixteen H subtypes and nine N subtypes of influenza A virus have been identified.

### 1.3.3 Morphology

The influenza A virus particle or virion is 80–120 nanometers in diameter and usually spherical, although filamentous forms may occur. The influenza A genome is unusual as it contains eight pieces of segmented, single stranded negative-sense ribonucleic acid (RNA).

### 1.3.4 Genus Characteristics

This genus has the following characteristics:

- It consists of a single species: influenza A virus.
- The multipartite genome is encapsulated, and each segment is in a separate nucleocapsid. Eight different segments of negative-sense single-stranded RNA are present; this allows for both autologous and heterologous genetic reassortment and may result in novel strains that are divergent from the initial infecting strain.
- The genome consists of 10 genes encoding for different proteins (eight structural proteins and two nonstructural proteins). These include the following: three transcriptases (PB2,

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<sup>2</sup> Center for Food Security and Public Health (CFSPH), Iowa State University & Institute for International Cooperation in Animal Biologics. “High Pathogenicity Avian Influenza.” 2014. [http://www.cfsph.iastate.edu/Factsheets/pdfs/highly\\_pathogenic\\_avian\\_influenza.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/highly_pathogenic_avian_influenza.pdf).

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PB1, and PA), two surface glycoproteins (HA and NA), two matrix proteins (M1 and M2), one nucleocapsid protein (NP), and two nonstructural proteins (NS1 and NS2).

- The virus envelope glycoproteins (HA and NA) are distributed evenly over the virion surface, forming characteristic spike-shaped structures. Antigenic variation in these proteins is used as part of the influenza A virus subtype definition (but not used for influenza B or C viruses).

### 1.3.5 Influenza A Virus Subtypes

The subtypes have the following characteristics:

- There are 16 different HA antigens (H1 to H16) and nine different NA antigens (N1 to N9) for influenza A.
- Some influenza A subtypes can cause LPAI; H5 and H7 subtypes include both HPAI and LPAI strains.
- Low pathogenicity viruses typically result in few clinical signs in domestic poultry; highly pathogenic viruses generally cause severe clinical signs and results in high mortality in domestic poultry.
- Genetic characterization of HPAI H5N1 strains involved in the current panzootic has demonstrated multiple distinct phylogenetic clades.<sup>3</sup>

### 1.3.6 Influenza A Virus Identification

Subtypes are classified into strains, which are described by a number of characteristics, including type, host, place of first isolation, strain number, year of isolation, and antigenic subtype. For example, a description of a strain would be A/chicken/Hong Kong/y385/97(H5N1).<sup>4</sup> Typically, the host is omitted for human strains.

### 1.3.7 Currently Circulating HPAI Viruses

Data reported to the World Organization for Animal Health (OIE) in 2014–2015 provides information of ongoing HPAI circulation as follows:

- H5N1–Africa, Bulgaria, Canada, China, India, the Middle East, Russia, South East Asia, and the United States;
- H5N2–Canada, China, and the United States;
- H5N3–China;
- H5N6–China and Vietnam;
- H5N8–Canada, China, Europe, Japan, Russia, the United States, and the United Kingdom;

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<sup>3</sup> World Organization for Animal Health (OIE). Highly Pathogenic Avian Influenza, Technical Disease Card. 2014. [www.oie.int](http://www.oie.int).

<sup>4</sup> CFSPH, Iowa State University & Institute for International Cooperation in Animal Biologics. “High Pathogenicity Avian Influenza.” 2014. [http://www.cfsph.iastate.edu/Factsheets/pdfs/highly\\_pathogenic\\_avian\\_influenza.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/highly_pathogenic_avian_influenza.pdf).

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- H7N2–Australia;
  - H7N3–Mexico; and
  - H7N7–the United Kingdom.<sup>5</sup>

## 1.4 Ecology

### 1.4.1 Susceptible Species

Many avian species are susceptible to infection with HPAI viruses, including:

- chickens,
- turkeys,
- ducks,
- geese,
- guinea fowl, and
- a wide variety of other birds, including migratory waterfowl and shorebirds.

