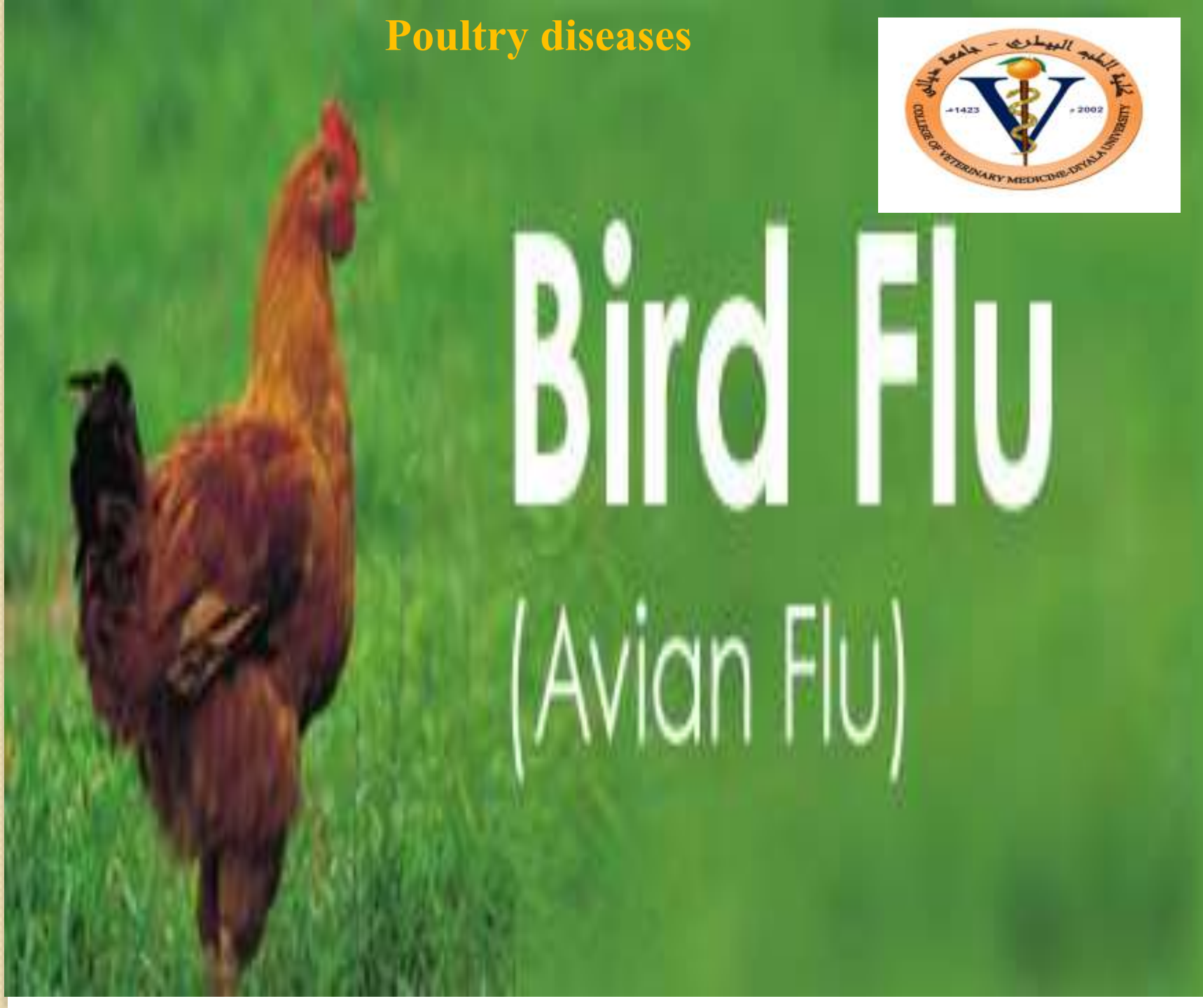
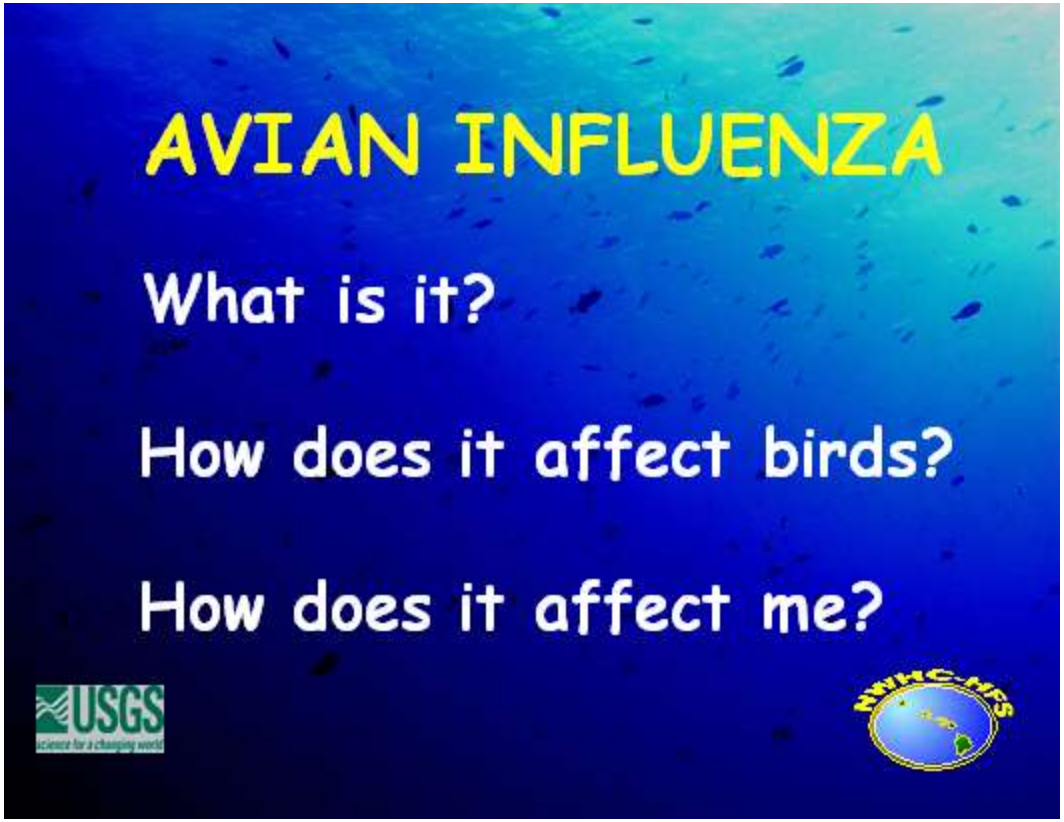
