

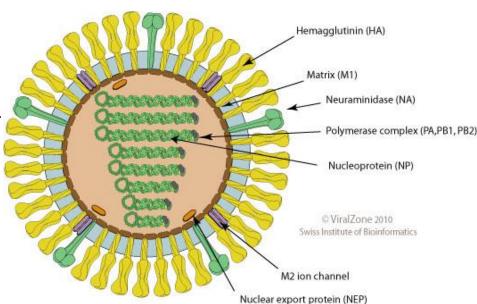
Avian, Swine & Equine Influenza

Greek myxa = mucus and orthos = correct or right.

AB's Veterinary Www.veterinarymicrobiology.in

Properties of Virus

- Enveloped virus
- Virions are pleomorphic, spherical, or filamentous
- Size:80-120 nm in diameter
- Genome: linear negative-sense, singlestranded RNA, divided into eight or seven or six segments,
- Nucleocapsid: helically, symmetrical
- Consist of an envelope with large peplomers surrounding eight (genus Influenzavirus A and Influenzavirus B), seven (genus Influenzavirus C), or six (genus Thogotovirus) segments
- There are two kinds of peplomers H & N
 Transcription and
- RNA replication occur in the nucleus





Classification

Group V: Negative sense ssRNA viruses

Family: <u>Orthomyxoviridae</u>

Genus: Influenzavirus A

• <u>Alphainfluenzavirus</u> - A

•<u>Betainfluenzavirus</u>- B

•Gammainfluenzavirus- C

• Deltainfluenzavirus

• *Isavirus*

Quaranjavirus

Thogotovirus

Subtype: <u>H1N1</u>

H5N1



Surface Antigens & Subtypes

18 HA and 11 NA for influenza A

Hemagglutinin (HA)

Function:

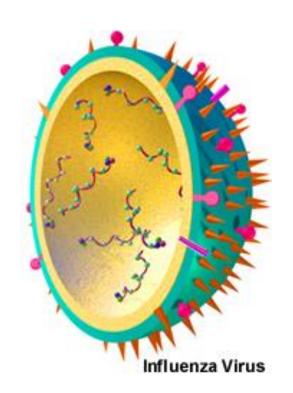
Sites for <u>attachment</u> to infect host cells

Neuraminidase (NA)

Function:

Remove neuraminic acid from mucin & release from cell

Antigenic drift Antigenic shift





Antigenic Drift: Is the minor mutation of the surface glycoproteins, namely haemagglutinin (HA) & Neuraminidase (NA) of the influenza virus over a long period of time.

Antigenic Shift: Is a major change to the virus structure to create absolutely new subtype of virus by genetic reassortment.

Avian Influenza Virus



Highly pathogenic AI (HPAI)

Any influenza virus lethal for 6,7 or 8 of eight, 4-8 weeks old susceptible chickens within 10 days following i/v inoculation with 0.2 ml of a 1/10 dilution of a bacteria free, infective allantoic fluid.

Any virus with IVPI greater than 1.2

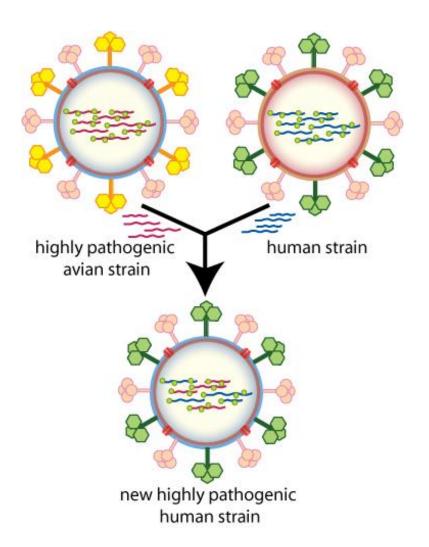
Low pathogenic AI (LPAI)

