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PROFESSIONAL VERSION

Avian Influenza in Poultry and Wild Birds

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Avian influenza (AI) is a viral infection found in domestic poultry and a wide range of other birds. Some strains sporadically spill over into wild and domestic mammals and humans. Wild waterfowl and shorebirds are often subclinically affected carriers of the AI virus. In poultry, low-pathogenicity strains can cause subclinical infections; however, some strains typically cause respiratory signs or decreased egg production. High-pathogenicity strains can cause widespread organ failure and sudden death, often with high mortality rates. Diagnosis is based on detection of the viral genome or specific antibodies or on virus isolation. Antimicrobials can help control secondary bacterial infection in flocks affected by low-pathogenicity strains. Antiviral drugs are not approved or recommended. Prevention is best accomplished by biosecurity measures. Vaccines matched for antigenic type can greatly increase resistance to infection, prevent clinical signs, decrease viral shedding in infected flocks, stop transmission between birds, prevent farm-to-farm spread, and decrease outbreaks.

Avian influenza (AI) is a viral infection that **affects primarily domestic poultry and pet, zoo, and wild birds**. In domestic poultry, **AI viruses are typically of low pathogenicity (LPAI)** and cause subclinical infections, respiratory disease, or decreased egg production. Some AI viruses, however, have **high pathogenicity (HPAI)** and cause severe systemic disease with multiple organ failure and high mortality rates, principally in chickens, turkeys, and other gallinaceous poultry.

The form of AI resulting from HPAI viruses has historically been called **fowl plague** or **fowl pest**.

Etiology of Avian Influenza

Avian influenza viruses are **type A orthomyxoviruses** (*Alphainfluenzavirus* or *Influenzavirus A*) characterized by antigenically homologous nucleoprotein and matrix protein, which are identified by serological testing such as agar gel immunodiffusion (AGID) or ELISA.

AI viruses are further divided into **17 hemagglutinin subtypes (H1–H16 and H19) and 9 neuraminidase subtypes (N1–N9)**. Within each hemagglutinin subtype there can be additional subclassifications, such as distinct virus lineages, genetic clades, and genotypes.

From 1959 through June 2025, 52 distinct virus lineages caused HPAI outbreaks or events.

Epidemiology of Avian Influenza

Low-pathogenicity avian influenza (LPAI) viruses are distributed worldwide and are recovered frequently from clinically normal shorebirds (order Charadriiformes) and migrating waterfowl (order Anseriformes), especially dabbling ducks. Occasionally, LPAI viruses are recovered from pet birds and ratites.

LPAI viruses can be present in village or backyard poultry flocks and in other birds sold through live poultry markets. In the US and Europe, most commercially raised poultry are free of AI viruses. H9N2 LPAI is common in commercial and live poultry market birds in Asia, the Middle East, and Africa; however, any LPAI virus subtype can cause sporadic infections.

High-pathogenicity avian influenza (HPAI) viruses arise **from the mutation of some H5 and H7 LPAI viruses**, and 52 unique virus lineages have caused outbreaks. Biosecurity, surveillance, and stamping-out programs have resulted in quick elimination of emergent HPAI viruses (49 of 52 outbreaks) (1, 2, 3). In various geographical areas, however, the **following three HPAI viruses have become endemic:**

- H5Nx A/goose/Guangdong/1/1996 (Gs/GD)–related panzootic HPAI viruses (Africa, Asia, Europe, North and South America, and Antarctica)
- H7N3 North American lineage epizootic HPAI viruses (Mexico)
- H7N9 Eurasian lineage epizootic HPAI viruses (China)

The **incubation period** of AI viruses is highly variable, ranging from a few days in individual birds to 2 weeks in a flock. The WOAH recognizes **14 days as the incubation period** for control programs.

The morbidity and mortality rates caused by LPAI viral infections are usually low, unless the infection is accompanied by secondary bacterial or viral infections or aggravated by environmental stressors. Even in the absence of secondary pathogens, **HPAI viruses cause severe, systemic disease with high mortality rates in chickens, turkeys, and other gallinaceous poultry**; mortality rates can be as high as 100% a few days after exposure in unvaccinated birds.

