

# Outline



- Introduction
- Causitive agent(s)
- Economic Impact
- Transmission
- Clinical Signs
- Diagnosis and Treatment
- Prevention and Control





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# Introduction

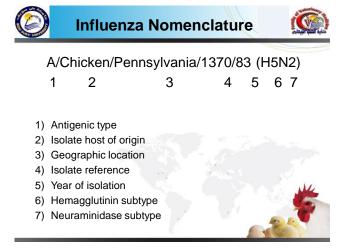
- Type A influenza viruses can infect both mammals including humans, pigs, horses, cats, dogs, ferrets, and sea mammals as well as domestic and wild birds.
- Although influenza A viruses shows host range restrictions, interspecies transmission have been documented at many occasions.





### Introduction

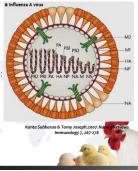
- Influenza is a highly contagious disease causing serious health and economic threats to both humans and animals worldwide
- Al is naturally found in wild birds
- All type Al viruses are thought to originate from wild birds





# Etiology

- Belongs to the family Orthomyxoviridae
- Single-stranded, negative-sense enveloped RNA virus
- 8 segmented genes encoding at least 10 proteins
- Typed based on internal proteins: NP or M1: Influenza A, Influenza B, Influenza C, Isavirus and Thogotovirus
- Influenza A virus : subtyped based on surface proteins: 17 HA and 10 NA subtypes





### Antigenic Drift

- Antigenic 'drift' occurs in HA and NA and induced by:
  - Vaccination pressure: the virus produce escape mutants
  - Influenza virus genes, made of RNA, are more prone to mutations than genes made of DNA.
  - HA gene changes, cause change of HA shape,

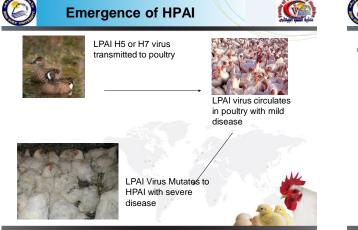




#### Antigenic Shift (Reassortment of Gene Segments)

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- Influenza has 8 separate gene segments that encode 10 different proteins
- When a host cell is infected with two different influenza viruses, the progeny virus can be a mixture of both "parent" viruses
- Reassortment provides for increased biological variation that increases the ability of the virus to adapt to new hosts







### **Al Pathotypes**

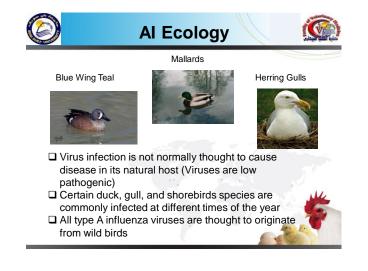


- In all AI viruses, the haemagglutinin produced as a precursor, HA0, which requires post-translational cleavage by host proteases. Based on this AI divided into:
  - A. LPAI: where the HA0 precursor proteins has limited cleavage by host proteases such as trypsin-like enzymes and thus restricted replication i.e. the respiratory and intestinal tracts.
  - B. HPAI: e.g. H5 and H7 HPAI: viruses possess multiple basic amino acids at the HA0 cleavage sites, and are cleavable by ubiquitous proteases, so they are able to replicate throughout the bird



### Waterfowl- Reservoir

- Most HA and all NA subtypes have been found in wild waterfowl
- The distribution of subtypes is not uniform-H6, H3, and H4 tend to predominate in North America
- Some important influenza hemagglutinin subtypes are found uncommonly in birds, including H5 and H7
- The distribution of hemagglutinin subtypes differ from year to year at the same location





mammals

## Hosts restrictions

Al viruses have been shown to infect birds and

The main factors that influence susceptibility to

Human type: SA-α2,6-Gal-terminated saccharides

However, this barrier is not insurmountable.

infection is the receptor conformation on the host cells.

Avian type: sialic acid (SA)-α2,3- Gal-terminated saccharides



### Avian Influenza in Poultry



- AIV is not normally found in domestic ducks, chickens and turkeys
- Transmission of AIV from wild birds to domestic poultry species occurs commonly (ducks>turkeys> chickens)
- AIV on rare occasions may become established in chickens and turkeys and result in serious disease outbreaks
- AIV once adapted to chickens and turkeys can be difficult to eradicate

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# Transmission

- Transmission from bird to bird occurs as a result of close proximity between infected and naive hosts.
- Direct contact with infected birds or with contaminated exudates or droppings are necessary for transmission
- This also indicates that airborne spread over large distances is an unlikely event.



### Transmission



- Bird-to-bird transmission is extremely complex and depends on:
  - the strain of virus,
  - the species of bird
  - environmental factors.
- In both natural and experimental infections:

HPAI are poorer transmitted than LPAI. Because spread related to the amount of virus released by the respiratory or intestinal route.



Clinical signs-LPAI



#### Turkeys :

- · Highly susceptible
- In pullets, LPAI infections cause anorexia and respiratory distress (rales, snicking and light coughing, and swelling of infraorbital sinuses)
- · Significant mortalities may be observed
- · Drastic drops in egg production is big problem





# Clinical signs-LPAI

#### Chickens:

- · Less susceptible than turkeys
- In broiler chickens, LPAI infections are often in-apparent and, when present, can be confused with other conditions.
- Anorexia and mild respiratory distress (rales, snicking and light coughing,
- Some LPAI viruses have been reported to cause serious health problems in broilers. e.g. H9N2 subtype and significant mortality (upto 50%)
- In laying birds: eggs are misshapen and/or with loss of colour may be laid in significant quantities.