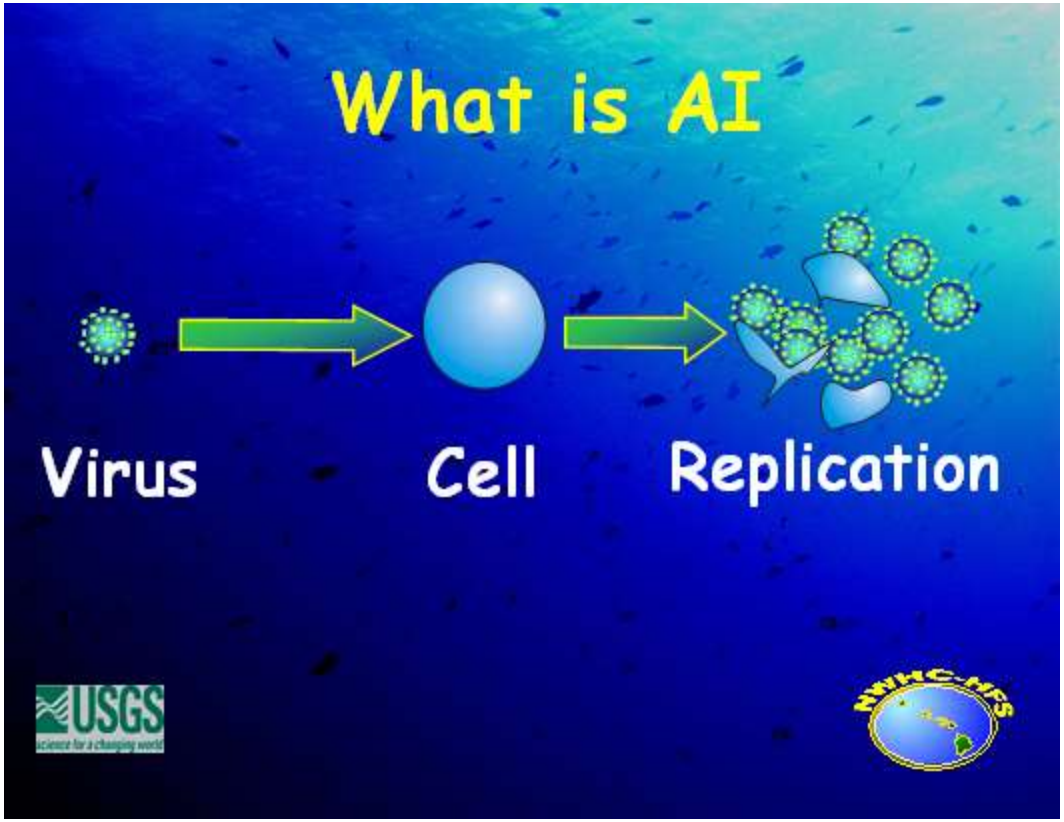
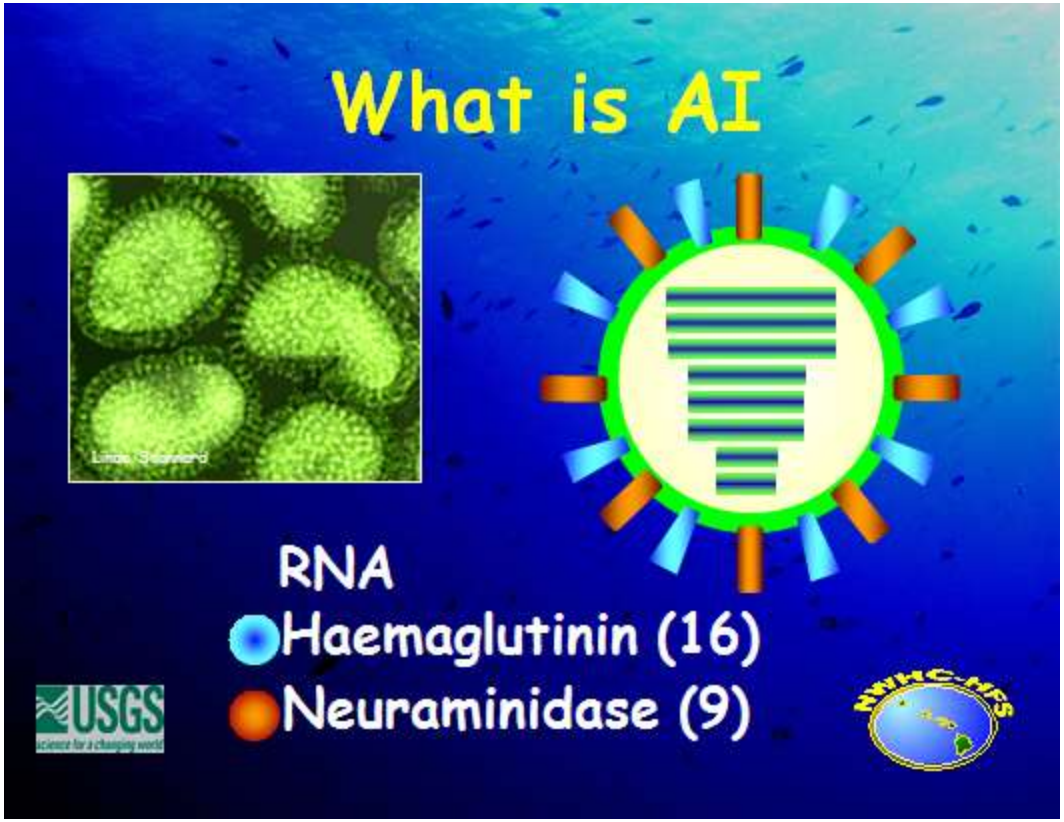
Poultry diseases