AI viruses are **transmitted between individual birds by ingestion or inhalation**. Spread between farms results from breaches in biosecurity practices, principally **via the movement of infected poultry or on fomites** (eg, equipment or clothing) **contaminated with infectious feces and respiratory secretions**. Airborne dissemination between farms might be important over short distances.

Pearls & Pitfalls

- Spread of avian influenza between farms results from breaches in biosecurity practices, principally via the movement of infected poultry or on fomites (eg, equipment or clothing) contaminated with infectious feces or respiratory secretions.

The H5Nx Gs/GD-related HPAI viruses have been transmitted by wild aquatic birds that have, since 2005, been associated with five transcontinental movements. The rate of transmission from wild birds to poultry has increased since 2020. Dispersion by wild birds has not been typical of most other lineages of HPAI viruses. Other HPAI strains and all LPAI strains have minimal potential to infect dogs and cats.

Since autumn 2020, the **genetic clade 2.3.4.4b of the H5N1 Gs/GD-related HPAI viruses has caused a global crisis**, a panzootic. Infections have been reported in domestic and wild birds (in > 500 species, > 50 families, and 20 orders of birds), domestic and wild mammals, and humans in Asia, Africa, Europe, North and South America, and Antarctica.

Geographical **spread of these viruses has been driven by migratory aquatic birds** of variable signalment and degree of infection, ranging from subclinically affected (eg, dabbling ducks) to severely infected with high death rates. This spread by migratory birds has led to massive die-offs of various tern species in colonies in the North Sea of Europe.

Through March 2026, HPAI infections by viruses of genotype B3.13 in the 2.3.4.4b clade were **confirmed in dairy cattle in the US**; > 1,088 herds were affected in 19 (mostly western) states (4). Infection by genotype D1.1 viruses has also been diagnosed, on a few Arizona, Nevada, and Wisconsin dairy farms (5).

Sporadic natural and experimental infections caused by H5Nx Gs/GD-related HPAI viruses have been **reported in cats and dogs, as well as in wild mammals such as red foxes, feral cats, and raccoons**. Such experimental infections have occurred after aerosol or respiratory exposure, ingestion of infected chickens or wild birds, or close-contact exposure.

Since 2024, some HPAI **infections of cats have been linked to consumption of raw milk and uncooked raw poultry** meat products (6, 7, 8, 9, 10, 11, 12, 13).

Potentially, domestic pets could serve as a transmission vector for AI between farms. However, the ability of AI viruses other than the H5Nx Gs/GD-related HPAI group, including other HPAI strains, to infect pets is unknown.

Laboratory mammals that have been experimentally infected with H5Nx Gs/GD-related HPAI viruses include **pigs, ferrets, rats, rabbits, guinea pigs, mice, mink, and nonhuman primates**. Since 2022, cases of H5N1 clade 2.3.4.4b Gs/GD-related HPAI have been reported also in farmed mink, foxes, sable, and raccoon dogs.

Natural HPAI infections have been reported in 66 species of terrestrial wild mammals and 19 species of sea mammals (14), including **sea lions, sea otters, elephant seals, and harbor seals**. Isolated cases

of 2.3.4.4b HPAI virus infections have been reported in **sheep, pigs, goats, and alpacas** in the US. Serological or virological evidence of rare H5 infections have been reported in sheep in Norway (15) and the United Kingdom (16), and in dairy cattle in the Netherlands (17).

In certain geographical areas, dogs and cats might be commonly infected by specific influenza A viruses that are adapted to each specific species (H3N8 and H3N2 in dogs; H7N2 in cats).

Zoonotic Risk of Avian Influenza

Avian influenza viruses exhibit host adaptation to birds. **AI infections have occurred in humans, usually as isolated, rare, individual cases.** Most human cases have originated from infection with H5Nx Gs/GD-related HPAI viruses and H7N9 Eurasian lineage LPAI and HPAI viruses.