HPAI has primarily been isolated from chickens and turkeys.<sup>6</sup> Psittacine birds (such as parrots and cockatiels) are more rarely affected. Mammalian hosts, including humans, may be vulnerable to infection by some AI strains, including HPAI H5 and HPAI H7 subtypes.

Infection in birds can give rise to a wide variety of clinical signs that may vary according to the host, strain of virus, host's immune status, presence of any secondary exacerbating organisms, and environmental conditions. Preliminary diagnosis is made through serology or molecular probe (for example, a real-time reverse transcriptase polymerase chain reaction [rRT-PCR]). Diagnostic confirmation is done by isolation and characterization of the virus through sequencing.

### 1.4.2 Reservoirs

AI viruses usually infect migratory waterfowl, particularly Anseriformes (ducks and geese) and Charadriiformes (shorebirds) that can carry LPAI viruses without showing illness. Infection rates in these populations peak at autumn migratory staging locations, where large numbers of immunologically naive juvenile birds congregate.<sup>7</sup> LPAI virus strains occur worldwide, and have been isolated from more than 100 different species of birds.<sup>8,9</sup> The wild bird reservoir of LPAI viruses is considered a major potential source of infection for domestic birds, particularly

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<sup>5</sup> OIE. "Update on Highly Pathogenic Avian Influenza in Animals (Type H5 and H7)." 2014–2015. <http://www.oie.int/animal-health-in-the-world/update-on-avian-influenza/>.

<sup>6</sup> OIE. Highly Pathogenic Avian Influenza, Technical Disease Card. 2014. [www.oie.int](http://www.oie.int).

<sup>7</sup> Hall, Jeffrey S., Robert J. Dusek, and Erica Spackman. "Rapidly Expanding Range of Highly Pathogenic Avian Influenza Viruses." *Emerging Infectious Diseases* 21, no. 7 (2015): 1251-252.

<sup>8</sup> Swayne, David E. "Epidemiology of Avian Influenza in Agricultural and Other Man-Made Systems." *Avian Influenza*, (2015): 59-85. doi:10.1002/9780813818634.ch4.

<sup>9</sup> Olsen, B. "Global Patterns of Influenza A Virus in Wild Birds." *Science* 312, no. 5772 (2006): 384-88.



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free- and open-range poultry. Following transmission from wild to commercial birds, the virus can mutate or reassort in gallinaceous poultry flocks, resulting in an HPAI virus.<sup>10</sup>

There is not currently evidence of HPAI generation in a reservoir. However, HPAI viruses, particularly HPAI H5N, have occasionally been isolated from free living wild birds, suggesting that dissemination of HPAI by wild waterfowl may be possible.<sup>11</sup> It remains unclear how long and to what extent wild birds can maintain HPAI viruses, but there is evidence that certain species of Anseriformes can carry and shed certain HPAI viruses without clinical signs.<sup>12</sup> This poses a serious transmission risk to commercial poultry.

### 1.4.3 Introduction and Transmission of HPAI Virus

Contact with infected wild birds or their secretions is a common mode of introduction of AI into a poultry population. Because of the LPAI reservoir in wild birds, and the ability of this influenza virus to mutate or reassort into an HPAI strain, minimizing contact between domestic birds and wild birds is fundamental to preventing HPAI infections. There is strong evidence that the 2014–2015 outbreak in the United States was introduced from wild birds to poultry. Additionally, live poultry markets have been documented as a source of introduction and further dissemination of both LPAI and HPAI in past outbreaks both in Asia and the Americas.

HPAI virus is usually transmitted via direct exposure to HPAI infected birds, feces, or secretions from infected birds. Transmission of the virus can also result from movement of contaminated fomites, including by people, on contaminated clothing, equipment, and vehicles. Airborne transmission is not likely a primary mode of transmission, although it may occur over short distances as an aerosol.<sup>13,14</sup> When a hen is infected, the HPAI virus is also likely to be present on the eggshell and internal egg contents, though to date, there is no evidence demonstrating vertical transmission of the virus.<sup>15</sup> Transmissibility can also vary by HPAI strain.<sup>16</sup>

HPAI can be transmitted mechanically by invertebrate and vertebrate vectors. However, there is no evidence to suggest that invertebrates are involved in biological transmission.