AVIAN INFLUENZA
What is it?
How does it affect birds?
How does it affect me?
USGS
NWHC-HFS


What is AI
Virus
Cell
Replication
USGS
NWHC-HFS


What is AI
RNA
Haemaglutinin (16)
Neuraminidase (9)
USGS
NWHC-HFS

Avian Influenza

- Avian influenza refers to infection of birds with avian influenza type A viruses which was the respiratory infection of chickens and turkeys characterized by upper respiratory involvement, mortality and decreased egg production in adults birds.
- From the late 1870s to 1981, HP AI was known by various names including fowl plague (most common), fowl pest, bird plague.
- Infection of domestic poultry by avian influenza (AI) viruses typically produces syndromes ranging from asymptomatic infection to respiratory disease and drops in egg production to severe, systemic disease with near 100% mortality.
- Because of the **zoonotic as well as pandemic** potential, the disease gained much public health importance.

Water birds and migratory birds act as carriers and spread the infection.

These viruses occur naturally among wild aquatic birds worldwide and can infect domestic poultry and other bird and animal species.

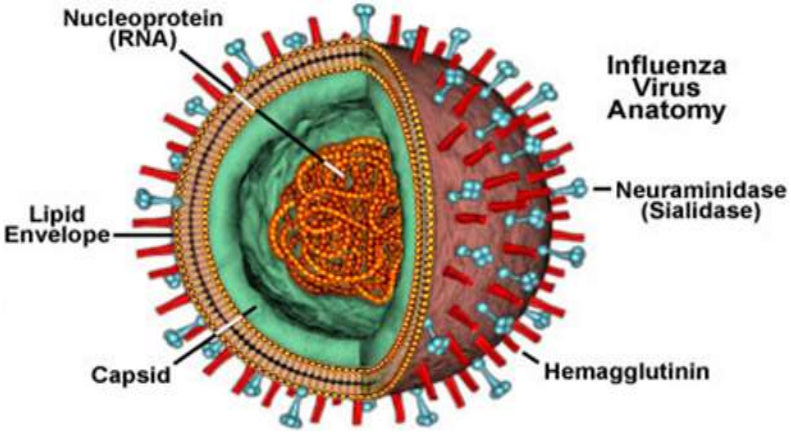
Wild aquatic birds can be infected with avian influenza A viruses in their intestines and respiratory tract, but usually do not get sick. However, avian influenza A viruses are very contagious among birds and some of these viruses can sicken and even kill certain domesticated bird species including chickens, ducks, and turkeys.

In the last 15 years the field of avian influenza (AI) has undergone something of a revolution. Not just because of the actual zoonoses and the potential threat of a pandemic emerging, which has highlighted public awareness, but also because many aspects of the epidemiology of AI infections in poultry and other birds appear to have changed dramatically. Not only have the number of outbreaks of the highly pathogenic disease (HPAI) increased in the last 10 years, but the impact in terms **of the number of birds involved and the costs of control** disease have dramatically escalated. In addition the apparently unprecedented emergence and spread of the HPAI H5N1 virus in **SE Asia** and beyond has brought AI to the forefront of important animal diseases.

ETIOLOGY:Classification

Influenzavirus A genus of the Orthomyxoviridae family , of which three types (A, B and C) are recognized. . They are enveloped, negative stranded RNA viruses, virions are typically spherical to pleomorphic but can be filamentous. The surface antigens haemaglutinin (H) and neuraminidase (N) are of particular importance and are used in the classification of the subtype A viruses.

Influenza subtypes A viruses naturally infect the intestinal tract of wild birds and are frequently asymptomatic in natural hosts. Individual virions range in diameter from 80—120 nm. The surface is covered by two types of glycoprotein projections (10—14 nm in length and 4—6 nm in diameter).



At present sixteen hemagglutinin subtypes (H 1-16) and nine neuraminidase subtype (N 1-9) of influenza A viruses have been recognized, all this types were extremely variable in virulence. **H5 and H7 are the most dangerous**, highly virulent AI viruses cause the disease fowl plague.