From 2003 to May 27, 2025, the total number of human cases of infection by H5N1 Gs/GD-related HPAI viruses, most in Asia and Africa, was 993, of which 477 were fatal (18). The US has reported 71 human cases, with 2 fatalities; 65 of the surviving 70 people had occupational exposure (41 were workers on affected dairy farms, and 24 were poultry workers, mostly on depopulation crews), and 5 others were exposed in other/unknown ways (19). The patient who died was infected via exposure to an infected backyard flock.

Globally, the **primary risk factor for AI infection in humans has been direct contact with live or dead infected poultry** in live poultry markets. However, rare cases have resulted from consumption of uncooked, infected poultry products; defeathering of infected wild swans; or close contact with infected dairy cattle, or poultry culling operations.

The H5N6 Gs/GD-related HPAI virus has caused 93 laboratory-confirmed cases of infection in humans in China and Laos, with 57 deaths. The H9N2 LPAI virus has caused 157 human cases (2 deaths) in Asia and Africa (20).

Respiratory infection has been the most frequent clinical sign in human H5 and H9N2 AI cases; however, workers on affected dairy farms have commonly had conjunctivitis. Of the AI viruses, only a few H5 Gs/GD-related HPAI viruses have caused limited human-to-human transmission (21).

For the H7N9 LPAI and HPAI viruses, the total number of human cases in China between 2013 and June 13, 2024, was 1,568, of which 616 were fatal (20). Most of the affected individuals had exposure risk to live poultry markets.

Conjunctivitis was the most common sign in human cases of H7N7 HPAI virus infection in the Netherlands during 2003, with 89 confirmed cases and 1 death (22). Other HPAI and LPAI viruses have either rarely or not at all produced infections in humans (including rare cases of H5N8 in poultry workers in Russia, H7N3 in Canada, and H7N4 in China) (23, 24, 25).

Clinical Findings of Avian Influenza in Birds

Clinical signs, severity of disease, and mortality rates of avian influenza vary, depending on the AI virus strain and the host species.

Most AI viruses (subtypes H1–H16 and H19) are LPAI viruses. However, some H5 and H7 AI viruses are HPAI viruses that are highly lethal for chickens, turkeys, and related gallinaceous domestic poultry.

Pearls & Pitfalls

- Most AI viruses are LPAI viruses, but some are HPAI viruses that are highly lethal for chickens, turkeys, and related gallinaceous domestic poultry.

In most wild birds, AI viral infections are subclinical; the **H5 Gs/GD-related HPAI viruses are an exception**. These viruses have been **associated with death in wild and domestic waterfowl and other species of wild and domestic birds**. In some situations, they have caused major die-offs in wild birds such as common cranes (Israel, 2021); turkey and black vultures (US, 2022); and various pelican species (2022–2024), tern species (2023), and brown skuas and penguins (Antarctic region, 2024–2025).

Clinical Findings of Low-Pathogenicity Avian Influenza

Infection by LPAI viruses in poultry typically produces respiratory signs such as **sneezing, coughing, ocular and nasal discharge, and swollen infraorbital sinuses**. Sinusitis is common in domestic ducks, quail, and turkeys (see *Mycoplasma gallisepticum* Infection in Poultry). Lesions in the respiratory tract typically include congestion and inflammation of the trachea and lungs.

In layers and breeders, signs of avian influenza can include **decreased egg production or infertility**, ovum rupture (evidenced by yolk in the abdominal cavity) or involution, or mucosal edema and inflammatory exudates in the lumen of the oviduct. Rarely, layer and breeder chickens have acute renal failure and visceral urate deposition (visceral gout).

Clinical Findings of High-Pathogenicity Avian Influenza

In peracute cases of avian influenza, **clinical signs or gross lesions might be lacking before death**. **In acute cases, lesions can include the following:**

- cyanosis and edema of the head, comb, wattles, and snood (turkey)
- ischemic necrosis of the comb, wattles, or snood (see ischemic necrosis image)
- edema and red discoloration of the shanks and feet due to subcutaneous ecchymotic hemorrhages (see subcutaneous hemorrhage image)
- petechial hemorrhages on visceral organs and in muscles
- blood-tinged oral and nasal discharges
- cloudy corneas (ducks only)

In severely affected birds, greenish diarrhea is common.