### 1.4.4 Incubation and Infectious Periods

Incubation periods for HPAI are variable. Typically, the incubation period in naturally infected chickens ranges from 3 to 14 days.<sup>17</sup> The OIE *Terrestrial Animal Health Code* (2015) gives the

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<sup>10</sup> OIE. Highly Pathogenic Avian Influenza, Technical Disease Card. 2014. [www.oie.int](http://www.oie.int).

<sup>11</sup> Gaidet, N., et al. “Evidence of Infection by H5N2 Highly Pathogenic Avian Influenza Viruses in Healthy Wild Waterfowl.” *PLoS Pathogens*, (2008). doi:10.1371/journal.ppat.1000127.

<sup>12</sup> Gaidet, N., et al. “Evidence of Infection by H5N2 Highly Pathogenic Avian Influenza Viruses in Healthy Wild Waterfowl.” *PLoS Pathogens*, (2008). doi:10.1371/journal.ppat.1000127.

<sup>13</sup> United Nations (UN) Food and Agriculture Organization (FAO). “Questions and Answers: The Facts of Bird Flu.” 2015. <http://www.fao.org/avianflu/en/qanda.html>.

<sup>14</sup> USDA APHIS VS. “Epidemiologic and Other Analyses of HPAI-Affected Poultry Flocks: June 15, 2015 Report.” 2015. [http://www.aphis.usda.gov/animal\\_health/animal\\_dis\\_spec/poultry/downloads/Epidemiologic-Analysis-June-15-2015.pdf](http://www.aphis.usda.gov/animal_health/animal_dis_spec/poultry/downloads/Epidemiologic-Analysis-June-15-2015.pdf).

<sup>15</sup> OIE. Highly Pathogenic Avian Influenza, Technical Disease Card. 2014. [www.oie.int](http://www.oie.int).

<sup>16</sup> Swayne, David E. “Epidemiology of Avian Influenza in Agricultural and Other Man-Made Systems.” *Avian Influenza*, (2015): 59-85. doi:10.1002/9780813818634.ch4.

<sup>17</sup> Swayne, David E. “Epidemiology of Avian Influenza in Agricultural and Other Man-Made Systems.” *Avian Influenza*, (2015): 59-85. doi:10.1002/9780813818634.ch4.

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incubation period for AI as 21 days.<sup>18</sup> HPAI may have longer incubation periods in some species than others, and the length of the incubation period will vary depending on the dose of the virus, transmission pathway, and other environmental factors.<sup>19</sup>

It is possible for a bird to shed the virus before and after the appearance of clinical signs. The infectious period is particularly important in outbreak response measures because some species, such as domestic ducks, may not show clinical signs so the incubation period may not be known.<sup>20</sup>

#### 1.4.5 Morbidity and Mortality in Birds

HPAI often causes morbidity and mortality rates in domestic poultry between 90–100 percent. Ducks and geese infected with HPAI do not usually show clinical signs. Wild birds typically do not experience mortality and are usually asymptomatic when infected with HPAI.<sup>21</sup>

#### 1.4.6 Human and Animal Influenza Viruses

Influenza viruses are usually adapted to a particular host species. HPAI is zoonotic, and although it appears to have a relatively high species-specific transmission barrier, specific HPAI subtypes, including H5N1, have been demonstrated to infect and be fatal to humans under certain circumstances. In total, approximately 1,500 cases of AI have been documented in humans in the last 50 years.<sup>22</sup> Key, currently circulating zoonotic strains of HPAI include H5N1 and H5N6;<sup>23</sup> occasional cases of HPAI H7N7 have also been reported.<sup>24</sup> Most of these cases had a clear epidemiological link to direct contact with live, infected poultry.<sup>25</sup>

#### 1.4.7 Antigenic Shift and Antigenic Drift

The reservoirs for all 16 H and 9 N influenza viruses are wild birds.<sup>26</sup> In wild waterfowl, the H and N subunits appear to be stable.<sup>27</sup> However, influenza A viruses can evolve through both

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<sup>18</sup> OIE. Article 10.4.1: General Provisions, Terrestrial Animal Health Code. 2015. [www.oie.int](http://www.oie.int).