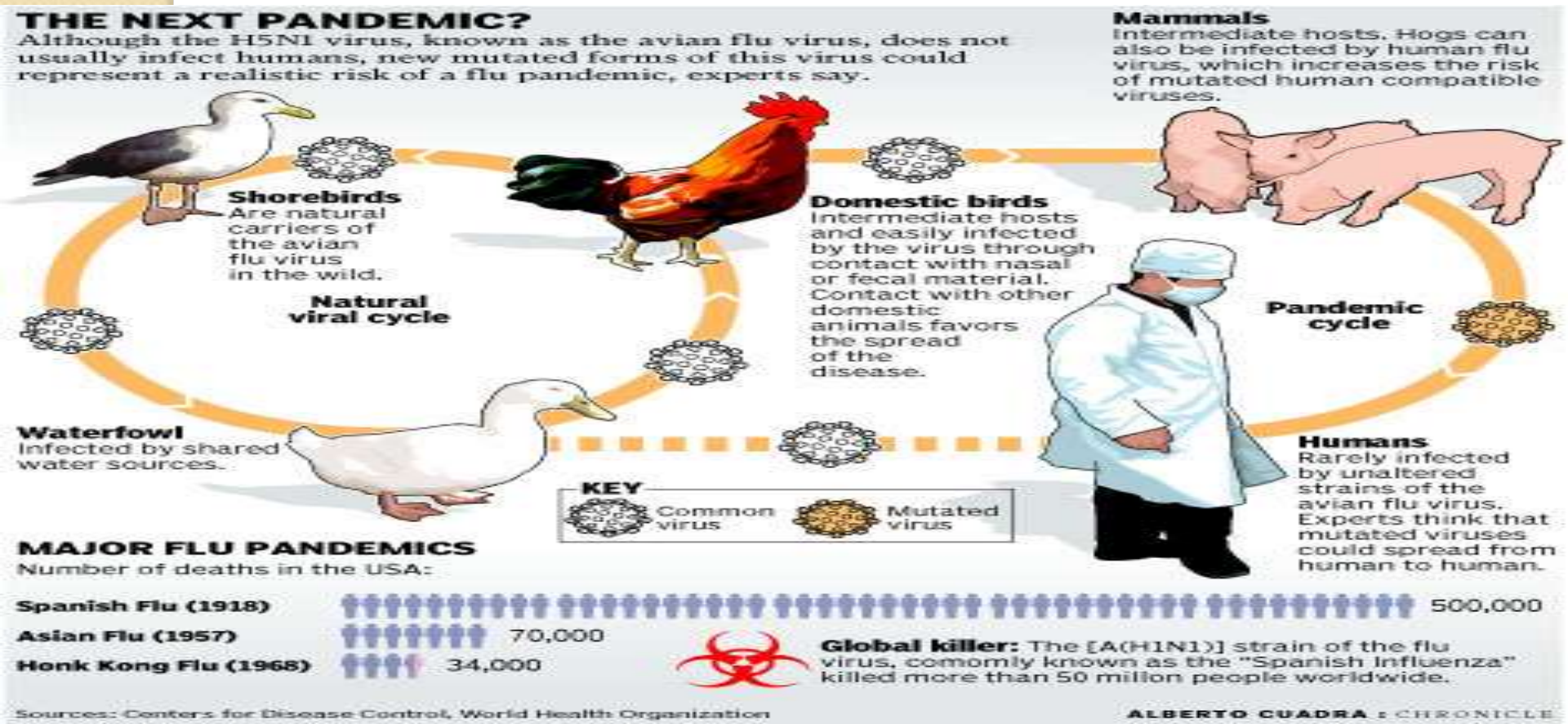
The nucleocapsid is helical.

The virus **have 2 Pathotypes:**

- **HPAI**
- **LPAI**

- **Incubation Period**

Variable from as short as a few hours in intravenously inoculated birds to 3 days in naturally-infected individual birds and up to 14 days in a flock, whereas the course of disease continue from 1-2 weeks depends on strain of the virus and theroute of exposure.



Economic Significance

Economic losses from avian influenza have varied depending on:

1- The strain of virus
2- Species of bird infected
3- Number of farms involved
4- Control methods used
5-The speed of implementation of control or eradication strategies.

❖ Generally, the greatest losses have occurred during epizootics of HP AI in domestic poultry raised on commercial farms of intensive production areas which lead to:

1-Direct losses in HP AI outbreaks have included depopulation and disposal costs.
2- High morbidity and mortality losses.
3- Quarantine and surveillance costs.
4- Indemnities paid for elimination of marketing birds.

- **Incidence and Distribution**
- Avian influenza viruses have a worldwide distribution.
- The greatest variety of Avian Influenza viruses has been isolated from wild birds, particularly from waterfowls.
- These birds perpetuate only viruses of low pathogenecity.
- Waterfowls are resistant to the disease induced by HPAI viruses.
- Domestic Poultry does not appear to be the natural host of these viruses, therefore the degree of adaptation to the host is low and this could possibly explain why documented virus mutation has virtually always occurred in domestic poultry.
- Contact with infected birds, ethnic slaughter house and distribution also very important, live bird markets are a problem.

Transmission

AI virus is excreted from the nares, mouth, conjunctiva, and cloaca of infected birds into the environment because of virus replication in the respiratory, intestinal, renal, and/or reproductive organs.

The virus is transmitted by direct contact between infected and susceptible birds or indirect contact through aerosol droplets or exposure to virus-contaminated fomites.

Aerosol generation from the respiratory tract is a significant mode of transmission because of high virus concentrations in the respiratory tract, but the large volume of lower concentration AI virus in infected feces makes fomites a major mode of transport. Thus, AI viruses are readily transported to other premises by people (contaminated shoes and clothing) and equipment shared in production, live-haul, or live-bird marketing.

Morbidity and Mortality

Variable

Usually doesn't exceed 10% unless fowl plague (high path) virus. Then can reach 80-100% mortality.

Susceptibility to Chemical and Physical Agents

Avian influenza viruses are relatively unstable in the environment.

Physical factors such as heat, extremes of pH, and dryness can inactivate AI viruses. Because AI viruses have lipid envelopes, they are inactivated by **organic solvents and detergents**, such as **sodium desoxycholate** also AI virus can be destroyed by chemical inactivants such as **aldehydes (formaldehyde or gluteraldehyde).**

Pathogenicity

For regulatory purposes, AI viruses from poultry are classified as HP (fowl plague-like) or non-HP. The term “mildly pathogenic (MP)” is most often used in lieu of non-HP

Clinical Signs:

However, clinical signs of disease are extremely variable and depend on other factors including host species, age, sex, concurrent infections, acquired immunity, and environmental factors.

Mildly Pathogenic Avian Influenza Viruses results from infection by viruses, of any HA or NA subtype, but with co-infection by secondary pathogens which lead to:

1- The mortality rates vary but range from 5—50% with the highest mortality occurring in young birds.

