**Newcastle Disease** is a highly contagious viral infection that affects many species of domestic and wild birds to varying degrees. Domestic fowl, turkeys, pigeons and parrots are most susceptible while a mild form of the disease affects ducks, geese, pheasants, quail and guinea fowl. The disease can result in digestive, respiratory and/or nervous clinical signs, which range from a mild, almost inapparent respiratory disease to very severe depression, drop in egg production, increased respiration, profuse diarrhoea followed by collapse, or long-term nervous signs (such as twisted necks), if the birds survive. Severe forms of the disease are highly fatal.

**What causes Newcastle Disease?**

Newcastle Disease is caused by a paramyxovirus that can vary in pathogenicity from mild to highly pathogenic. Spread is usually by direct physical contact with infected or diseased birds. The virus is present in manure and is breathed out into the air. Other sources of infection are contaminated equipment, carcasses, water, food and clothing. People can easily carry the virus from one shed or farm to another. Newcastle Disease virus does not affect humans in the same way that it does birds but it can cause conjunctivitis in humans.

**Prevention and treatment of Newcastle Disease**

There is no treatment for Newcastle Disease, although treatment with antibiotics to control secondary infections may assist. The virus can remain alive in manure for up to 2 months and in dead carcasses for up to 12 months, however it is easily killed by disinfectants, fumigants and direct sunlight. Prevention relies on good quarantine and biosecurity procedures and vaccination. Newcastle Disease vaccination of commercial meat and egg layer chickens has been made compulsory in many Australian States. For information on compulsory Newcastle Disease vaccination in your State contact your State department of primary industries or agriculture.

**Bird Flu**

Avian influenza (AI) is a viral infection of domestic poultry, and pet, zoo, and wild birds. In domestic poultry, AI viruses are typically of low pathogenicity (LP), causing subclinical infections, respiratory disease, or drops in egg production, but a few AI viruses are highly pathogenic (HP), causing severe systemic disease with multiple organ failure and high mortality.

## Etiology of Avian Influenza

Avian influenza viruses are type A orthomyxoviruses (*Influenzavirus A*) characterized by antigenically homologous nucleoprotein and matrix internal proteins, which are identified by serology in agar gel immunodiffusion (AGID) tests. AI viruses are further divided into 16 hemagglutinin (H1-16) and 9 neuraminidase (N1-9) subtypes.

## Epidemiology and Transmission of Avian Influenza

Low pathogenicity avian influenza viruses are distributed worldwide and are recovered frequently from clinically normal shorebirds (Chadriiformes) and migrating waterfowl (Anseriformes). Occasionally, LP viruses are recovered from imported pet birds and ratites. The viruses may be present in village or backyard flocks and other birds sold through live-poultry markets, but most commercially raised poultry in developed countries are free of AI viruses. The HPAI viruses arise from mutation of some H5 and H7 LPAI viruses and cause devastating epidemics. Stamping-out programs are used to quickly eliminate the HPAI viruses in developed countries, but some developing countries may use vaccines and management strategies to control HPAI viruses.

The incubation period is highly variable and ranges from a few days in individual birds to 2 weeks in the flock. Transmission between individual birds is by ingestion or inhalation. Spread between farms is the result of breaches in biosecurity practices, principally by movement of infected poultry or contaminated feces and respiratory secretions on fomites such as equipment or clothing. Airborne dissemination between farms may be important over limited distances. Limited spread by wild birds of the Eurasian H5 HPAI virus has occurred but has not been typical of other HPAI viruses. Other HPAI and all LPAI strains have minimal potential to infect dogs and cats.

Sporadic natural and/or experimental infections have occurred in cats and dogs with H5 Eurasian HPAI viruses. Such experimental infections occurred after aerosol or respiratory exposure, ingestion of infected chickens, or contact exposure. Potentially, domestic pets could serve as a transmission vector between farms, but the ability of other AI viruses, including other HPAI strains, to infect pets is unknown. Other mammals have been experimentally infected with H5 HPAI viruses, including pigs, ferrets, rats, rabbits, guinea pigs, mice, mink, and nonhuman primates.