Avian influenza, ischemic necrosis of comb and wattles, chicken

IMAGE



A chicken with avian influenza. Note the evidence of hemorrhage and ischemic necrosis in the unfeathered regions (comb and wattles).

COURTESY OF DR. DAVID E. SWAYNE.

Avian influenza, subcutaneous hemorrhage on feet and shanks, chicken

IMAGE



The legs of a chicken with avian influenza. Note the evidence of subcutaneous hemorrhage on the feet and shanks.

COURTESY OF DR. DAVID E. SWAYNE.

Birds that survive peracute AI infection might develop CNS involvement evident as **torticollis, opisthotonos, incoordination, paralysis, and drooping wings**. Microscopic lesions are highly variable

in both location and severity; they can consist of edema, hemorrhage, and necrosis in parenchymal cells of multiple visceral organs, the skin, and the CNS.

Diagnosis of Avian Influenza in Birds

- Detection of AI-specific viral RNA
- Detection of AI-specific antibodies
- AI virus isolation

Clinical signs alone are not diagnostic for avian influenza but can be highly suggestive of infection that must be confirmed by laboratory findings.

Pearls & Pitfalls

- Clinical signs alone are not diagnostic for avian influenza but can be highly suggestive of infection that must be confirmed by laboratory testing.

LPAI and HPAI viruses can be readily **isolated from oropharyngeal and cloacal swabs** from domestic and wild birds and, in the case of HPAI viruses, from many internal organs. AI viruses grow well in the allantoic sac of 9- to 11-day-old embryonating chicken eggs, and they agglutinate RBCs. Such hemagglutination is not inhibited by antisera for Newcastle disease virus or other paramyxoviruses.

Virus isolation should be attempted only in approved laboratories with high-level biocontainment. Most countries restrict which laboratories and personnel can work with HPAI viruses. Veterinarians in the US can contact the National Animal Health Laboratory Network (NAHLN) to identify a laboratory where they can send samples for testing.

Identification of AI viruses is based on the following:

- influenza A matrix or nucleoprotein antigens, demonstrated by AGID or other suitable immunoassays
- viral RNA, demonstrated by influenza A–specific RT-PCR assay
- reaction with antibodies specific for AI virus

AI viruses are further classified into hemagglutinin subtypes (H1–H16 and H19) and neuraminidase subtypes (N1–N9) on the basis of the hemagglutinin inhibition and neuraminidase inhibition tests, respectively, performed at a national or international reference laboratory, or by genetic analysis of sequence data.

Laboratory Tests for Avian Influenza Antibodies

Avian influenza infections in birds that have recovered from the disease can be **confirmed by serological testing for influenza virus A (using AGID or ELISA)** and further classified by

hemagglutinin and neuraminidase subtype on the basis of hemagglutinin inhibition and neuraminidase inhibition tests, respectively.

Differential Diagnoses of Avian Influenza

LPAI infections must be differentiated from other respiratory diseases or causes of decreased egg production, including the following:

- acute to subacute viral diseases such as infectious bronchitis, infectious laryngotracheitis, avian metapneumovirus infections, low virulent Newcastle disease, and infections by other paramyxoviruses
- bacterial diseases such as mycoplasmosis, infectious coryza, ornithobacteriosis, turkey coryza, and the respiratory form of fowl cholera
- fungal diseases such as aspergillosis

HPAI infections must be **differentiated from other causes of high mortality rates**, such as virulent Newcastle disease, the peracute septicemic form of fowl cholera, heat exhaustion, and severe water deprivation.

Treatment of Avian Influenza in Birds

- Antimicrobials against secondary pathogens
- Supportive care

Treating LPAI-affected flocks (H1–4, H6, H8–16, and H19 subtypes) by administering **broad-spectrum antimicrobials to control secondary pathogens**, **increasing house temperatures**, and providing supportive care with fluids and supplemental feeding can decrease morbidity and mortality rates.