2-Most infections by MP AI viruses in wild birds produce no clinical signs.

3-In domestic poultry (chickens and turkeys), clinical signs reflect abnormalities in the respiratory, digestive, urinary, and reproductive organs.

4-Mild to severe respiratory signs such as coughing, sneezing,
rales, rattles, and excessive lacrimation.

5-In layers and breeders, hens may exhibit increased broodiness and decreased egg production.

6- In addition, domestic poultry will exhibit generalized clinical signs including huddling, ruffled feathers, depression, decreased activity, decreased feed and water consumption, and occasionally diarrhea.

Highly Pathogenic Avian Influenza Viruses:

highly virulent clinical group results from infection by HP H5 or H7 AI viruses usually in chickens.

highly fatal systemic disease that affects most organ systems including the nervous and cardiovascular systems.

lead to:

1-Depression is common

2-Decreased feed and water consumption

3-Precipitous drops in egg production

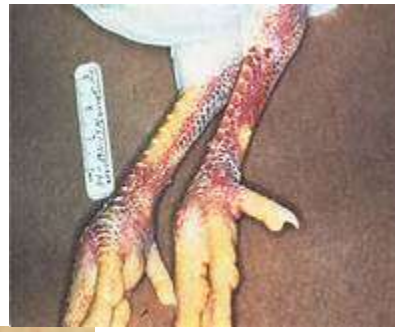
4-Mild to severe rales, sneezing, and coughing sinusitis and edema of head and wattles.

5-Diarrhea

6-Whitens the shell of broiler breeder eggs. Shell color isn't being laid down in the oviduct..

7- Morbidity and mortality approach 100%.

