# Hygiene Protocols for the Prevention and Control of Diseases (Particularly Beak and Feather Disease) in Australian Birds

## Avian Influenza

Australian Government

Department of the Environment and Heritage

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# Avian Influenza

**Definition**: An infectious disease of birds, pigs, Horses, seals, whales, cats, mink, primates and humans, caused by strains of influenza A virus. The disease in chickens and turkeys can be peracute, highly contagious and fatal.

**Synonyms:** Influenza, AI, highly pathogenic avian influenza, HPAI, fowl plague, FP, avian flu. Fowl plague is an historic term and denotes an acute, highly virulent disease of the domestic fowl caused by influenza A virus subtype H7, associated with any N subtype.

**Aetiology:** Influenza viruses types A, B and C belong to the Family *Orthomyxoviridae*. Type specificity is determined by the nature of the nucleoprotein and matrix antigens, which are **antigenically similar** among all influenza A viruses. Types B and C affect only humans.

The influenza virion consists of a sphere about 100nm in diameter, covered with "spikes": densely arranged radial projections. These spikes are of two different kinds. One kind combines with erythrocytes and causes them to agglutinate (haemagglutinin - H) and the other dissolves the linkage between the H spike and the erythrocyte. The second spike is an enzyme, neuraminidase (N).

The hemagglutinin causes influenza viruses to attach themselves to cells. After infection antibodies to the H spike are formed by the host, preventing reinfection by the same strain of influenza virus. Considerable antigenic variation is observed in the hemagglutinin molecule.

The N spike is completely different in appearance and function from the H spike. Neuraminidase may be responsible for getting the assembled virion out of infected cells. Antibodies are formed against N after infection but they are of less importance in providing protection from infection. The N molecule also shows antigenic variation, although it is less variable than that which occurs in the H molecule.

Influenza A viruses display two kinds of antigenic variation in their main H and N antigens. The first kind of change, called **antigenic drift**, consists of a series of minor alterations within a group of similar H or N molecules. The second variation is called **antigenic shift** and is an abrupt and major change in the composition of either the H or N antigens (or both), which, by convention among virologists, are designated $H_0$, $H_1$, $H_2$, $H_3$, $N_1$, $N_2$ and so forth. Antigenic drift, therefore, occurs within a subtype, while antigenic shift denotes a change from one subtype to another.

Influenza viruses differ from most other animal viruses in that the RNA, or ribonucleic acid, that contains their genetic information is replicated and included in the virion as **eight separate single-strand segments**. This segmentation of the RNA means that genetic recombination, or reassortment, can occur readily during mixed infection with different influenza A strains. The recombination of RNA segments is probably of key importance in accounting for major antigenic variations of influenza viruses. Each RNA

segment has been shown to include the genetic information required to code for a single virus protein; eight proteins are synthesised by the virus in the infected cells.

**Epidemiology:** All the subtypes of influenza A virus have been isolated from waterfowl, and the virus appears to produce unapparent intestinal infections in these birds. Even though the birds show no signs, they excrete the virus into the environment from their respiratory tract, conjunctival secretions and faeces. Other birds are infected horizontally either by direct or indirect contact. The incubation period can be as short as a few hours. All ages are susceptible. The virus is not transmitted vertically, since the embryos die before hatching. The 8-segmented viral genome allows the segments to reassort when a cell is infected with two different influenza viruses, yielding a potential 256 genetically different virions. Such mixed infections are not uncommon in nature - two or more antigenically different viruses have been isolated recovered from free-flying ducks and gulls. Genetic reassortment between human and avian viruses has been suggested as the mechanism by which new human pandemic strains arise.

The virus attacks all cells of the body, including those of the heart, and so cardiac output decreases, resulting in interstitial oedema throughout the body, most evident subcutaneously. Some viruses will cause severe disease in one species of bird, and no signs in another. In addition viruses that appear antigenically similar will also differ in the pathogenicities for one species.

**Signs**: Signs depend on the species of bird affected, age, sex, environmental factors and the virulence of the virus, and include respiratory, GIT and nervous. Often there is sudden death, with no other obvious signs or lesions. In less acute infections, the birds become depressed, do not eat, and there is decrease egg production. Mild to severe coughing and sneezing with naso-lacrimal lacrimation and oedema and cyanosis of the head, combs and wattles are also seen. Diarrhoea will also be seen, and nervous signs usually occur only in adults. With highly pathogenic viruses, morbidity and mortality may be 100%.

**Lesions:** Lesions vary within and depend on the species infected and the pathogenicity of the virus. In a flock, all lesions will be seen, but not all lesions occur in the one bird. In the case of highly pathogenic viruses, there may be no lesions because the birds have died so rapidly. There may be severe subcutaneous oedema, especially noticeable over the wattles, comb and legs. These areas will also appear cyanotic (reduced cardiac output plus vascular stasis). Widespread visceral petechiation will also be seen. In layer females, the ovary is flaccid, and several yolks will have ruptured, producing a pseudo "egg peritonitis". A severe inflammation of the GIT will be present, sometimes so severe that the mucosa is necrotic.

**Diagnosis:** Definitive diagnosis depends on isolation and characterisation of the virus. Differentiate from ND, mycoplasmosis, fowl cholera and chlamydophilosis. Avian influenza is a NOTIFIABLE DISEASE, rapid diagnosis is important.

**Treatment**: None permitted in Australia. An outbreak would be handled by slaughter of clinical flocks, disinfection of depopulated premises, quarantine, geoserology, slaughter of seropositive populations and disinfection of depopulated seropositive farms, buildings and equipment.

**Control**: Use of vaccines not permitted. Prevent access of wild birds to poultry sheds, feed stores and water supply.
Be aware of how infectious agents spread and use accepted principles of biosecurity and good management.

**Biosecurity Measures**:

Poultry producers should take the following steps to keep diseases like AI from infecting their flocks:

- Permit only essential workers and vehicles to enter the farm.
- Provide clean clothing and disinfection facilities for employees.
- Clean and disinfect vehicles (including tyres) entering and leaving the farm.
- Avoid visiting other poultry farms.
- Do not loan or borrow equipment or vehicles from other farms.
- Keep an "all-in/all-out" philosophy of farm management.
- Control the movement of all poultry and poultry products from farm to farm.
- Never "skim" mature birds from a flock for sale to a live poultry market.
- Thoroughly clean and disinfect poultry houses between each lot of birds.
- Prevent contact with wild or migratory birds.
- Do not use water that may have been contaminated by wild birds.

**Markets**:

- Use plastic instead of wooden crates for proper cleaning and disinfection.
- Keep scales and floors clean of manure, feathers, and other debris.
- Disinfect all equipment, crates, and vehicles **before** returning them to the farm.
- Keep incoming poultry separate from unsold birds.
- Clean and disinfect the marketplace after every day of sale.