All poultry on HPAI-affected farms should be depopulated and disposed of by an environmentally sound method. Most H5- and H7-infected poultry are similarly depopulated.

Pearls & Pitfalls

- Treating LPAI-affected flocks by administering broad-spectrum antimicrobials to control secondary pathogens and increasing house temperatures can decrease morbidity and mortality rates.

Treatment with antiviral compounds is not approved or recommended for poultry.

Prevention of Avian Influenza in Birds

Exclusion biosecurity strategies to prevent the introduction of avian influenza into poultry are the best preventive measure. **Suspected outbreaks should be reported to appropriate regulatory authorities.**

Antigenically matched and properly administered **vaccines can prevent AI infections, clinical signs, and death**. If birds become infected, vaccines greatly **decrease viral replication and shedding from the respiratory and GI tracts**, and help to stop spread between farms.

To prevent the spread of AI to species such as dogs and cats, **any poultry or poultry products fed to pets should be cooked (not raw), and any milk given to pets should be pasteurized**.

Pearls & Pitfalls

- To prevent the spread of AI to species such as dogs and cats, any poultry or poultry products fed to pets should be cooked (not raw), and any milk given to pets should be pasteurized.

Specific protection against AI is achieved through autogenous virus vaccines and through vaccines prepared from the AI virus of the same hemagglutinin subtype. Antibodies against homologous viral neuraminidase antigens can provide partial protection.

In the US, only inactivated whole AI virus, H5 subunit hemagglutinin, DNA of H5 hemagglutinin, RNA particle (defective eastern equine encephalitis virus) with H5 hemagglutinin insert, recombinant fowlpox-AI-H5, and recombinant herpesvirus-turkey-AI-H5 (rHVT-AI-H5) vaccines are licensed.

Vaccination against AI viruses is highly regulated and restricted in many countries. In the US, the following regulations are in place:

- Only USDA autogenous, conditional or fully licensed vaccines can be considered for veterinary use.
- The use of any licensed AI vaccine for H1–H4, H6, H8–H16, and H19 hemagglutinin subtypes requires approval by the state veterinarian of the state in question.
- Autogenous vaccines against H1N1 and H3N2 swine influenza are approved in some states for use against AI in breeder and meat turkeys.
- The use of H5 and H7 AI vaccines in the US requires declaration of an emergency and approval by the secretary of agriculture or chief veterinary officer. **Currently, only wild or captive California condors (*Gymnogyps californianus*) are approved for H5 vaccination in the US.**

Multiple countries in Asia, Africa, Europe, North America, and South America are using vaccination of poultry as a tool, along with biosecurity, surveillance, and stamping-out of infected flocks, to assist in preventing and controlling HPAI infection.

A global strategy to prevent and control HPAI has been developed and implemented by WOAH and the Food and Agriculture Organization of the United Nations (FAO).

Key Points

- **Avian influenza viruses are detected seasonally in subclinically affected migratory waterfowl and shorebirds.**
- **Two clinical types of avian influenza occur in poultry: a low-pathogenicity form (LPAI), as subclinical infections, respiratory disease, or decreased egg production; and a high-pathogenicity form (HPAI), as severe systemic disease with multiple organ failure and high mortality rates.**
- **Diagnosis of AI is based on the detection of viral RNA or AI-specific antibodies, or by virus isolation.**
- **Prevention is by exclusion biosecurity strategies and vaccination; however, vaccination is highly regulated and restricted in many countries.**
- **Zoonotic infections are rare, but AI has been reported in humans either without clinical signs or accompanied by conjunctivitis, respiratory disease, or multiple organ failure and death.**

For More Information

- Avian Influenza. USDA Animal and Plant Health Inspection Service (APHIS).
- Avian Influenza. WOAH.
- New Global Strategy for the Prevention and Control of High Pathogenicity Avian Influenza. WOAH.
- OFFLU (WOAH-FAO Network of Expertise on Animal Influenza).
- Also see pet owner content regarding bird flu.

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