The last spread of AIV in Diyala 2016



Cyanosis

Dyspnea

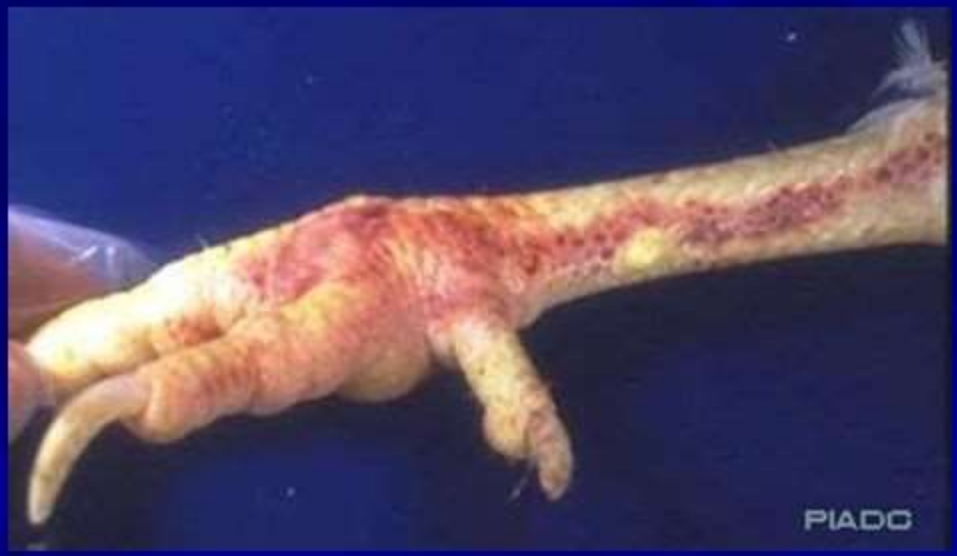
Sinusitis

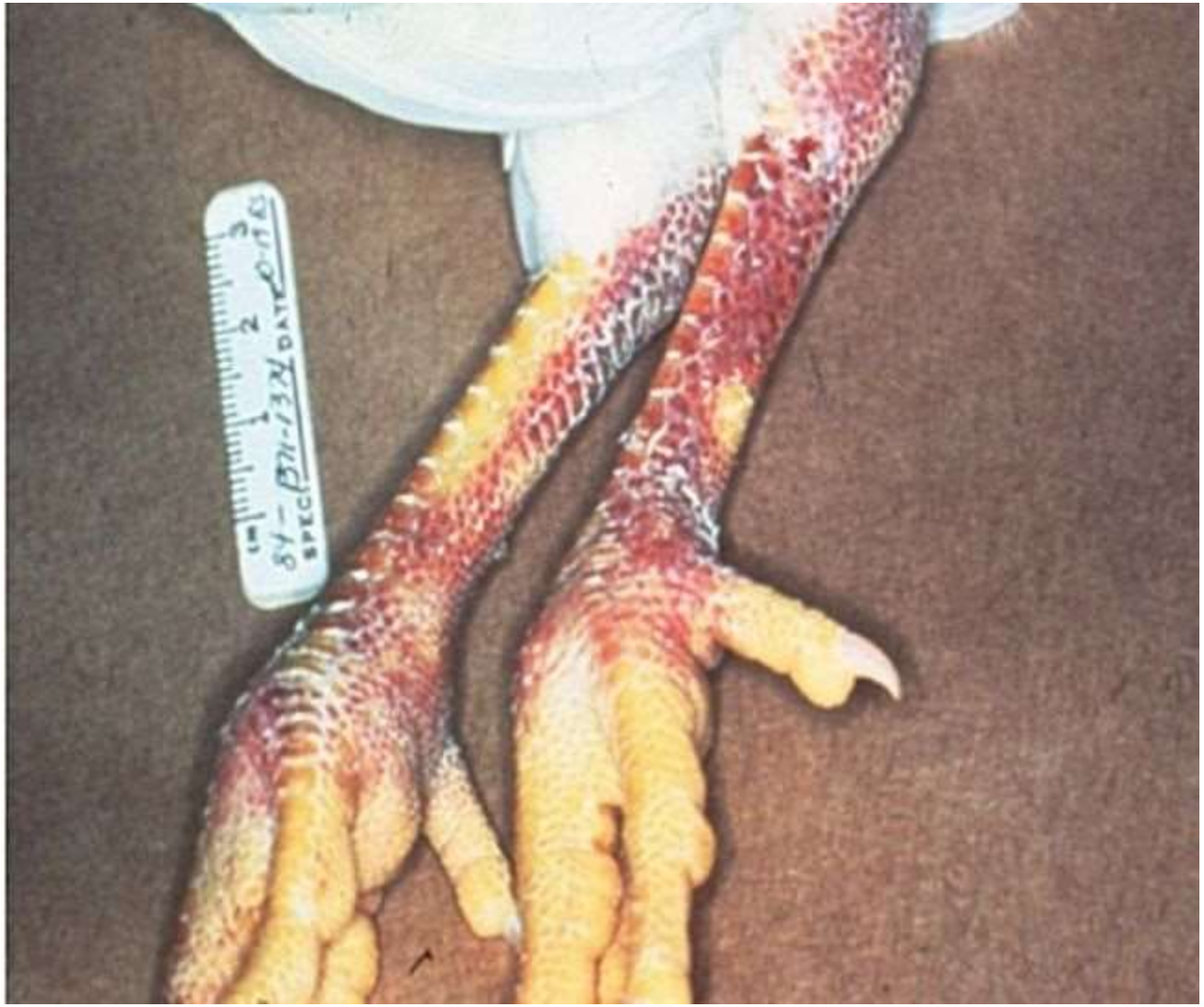
Ruffled feathers

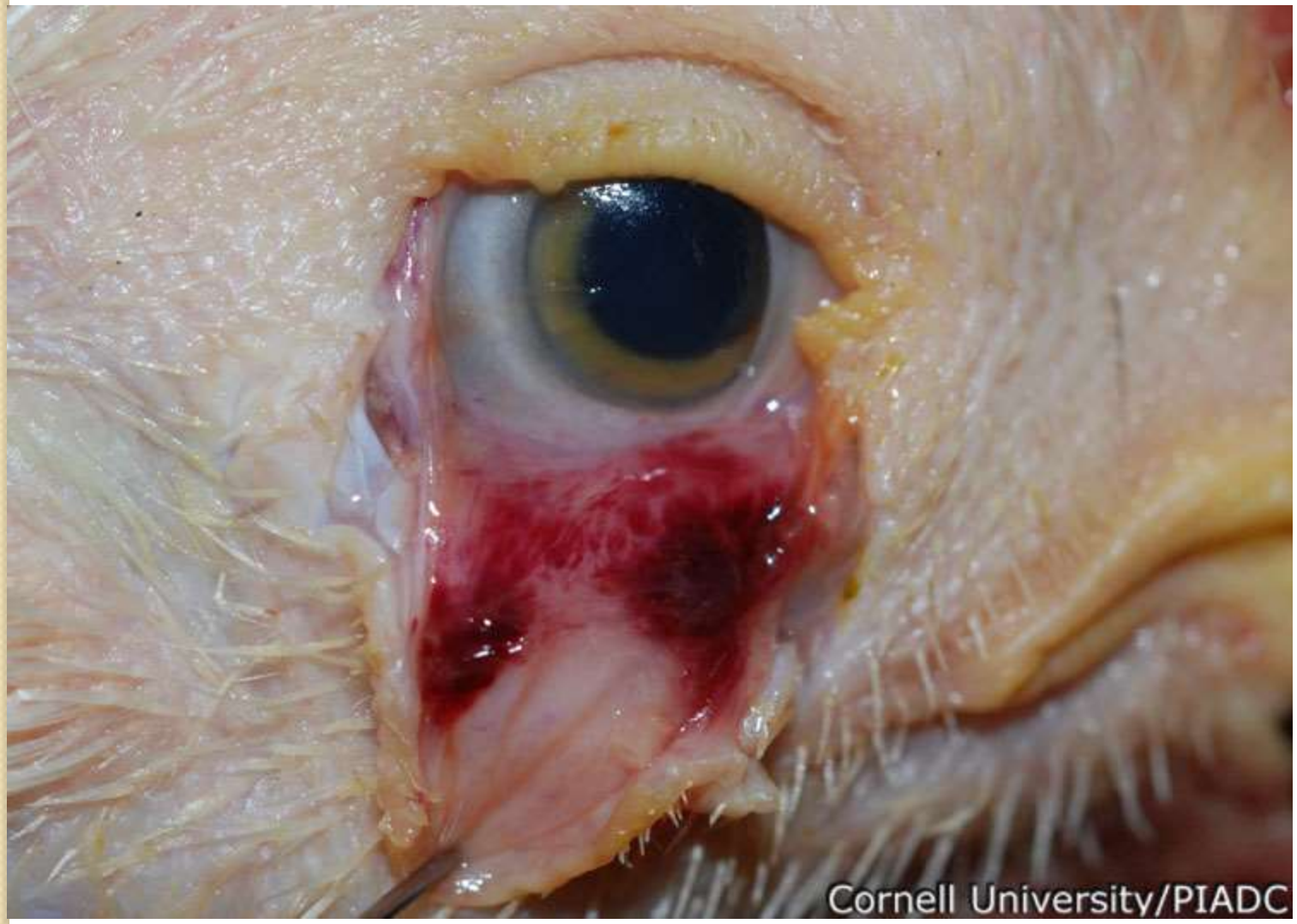
Respiratory Symptoms

Clinical Signs

- Subcutaneous hemorrhage of shanks






Cornell University/PIADC

Swollen blue, *cyanotic* combs and wattles

Gross lesions

Have been extremely variable with regard to their location and severity, depending greatly on the host species, pathogenicity of the infecting virus, and presence of secondary pathogens.