#### · Chickens:

- · Mild and restricted pulmonary and tracheal congestion
- · Extensive hyperaemia of the respiratory system
- In broiler breeders, Ovarian follicles often appear haemorrhagic, oedematous and colliquated. The oviduct may be oedematous, with
- catarrhal or fibrinous egg-yolk peritonitis
  - · Mild pancreatitis is occasionally seen.
- Turkeys:
  - Caseous clot in the sinuses and trachea, which may cause suffocation.
  - Edematous trachea and lungs and congested Fibrinous air sacculitis

  - The spleen is often enlarged and congested



# **Clinical signs-HPAI**

#### Chickens:

- · Highly susceptible
- · The transmission is very fast (litter system) and flock mortality may be as high as 100%
- Anorexia, depression and cessation of egg-laying in breeders
- Nervous signs, characterized by prostration, complete reluctance • to move, tremors of the head, paralysis of the wings and incoordination
- Cyanosis of the comb and wattles •
- Haemorrhages on the shank •
- Sudden death occurs in a recumbent position and is preceded by • pedalling movements and gasping.









#### □ Turkeys:

- Some birds found dead prior to any clinical signs.
- 100% flock mortality may be observed
- Sudden and dramatic drop in food consumption
- Nervous signs, mainly tremors and incoordination.
  The birds exhibit shaking of the head, paralysis of the wings, abnormal gait
- · Dramatic and acute nature of the nervous signs





#### · Chickens:

- · Congested internal organs
- · Swelling of the head and upper neck
- Haemorrhages and cyanosis of the skin (wattles, combs and legs)
- Haemorrhages on serosal or mucosal surfaces and abdominal fat
- Haemorrhages on the epicardium, pericardium
- · Pancreas: focal to diffuse necrosis of the acinar ce
- The lungs and trachea are congested







#### • Turkeys:

- · Congested internal organs
- Peracute nature of the disease no gross lesions
- Haemorrhages on serosal or mucosal surfaces and foci of necrosis within parenchyma of visceral organs.
- Pinpoint haemorrhages are seen on the epicardium.
- Congestion and necrosis



# In ducks and geese



- Clinical signs
- · Enlarged nerves and lymphoid tumors in various viscera.
- The absence of bursal tumors helps distinguish this disease from lymphoid leukosis
- Marek's disease can develop in chickens as young as 3 wk old
- Histology
- Histochemistry and PCR, respectively.













# In ducks and geese



#### Clinical signs

- · Ducks and geese are known to be more resistant
- In some cases causing > 50% mortality.
- Muscovy ducks exhibit nervous signs such as incoordination
  and tremors
- Pekin ducks are also considered to be resistant clinically
- Clinical signs include conjunctivitis and mild depression, followed by nervous signs such as torticollis, incoordination, tremors and seizures





# In ducks and geese

- Lesions
- · Stomach and gizzard
- Haemorrhages on the surface of the pancreas and trachea and air sacculitis
- Hyperaemia and pinpoint haemorrhages may be found on the surface of the brain
- Pancreatitis



# Diagnosis

### Virus Isolation

Samples – any (tissue, swabs)

### Advantages

- Gold standard
- Sensitive all subtypes
- Disadvantages – Expensive and labor intensive
- False negatives (sample
- mishandling)
- Special facilities needed





# Diagnosis

- Serologic Diagnosis
  AGID, ELISA, HI, NI
- Virus isolation Allantoic sac inoculation then HA
- Antigen capture
- Molecular diagnostics
  RT-PCR & Real-time rt-PCR



## **Prevention and control**

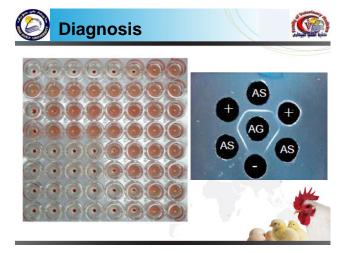
□Stamping out-identify infected flocks and destroy them to prevent spread to other flocks

□Vaccination in conjunction with stamping

out

□Vaccination only







spread

### To Vaccinate or Not To Vaccinate

• Vaccines will prevent clinical disease, but not infection

Good vaccines, properly administered, can reduce virus

· Vaccines will adversely affect export markets

· Bad vaccines may contribute to virus spread

· Costs of vaccination are not insignificant

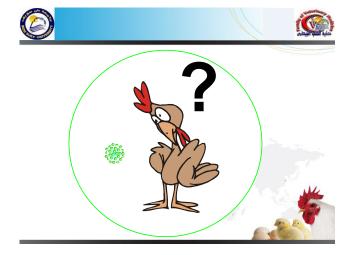
shedding from infected birds and reduce chance of virus



### **Stamping Out**



- This has been the method used in the U.S. for most foreign animal diseases including Avian Influenza
- Requires both good veterinary infrastructure and a diagnostic network
- Can be the most cost effective if outbreaks identified early
- Approach not practical when a disease is widespread in the country





### Vaccination

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- Proper vaccination programs must also include good surveillance, education, quarantines and animal movement controls
- Vaccination can be used to reduce the susceptible population, and when used with stamping out may be an effective tool
- Vaccination without the proper controls may reduce disease, but will not eliminate it