<sup>19</sup> Swayne, David E. “Epidemiology of Avian Influenza in Agricultural and Other Man-Made Systems.” *Avian Influenza*, (2015): 59-85. doi:10.1002/9780813818634.ch4.

<sup>20</sup> Swayne, David E. “Epidemiology of Avian Influenza in Agricultural and Other Man-Made Systems.” *Avian Influenza*, (2015): 59-85. doi:10.1002/9780813818634.ch4.

<sup>21</sup> CFSPH, Iowa State University & Institute for International Cooperation in Animal Biologies. “High Pathogenicity Avian Influenza.” 2014. [http://www.cfsph.iastate.edu/Factsheets/pdfs/highly\\_pathogenic\\_avian\\_influenza.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/highly_pathogenic_avian_influenza.pdf).

<sup>22</sup> Swayne, David E., and Colleen Thomas. “Trade and Food Safety Aspects for Avian Influenza Viruses.” *Avian Influenza*, (2008): 499-512.

<sup>23</sup> FAO. “Avian influenza A (H5N6): the latest addition to emerging zoonotic avian influenza threats in East and Southeast Asia.” <http://www.fao.org/3/a-i4199e.pdf>.

<sup>24</sup> WHO. “Antigenic and genetic characteristics of zoonotic influenza viruses and development of candidate vaccine viruses for pandemic preparedness.” 2013. [http://www.who.int/influenza/vaccines/virus/201309\\_h5h7h9\\_vaccinevirusupdate.pdf](http://www.who.int/influenza/vaccines/virus/201309_h5h7h9_vaccinevirusupdate.pdf).

<sup>25</sup> Doyle, M.P., and M.C. Erickson. “Emerging Microbiological Food Safety Issues Related to Meat.” *Meat Science*, 74, no. 1 (2006): 98-112.

<sup>26</sup> Perdue, Michael L. “Molecular Determinants of Pathogenicity for Avian Influenza Viruses.” *Avian Influenza*, (2008): 23-41. doi:10.1002/9780813818634.ch2.

<sup>27</sup> Reid, A. H., et al. “Relationship of Pre-1918 Avian Influenza HA and NP Sequences to Subsequent Avian Influenza Strains.” *Avian Diseases* 47, (2003): 921-25.

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antigenic drift and antigenic shift.<sup>28,29</sup> Antigenic drift is the anticipated accumulation of point mutations in the viral genome. This tends to cause only small changes in the biological behavior of the virus. Antigenic shift results when new combinations of H and N genes are generated by genetic reassortment, which occurs when cells of the host are coinfecting with two genetically different viruses.

Subtypes of influenza A have adapted to both humans and swine, and currently circulate.<sup>30</sup> Human infections with AI viruses have led the World Health Organization (WHO) to consider the possibility that a new human pandemic may be derived directly from birds. However, the evidence and triggers which cause an influenza A virus to adapt to a mammalian host remain less clear than the transition of an influenza A virus from a low pathogenicity to a highly pathogenic form in poultry.<sup>31</sup>

## 1.5 Environmental Persistence of AI Viruses

AI viruses can survive in cool and moist conditions, particularly when organic material is present.<sup>32</sup> The World Organization for Animal Health (OIE) states the following about the resistance of AI viruses to physical and chemical action.<sup>33</sup>