Mildly Pathogenic Avian Influenza Viruses:

In poultry, the most frequent lesions are in the respiratory tract.

1-Sinusitis with mucopurulent to caseous exudate.

2-The tracheal mucosa can be edematous with congestion and occasionally hemorrhages.

3-Fibrinous to fibrinopurulent air sacculitis may be present.

4-The infraorbital sinuses may be swollen.

5-Inflammatory exudates in the oviducts of laying birds, and the last few eggs laid will have reductions in calcium deposition.

6-Eggs may be misshapen and fragile with loss of pigmentation.

7-Ovaries will undergo regression.

Highly Pathogenic Avian Influenza Viruses:

1-Swelling of the head, face, upper neck, and feet are common as the result of subcutaneous edema and may be accompanied by petechial to ecchymotic hemorrhages.

2-Necrotic foci, hemorrhage, and cyanosis of the nonfeathered skin is common, especially wattles and combs.

3- Hemorrhages in proventriculus and heart.

4- Necrotic foci are common in pancreas, spleen, and heart, and occasionally in liver and kidney.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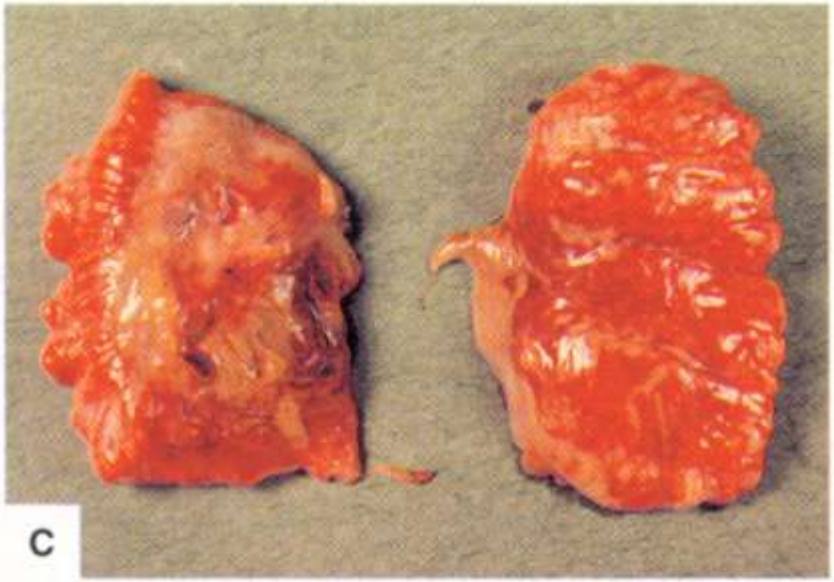
A


B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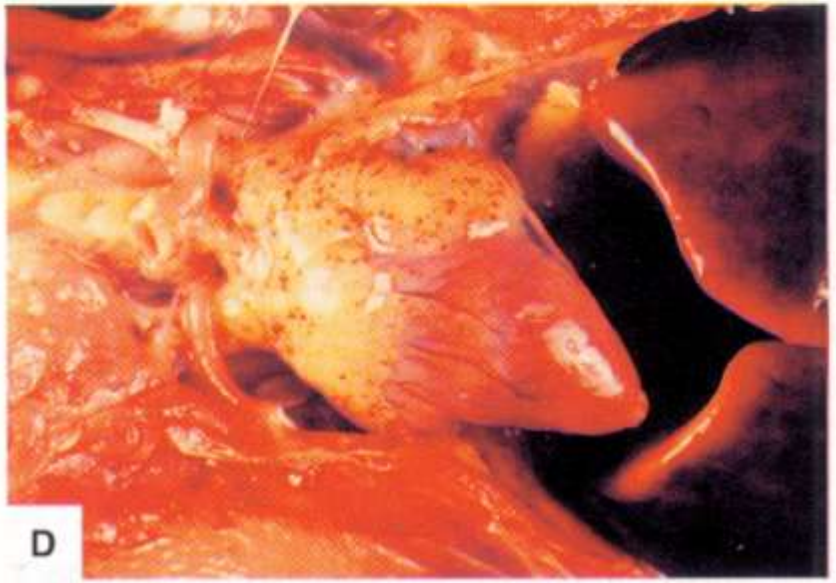

D


E


F

Pathogenesis

The process begins by inhalation or ingestion of infectious MP or HP AI virions. Because trypsin like enzymes in respiratory and intestinal epithelial cells allow cleavage of the surface hemagglutinin, multiple replication cycles occur in respiratory and/or intestinal tracts with release of infectious virions.

In poultry, the nasal cavity is a major site of initial replication.

HP AI viruses, the virions invade the submucosa, entering capillaries. The virus replicates within endothelial cells and spreads via the vascular or lymphatic systems to infect and replicate in a variety of cell types in visceral organs, brain, and skin.

MP AI viruses, replication usually is limited to the respiratory or intestinal tracts. Illness or death is most often from respiratory damage, especially if accompanied by secondary bacterial infections such as E.Coli.

Differential Diagnosis

1-Newcastle disease virus

2-Avian pneumovirus and other paramyxoviruses

3- infectious laryngotracheitis

4- infectious bronchitis

5- chlamydia, mycoplasma, and other bacteria

Microscopic

Mildly Pathogenic Avian Influenza Viruses:

produce pneumonia varying in character from ventromedial fibrinocellular to peribronchiolar lymphocytic.

In severe cases, the pneumonia may be diffuse with air capillary edema. Heterophilic to lymphocytic tracheitis and bronchitis have been common.

Birds that die from MP AI have lymphocyte depletion and necrosis or apoptosis of lymphocytes in the cloacal, bursa, thymus, spleen, and other areas with lymphocyte accumulations.