In certain geographic areas, [dogs](https://www.msdvetmanual.com/respiratory-system/respiratory-diseases-of-small-animals/canine-influenza-flu) (H3N8 and H3N2) and cats (H7N2) may be commonly infected by specific influenza A viruses that are adapted to each specific species.

## Clinical Findings and Lesions of Avian Influenza

Most avian influenza viruses (H1-16 subtypes) are LPAI, but some of the H5 and H7 AI viruses are HPAI and highly lethal for chickens, turkeys, and related gallinaceous domestic poultry. This HPAI form of the disease has historically been called fowl plague. In most wild birds, AI viral infections are subclinical except for the recent H5 HPAI viruses of Eurasian lineage, which have been associated with mortality in wild and/or domestic waterfowl and other species of wild and domestic birds. Clinical signs, severity of disease, and mortality rates vary, depending on AI virus strain and host species.

### Low Pathogenicity Avian Influenza Viruses

Low pathogenicity avian influenza viruses typically produce respiratory signs such as sneezing, coughing, ocular and nasal discharge, and swollen infraorbital sinuses in poultry. Sinusitis is common in domestic ducks, quail, and turkeys. Lesions in the respiratory tract typically include congestion and inflammation of the trachea and lungs. In layers and breeders, there may be decreased egg production or infertility, ova rupture (evident as yolk in the abdominal cavity) or involution, or mucosal edema and inflammatory exudates in the lumen of the oviduct. A few layer and breeder chickens may have acute renal failure and visceral urate deposition (visceral gout). The morbidity and mortality is usually low unless accompanied by secondary bacterial or viral infections or aggravated by environmental stressors. Sporadic infections by any subtype of LPAI viruses can occur, but H9N2 LPAI is common in commercial and live bird market poultry in Asia, the Middle East, and North Africa.

### High Pathogenicity Avian Influenza Viruses

**Avian influenza, hemorrhagic skin of feet, chicken**



COURTESY OF DR. DAVID E. SWAYNE.

**Avian influenza, hemorrhagic skin of head, chicken**



COURTESY OF DR. DAVID E. SWAYNE.

Even in the absence of secondary pathogens, HPAI viruses cause severe, systemic disease with high mortality in chickens, turkeys, and other gallinaceous poultry; mortality can be as high as 100% in a few days. In peracute cases, clinical signs or gross lesions may be lacking before death. However, in acute cases, lesions may include cyanosis and edema of the head, comb, wattle, and snood (turkey); edema and red discoloration of the shanks and feet due to subcutaneous ecchymotic hemorrhages; petechial hemorrhages on visceral organs and in muscles; and blood-tinged oral and nasal discharges. In severely affected birds, greenish diarrhea is common.

Birds that survive the peracute infection may develop CNS involvement evident as torticollis, opisthotonos, incoordination, paralysis, and drooping wings. The location and severity of microscopic lesions are highly variable and may consist of edema, hemorrhage, and necrosis in parenchymal cells of multiple visceral organs, skin, and CNS.

## Diagnosis of Avian Influenza

* Avian influenza virus isolation
* Detection of AI viral RNA
* Detection of AI-specific antibodies

The presence of clinical disease alone is not diagnostic. Low pathogenicity and high pathogenicity avian influenza viruses can be readily isolated from oropharyngeal and cloacal swabs, and 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 Newcastle disease or other paramyxoviral antiserum.

**AI viruses are identified by demonstrating the presence of:**

* Influenza A matrix or nucleoprotein antigens using AGID or other suitable immunoassays
* Viral RNA using influenza A-specific reverse transcriptase PCR
* Reaction with antibodies specific for AI virus

AI viruses are further classified into hemagglutinin (H1-16) and neuraminidase (N1-9) subtypes based on the hemagglutinin inhibition and neuraminidase inhibition tests, respectively, which are performed at a national or international reference laboratory or by genetic analysis of sequence data.

### Laboratory Tests for AI Antibodies

Birds that have recovered from clinical disease can be confirmed as AI infections based on serologic testing for influenza virus A (AGID or ELISA) and further subclassified as to hemagglutinin and neuraminidase subtype based on hemagglutinin inhibition and neuraminidase inhibition tests, respectively.