- Temperature: Pasteurization and cooking inactivate the AI virus. Cooking whole eggs at 60 °C for 188 seconds and 507 seconds for poultry meat will inactivate the virus. Cooking meat to a core temperature of 70 °C for 3.5 seconds will also inactivate AI. AI virus can survive indefinitely if frozen.
- pH: Inactivated by acidic pH of  $\leq 2$ .
- Chemicals: Inactivated by organic solvents and detergents, such as sodium desoxycholate and sodium dodecylsulphate. If organic matter is present, aldehydes,  $\beta$ -propiolactone and binary ethyleneimine should be used for inactivation. After organic matter has been removed, phenolics, quaternary ammonium compounds, oxidizing agents (such as sodium hypochlorite), dilute acids (if  $\text{pH} \leq 2$ ), hydroxylamine, and lipid solvents should be used.
- Disinfectants: For clean surfaces with no organic matter, use sodium hypochlorite (5.25 percent), sodium hydroxide (2 percent), phenols, acidified ionophors, chlorine dioxide, or strong oxidizing agents to inactivate.

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<sup>28</sup> Suarez, David L. "Influenza A Virus." *Avian Influenza*, (2008): 1-22.

<sup>29</sup> CFSPH, Iowa State University & Institute for International Cooperation in Animal Biologics. "High Pathogenicity Avian Influenza." 2014. [http://www.cfsph.iastate.edu/Factsheets/pdfs/highly\\_pathogenic\\_avian\\_influenza.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/highly_pathogenic_avian_influenza.pdf).

<sup>30</sup> Perdue, Michael L. "Molecular Determinants of Pathogenicity for Avian Influenza Viruses." *Avian Influenza*, (2008): 23-41. doi:10.1002/9780813818634.ch2.

<sup>31</sup> Perdue, Michael L. "Molecular Determinants of Pathogenicity for Avian Influenza Viruses." *Avian Influenza*, (2008): 23-41. doi:10.1002/9780813818634.ch2.

<sup>32</sup> Swayne, David E. "Epidemiology of Avian Influenza in Agricultural and Other Man-Made Systems." *Avian Influenza*, (2015): 59-85. doi:10.1002/9780813818634.ch4.

<sup>33</sup> OIE. Highly Pathogenic Avian Influenza, Technical Disease Card. 2014. [www.oie.int](http://www.oie.int).

- Survival: Can survive in surface waters. Viable in liquid feces for 30–35 days at 4 °C and for 7 days at 20 °C. Survived 4 days in chicken feces held between 25–32 °C in the shade. In water, can survive 26–30 days at 28 °C, and 94–158 days at 17 °C. Composting kills virus within poultry carcasses in <10 days.

### 1.5.1 In Eggs and Egg Products

The OIE recommends the following times and temperatures for the inactivation of AI virus in eggs and egg products (Table 1-1).<sup>34,35</sup>

**Table 1-1. Inactivation of AI in Eggs and Egg Products**

	Temperature (°C)	Time
Whole egg	60.0	188 seconds
Whole egg blends	60.0	188 seconds
	61.1	94 seconds
Liquid egg white	55.6	870 seconds
	56.7	232 seconds
10% salted yolk	62.2	138 seconds
Dried egg white	67.0	20 hours
	54.4	513 hours

Source: OIE *Terrestrial Animal Health Code*, 2015.

The OIE *Terrestrial Animal Health Code* (2015) states that the listed temperature achieves a 7-log kill.

Because AI virus can persist on the surface of eggs and fillers, these items also need to be sanitized.<sup>36,37</sup>

<sup>34</sup> OIE. *Terrestrial Animal Health Code*. 2015. [www.oie.int](http://www.oie.int).

<sup>35</sup> Swayne, David E., and Joan R. Beck. "Heat Inactivation of Avian Influenza and Newcastle Disease Viruses in Egg Products." *Avian Pathology* 33, no. 5 (2004): 512-18.

<sup>36</sup> OIE. *Highly Pathogenic Avian Influenza, Technical Disease Card*. 2014. [www.oie.int](http://www.oie.int).

<sup>37</sup> Swayne, David E. "Epidemiology of Avian Influenza in Agricultural and Other Man-Made Systems." *Avian Influenza*, (2015): 59-85. doi:10.1002/9780813818634.ch4.