Highly Pathogenic Avian Influenza Viruses:

the histologic lesions consist of multiorgan necrosis and/or inflammation. The most severely affected tissues are brain, heart, lung, pancreas, and primary and secondary lymphoid organs. Lesion in the brain showed Lymphocytic meningoencephalitis with focal gliosis, neuronal necrosis

Focal degeneration to multifocal-diffuse coagulative necrosis of cardiac myocytes has been reported.
Other common lesions include necrosis in skeletal myofibers, kidney tubules, vascular endothelial cells, corticotrophic cells of adrenal.

Diagnosis

A definitive diagnosis of avian influenza is established by

1) Direct detection of AI viral proteins or genes in specimens such as tissues, swabs, cell cultures, or embryonating eggs.

2) Isolation and identification of AI virus. By inoculated chicken embryos, 10—11 days old, via the allantoic cavity with approximately 0.2 mL of sample.

3) Serologic tests are used to demonstrate the presence of AI specific antibodies, which may be detected as early as seven days after infection. ELISA assays have been developed to detect antibodies to avian influenza viruses. Once influenza is detected by ELISA, HI tests can be used to determine the HA subtype.

Prevention:

1- Biosecurity is the first line of defense

2-Separation of susceptible birds from infected birds because transmission can occur when susceptible and infected birds are in close contact.

3- Another consideration is that there should be no contact with recovered flocks because the length of time birds within a population shed virus is not clearly defined.

4- The wild birds should be considered a major source of infection for domestic birds, particularly those on open range, so it is important to reduce the contact between these two groups.

5-Bury the dead birds.


I got Fever

Control

1-Control bird-to-bird transmission by cleaning and disinfection equipment.

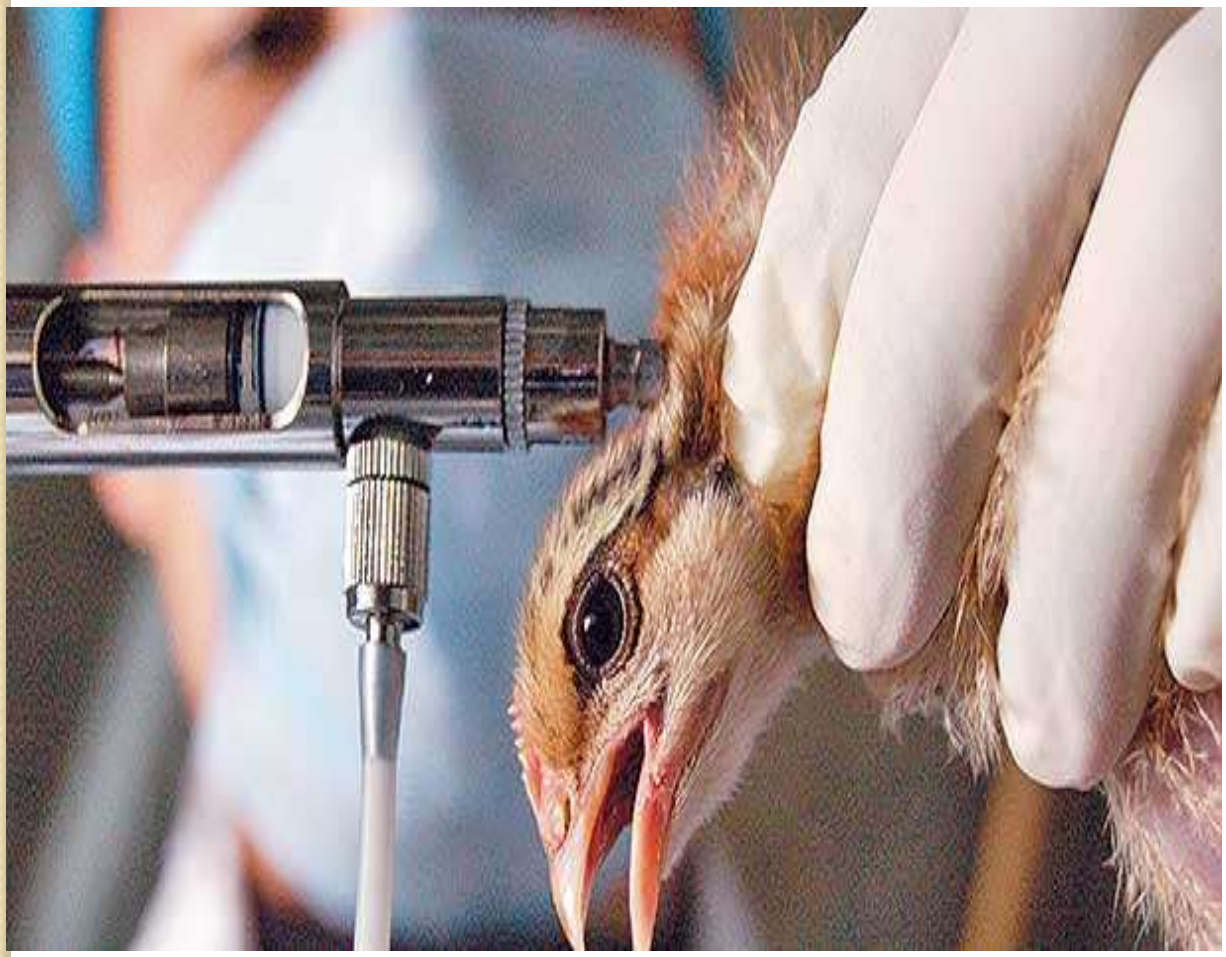
2-All methods for controlling the spread of influenza are based on preventing contamination and controlling the movement of people and equipment.

3-With an HP AI virus governmental eradication procedures (quarantine, slaughter, disposal, and clean-up) are employed.

4-Vaccination by using inactivated influenza virus vaccines, which have been used to preventing clinical signs and mortality.

Treatment

Presently, no practical, specific treatment exists for avian influenza virus. Amantadine has been shown experimentally to be effective in reducing the mortality.




NOBILIS INFLUENZA H7