

Highly Pathogenic Avian Influenza (HPAI)

Background

Fowl plague was described in 1878 as a serious disease of chickens in Italy. It was determined in 1955 that fowl plague (FP) virus is actually one of the influenza viruses. The AI viruses, along with the other influenza viruses, make up the virus family Orthomyxoviridae. The virus particle has an envelope with glycoprotein projections with hemagglutinating and neuraminidase activity. These two surface antigens, hemagglutinin (HA) and neuraminidase (NA), are the basis of describing the serologic identity of the influenza viruses using the letters H and N with the appropriate numbers in the virus designation e.g., H7N2. There are now 15 hemagglutinin and 9 neuraminidase antigens described among the Type A influenza viruses. The type designation (A, B, or C) is based upon the antigenic character of the M protein of the virus envelope and the nucleoprotein within the virus particle. All influenza viruses affecting domestic animals (equine, swine, avian) belong to Type A, and Type A influenza virus is the most common type producing serious epidemics in humans. Types B and C do not affect domestic animals. All highly pathogenic isolates have been Influenza A viruses of subtypes H5 & H7.

Classical fowl plague viruses have H7 as one of the surface antigens but can have different N antigens. It was once believed that all H7 viruses are highly pathogenic fowl plague viruses and that no other avian influenza viruses could produce a fowl-plague-like disease. When avirulent AI viruses with the H7 antigens were demonstrated in turkeys in 1971 and highly virulent AI viruses with the H5 antigen were first found in chickens in 1959, the necessity for redefining the term fowl plague or using other terminology became apparent. Because there are highly virulent AI viruses that possess H antigen other than the H7 and H7 AI viruses that do not produce clinical fowl plague, an international assembly of avian influenza specialists proposed that the term fowl plague no longer be used. They suggested that any AI virus, regardless of its HA designation, meeting specified virulence requirements in the laboratory be designated highly pathogenic avian influenza (HPAI). The criteria that serve as the basis for classifying an AI virus as HPAI has more recently been modified to include molecular considerations such as the type of amino acids at the cleavage site of its HA.

Agent Criteria

Infectious Dose: Unknown

Stability: Can be deactivated by Sodium Hypochlorite at 10,000 ppm (1%) with a contact time of 10 minutes; ethanol 70% with a contact time of 10 minutes; Ortho phenylphenol at 1200 ppm with a contact time of 10 minutes; Benzalkonium chloride at 1000 ppm with a contact time of 10 minutes. HPAI is deactivated by 15 minutes at 121°C, it can survive at 56°C for up to 3 hours,

and at 60°C for up to 30 minutes. HPAI is inactivated by acid pH. HPAI remains viable for long periods in tissues, feces and water (manure 105 days, dried mucous several hours).

Shedding patterns (for animal pathogens): Shed from bodily secretions until the animal dies or recovers.

Incubation Period: 3-7 days for birds, 2-8 days for humans

Mortality Rate: Close to 100% for most bird species, depending on the specific isolate. For humans, over 50%.

Morbidity Rate:

Duration of Illness: Death may occur within 24 hours of first signs of disease, frequently within 48 hours, or be delayed for as 12 days. Some severely affected hens may occasionally recover. In turkey's the disease may last 2-3 days longer.

For humans: 3-13 days, depending on the strain.

Severity of Illness: HPAI can range from mild disease with little or no mortality to highly fatal rapidly spreading epidemic. Infections result in marked depression with ruffled feathers, inappetence, excessive thirst, cessation of egg production, coughing and sneezing and watery diarrhea. Mature chickens frequently have swollen combs, wattles, and edema surrounding the eyes. The combs are often cyanotic at the tips and may have plasma or blood vesicles on the surface with dark areas of ecchymotic hemorrhage and necrotic foci. The last eggs laid, after the onset of illness, are frequently without shells. The diarrhea begins as watery bright green and progresses to almost totally white. Edema of the head, if present, is often accompanied by edema of the neck. The conjunctivae are congested and swollen with occasional hemorrhage. The legs, between the hocks and feet, may have areas of diffuse hemorrhage. Respiratory signs can be a significant feature of the disease, depending on the extent of tracheal involvement. Mucus accumulation can vary. It is not unusual in caged layers for the disease to begin in a localized area of the house and severely affect birds in only a few cages before it spreads to neighboring cages.

For Humans: Fever, cough, aching muscles, sore throat, eye infections and serious respiratory infections including pneumonia.

Duration of Infection: Unknown, few animals survive past clinical symptoms

Long term effects after infection: Birds that survive are usually in poor condition and resume laying only after a period of several weeks.

Allergen (yes/no): No

Carcinogenic/mutagenic (yes/no): No

Abortogenic (yes/no): Yes

Infection Mitigation Measures:

For human pathogens

Immunization: Yes (not widely used)

Prophylaxis: No

Post Infection Treatment: Some strains respond to amantadine and rimantadine, but Asian strains may only respond to oseltamivir and zanamivir, additional studies are pending to prove the effectiveness of oseltamivir and zanamivir against HPAI

Existence of Diagnostic tests: Yes (haemagglutination, immunofluorescence, ELISA, serotyping, PCR, gene sequencing)

Drug resistance: Drug resistant viruses have quickly emerged to anti-viral treatments, (Asian strains to amantadine and rimantadine)

For animal pathogens

Detection Possible: Yes (haemagglutination, immunofluorescence, ELISA, serotyping, PCR, gene sequencing)

Culling: Yes

Prophylaxis: No

Immunization: Yes (vaccine efficacy has been questioned)

Post Infection Treatment: Not recommended due to drug resistance

Drug resistance: Drug resistant viruses have quickly emerged to anti-viral treatments, so the drugs are not recommended for use in poultry.

Routes of Infection:

Inhalation: Yes

Ingestion: Yes

Percutaneous: Yes

Contact: Yes

Vector-Borne: No

Natural Routes of Infection:

Inhalation: Yes, in close proximity

Ingestion: Yes, through contaminated drinking water

Percutaneous: Yes (not normally)

Contact: Yes, the virus is present in bodily secretions

Vector-Borne: No

Sexual Transmission: Yes

Vertical Transmission: Possible

Communicability:

Human to Human: No evidence at this point in time, although some worry the virus will mutate to make this possible

Human to Animal: No

Animal to Animal: Yes

Animal to Human: Transmission to humans occurred in rare cases where people had prolonged contact with heavily contaminated environments

Multiple Species: Migratory waterfowl are the natural host range (especially ducks), all species of birds susceptible (including chickens, turkeys, pet birds and wild birds), humans, other known susceptible species: cats, dogs, pigs

Where is it present: Worldwide