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## 1.5.2 In Meat

The OIE recommends the following times and temperatures for inactivation of AI virus in meat (Table 1-2).<sup>38,39</sup>

**Table 1-2. Inactivation of AI in Meat**

	Temperature (°C)	Time
Poultry meat	60.0	507 seconds
	65.0	42 seconds
	70.0	3.5 seconds
	73.9	.51 seconds

Source: OIE *Terrestrial Animal Health Code*, 2015.

## 1.5.3 In Poultry Byproducts and Waste

All rendered meal, including that produced from viscera, blood, feathers, feet, heads, necks, and poultry offal, may contain AI, and may be added to pet foods. Such food is typically cooked at above 100 °C for several minutes to more than an hour, which is sufficient to kill the AI virus. However, if procedures are not properly followed, AI virus could persist in the byproducts for several weeks.

Waste—the unwanted byproduct of processing—may contain material contaminated with AI. Care should be taken that such material is treated in a manner to deactivate the virus, and that transport takes this biosecurity risk into consideration. The OIE *Terrestrial Animal Health Code* (2015) recommends, for the importation of feather meal and poultry meal, the following (Article 10.4.24):<sup>40</sup>

Regardless of the avian influenza status of the country of origin, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. These commodities were processed in an avian influenza free country, zone or compartment from poultry which were kept in an avian influenza free country, zone or compartment from the time they were hatched until the time of slaughter or for at least the 21 days preceding slaughter; or,
2. These commodities have been processed either:
  - a) With moist heat at a minimum temperature of 188 °C for a minimum of 40 minutes;
  - b) With a continuous hydrolyzing process under at least 3.79 bar of pressure with steam at a minimum temperature of 122 °C for a minimum of 15 minutes; or
  - c) With an alternative rendering process that ensures that the internal temperature throughout the product reaches at least 74 °C;

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<sup>38</sup> OIE. *Terrestrial Animal Health Code*. 2015. [www.oie.int](http://www.oie.int).

<sup>39</sup> Swayne, David E., and Joan R. Beck. "Heat Inactivation of Avian Influenza and Newcastle Disease Viruses in Egg Products." *Avian Pathology* 33, no. 5 (2004): 512-18.

<sup>40</sup> OIE. *Terrestrial Animal Health Code*. 2015. [www.oie.int](http://www.oie.int).

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AND

3. The necessary precautions were taken to avoid contact of the commodity with any source of the avian influenza virus.

#### 1.5.4 In Carcasses

AI viruses can survive in bird carcasses for several days at ambient temperatures, and a few weeks at refrigeration temperatures. Titers in carcasses will vary depending on the strain of the virus, species of bird, and time of death in relation to clinical stage of infection. Burying, incineration, or composting (virus inactivated in less than 10 days) is recommended for carcasses.<sup>41,42</sup>

### 1.6 Threat of HPAI to the United States

The long distances in the seasonal migration behavior of influenza A virus hosts causes concern about the spread of HPAI to the United States. Although the majority of migratory events occur within continents, intercontinental migration does take place.<sup>43</sup> Within the United States for example, there are four migratory waterfowl flyways: Pacific, Central, Mississippi, and Atlantic.<sup>44</sup> The Pacific flyway is a potential risk for AI introduction into North America as various migratory avian species from Asia enter the space and mix with North American species.<sup>45</sup>

The current H5 HPAI viruses, defined as novel Eurasian lineage clade 2.3.4.4, began rapidly spreading in January 2014 from South Korea to China and Japan. By November 2014, clade 2.3.4.4 H5N1 was present in Germany, the Netherlands, and the United Kingdom. Also in November 2014, a novel HPAI H5N2 virus consisting of a reassortment of Eurasian 2.3.4.4, four other Eurasian genes, and three North American wild bird lineage genes was reported in British Columbia, Canada. With the detection of very similar H5N8 Eurasian viruses in three continents (Asia, Europe, and North America), there is evidence that this particular reassortant is not only well adapted to specific species of waterfowl, it is able to survive extended migration periods.<sup>46</sup> The HPAI H5N8 virus has adapted to wild waterfowl hosts as few or no clinical signs have been reported in these hosts when infected with the virus. Thus, it seems probable that the virus was disseminated out of Russia into Europe, East Asia, and North America by migrating waterfowl during autumn 2014.<sup>47</sup>