### Differential Diagnosis

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

* acute to subacute viral diseases such as infectious bronchitis, infectious laryngotracheitis, low virulent Newcastle disease, and infections by other paramyxoviruses
* bacterial diseases such as [mycoplasmosis](https://www.msdvetmanual.com/poultry/mycoplasmosis/overview-of-mycoplasmosis-in-poultry), [infectious coryza](https://www.msdvetmanual.com/poultry/infectious-coryza/overview-of-infectious-coryza-in-chickens), ornithobacteriosis, turkey coryza, and the respiratory form of [fowl cholera](https://www.msdvetmanual.com/poultry/fowl-cholera/fowl-cholera)
* fungal diseases such as aspergillosis

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

## Prevention and Treatment of Avian Influenza

* preventive measures
* supportive care

### Preventive Measures

Practice of exclusion biosecurity strategies to prevent introduction of AI into poultry is the best preventive measure. Suspected outbreaks should be reported to appropriate regulatory authorities.

Antigenically matched and properly administered vaccines can prevent clinical signs and death and greatly reduce virus replication and shedding from the respiratory and GI tracts. Specific protection is achieved through autogenous virus vaccines or from vaccines prepared from AI virus of the same hemagglutinin subtype. Antibodies to the homologous viral neuraminidase antigens may provide partial protection. Currently, only inactivated whole AI virus, 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 in the USA.

The use of any licensed AI vaccine for H1-4, H6, and H8-16 hemagglutinin subtypes requires approval of the state veterinarian. In addition, use of H5 and H7 AI vaccines in the USA requires declaration of an emergency and approval by the Secretary of the USDA.

### Supportive Care

Treating LPAI-affected flocks with broad-spectrum antibiotics to control secondary pathogens and increasing house temperatures may reduce morbidity and mortality. Treatment with antiviral compounds is not approved or recommended.

## Zoonotic Risk of Avian Influenza

Avian influenza viruses exhibit host adaptation to birds. Human infections have occurred, usually as isolated, rare, individual cases. Most human cases have originated from infection with Eurasian H5 HPAI virus (A/Goose/Guangdong lineage), and, most recently, H7N9 LPAI virus (Chinese lineage). This lineage of H5N1 HPAI virus has total accumulated human cases in Asia and Africa from 2003–June 2019 of 861, of which 455 were fatal. The primary risk factor for human infection has been direct contact with live or dead infected poultry, but a few cases have resulted from consumption of uncooked poultry products, defeathering of infected wild swans, or close contact with human cases.

Respiratory infection has been the most frequent presentation of human H5 cases. This virus has very limited human-to-human transmission. For H7N9 LPAI, total accumulated human cases in China since 2013 is 1,568, of which 616 were fatal. Most cases had exposure risk to live-poultry markets. Conjunctivitis was the most frequent symptom in human cases of H7N7 HPAI virus infection in the Netherlands during 2003, with 89 confirmed cases and 1 fatality. Other HPAI viruses and all LPAI viruses have produced very rare or no human infections.

## Key Points

* **Avian influenza viruses are detected seasonally in asymptomatic migratory waterfowl and shorebirds.**
* **In developed countries, AI is a rare disease in indoor-reared commercial poultry but is more frequent in outdoor-reared poultry and poultry produced and sold in live poultry markets of large cities.**
* **Two clinical types of AI occur in poultry: a low pathogenicity (LP) form as asymptomatic infections, respiratory disease, or drops in egg production, and a highly pathogenic (HP) form as severe systemic disease with multi-organ failure and high mortality.**
* **Diagnosis is by virus isolation or detection of viral RNA or AI-specific antibodies.**
* **Prevention is by practice of exclusion biosecurity strategies and vaccination, but vaccination is highly regulated and restricted in many countries.**
* **Antiviral treatments are not approved for poultry, although supportive care can reduce mortality and morbidity.**
* **Zoonotic infections are rare but have been reported in humans without symptoms or accompanied by conjunctivitis, respiratory disease, or multi-organ failure and death.**