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<sup>41</sup> Swayne, David E. "Epidemiology of Avian Influenza in Agricultural and Other Man-Made Systems." *Avian Influenza*, (2015): 59-85. doi:10.1002/9780813818634.ch4.

<sup>42</sup> OIE. Highly Pathogenic Avian Influenza, Technical Disease Card. 2014. [www.oie.int](http://www.oie.int).

<sup>43</sup> Miller, Mark P., et al. "Intercontinental Genetic Structure and Gene Flow in Dunlin (*Calidris alpina*), a Potential Vector of Avian Influenza." *Evolutionary Applications* 8, no. 2 (2015): 149-71.

<sup>44</sup> Nallar, et al. "Demographic and Spatiotemporal Patterns of Avian Influenza Infection at the Continental Scale, and in Relation to Annual Life Cycle of a Migratory Host." *PLoS ONE*, (2015). doi:10.1371/journal.pone.0130662.

<sup>45</sup> Scotch, M., et al. "Diffusion of Influenza Viruses among Migratory Birds with a Focus on the Southwest United States." *Infection, Genetics and Evolution*, (2014): 185-193.

<sup>46</sup> Ip, Hon S., et al. "Novel Eurasian Highly Pathogenic Avian Influenza A H5 Viruses in Wild Birds, Washington, USA, 2014." *Emerging Infectious Diseases* 10, no. 5 (2015): 886-90.

<sup>47</sup> Hall, Jeffrey S., Robert J. Dusek, and Erica Spackman. "Rapidly Expanding Range of Highly Pathogenic Avian Influenza Viruses." *Emerging Infectious Diseases* 21, no. 7 (2015): 1251-252.

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Upon further analysis of Eurasian clade 2.3.4.4 after its introduction into the United States, it was determined that H5N8 survived introduction into North America. Furthermore, H5N8 introduction into North America is independent of introduction into Europe. Lastly, it is unknown how long the virus has been present in the Pacific flyway (California, Idaho, Nevada, Oregon, Utah, and Washington, USA), but it has been long enough for reassortment with LPAI North American lineage wild bird avian influenza virus.<sup>48</sup>

HPAI continues to be a threat in the United States after the latest 2014–2015 outbreak as the virus could still be present in asymptomatic wild waterfowl. With the 2015 fall migration of waterfowl approaching, chances of potential carriers of HPAI coming in contact with poultry increases. The real problem arises when HPAI is introduced into vulnerable poultry populations where the virus amplifies and spreads, resulting in extremely high morbidity and mortality, and associated significant, detrimental economic consequences.

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<sup>48</sup> Ip, Hon S., et al. “Novel Eurasian Highly Pathogenic Avian Influenza A H5 Viruses in Wild Birds, Washington, USA, 2014.” *Emerging Infectious Diseases* 10, no. 5 (2015): 886–90.

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## Attachment 1.B Abbreviations

AI	avian influenza
APHIS	Animal and Plant Health Inspection Service
CFSPH	Center for Food Security and Public Health
FAD PReP	Foreign Animal Disease Preparedness and Response Plan
FAO	Food and Agriculture Organization
H or HA	hemagglutinin
HPAI	highly pathogenic avian influenza
LPAI	low pathogenicity avian influenza
N or NA	neuraminidase
NP	nucleocapsid protein
OIE	World Organization for Animal Health
RNA	ribonucleic acid
rRT-PCR	real-time reverse transcriptase polymerase chain reaction
SOP	standard operating procedure
UN	United Nations
USAHA	United States Animal Health Association
USDA	U.S. Department of Agriculture
VS	Veterinary Services
WHO	World Health Organization