Low pathogenicity avian influenza

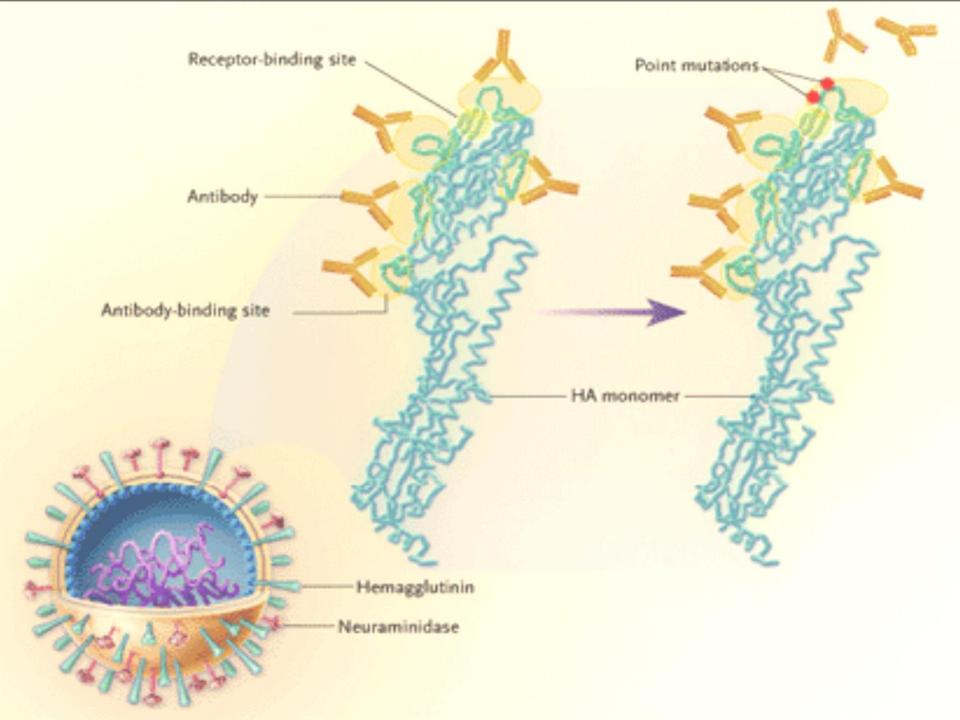
(LPAI) viruses typically cause little or no clinical signs in infected poultry.

H5 & H7 isolates that are not virulent for chickens and donot have an HAO cleavage site aminoacid sequence similar to those that have been observed in HPAI virus.



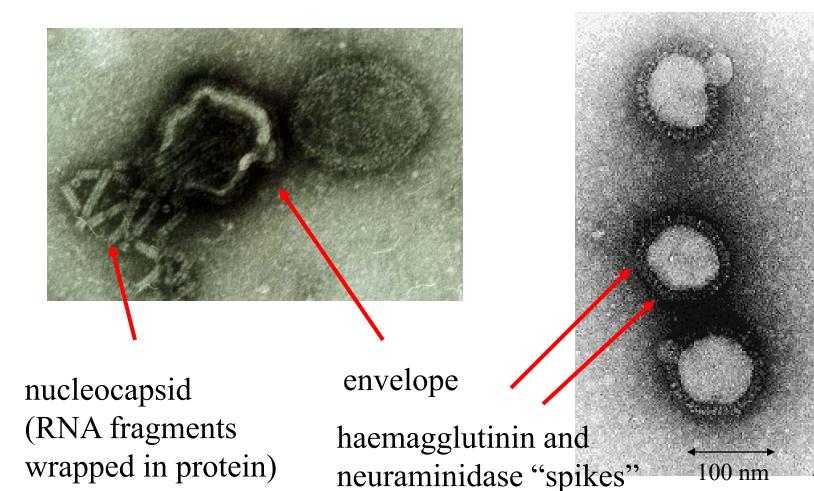


How antigenic shift, or reassortment, can result in novel and highly pathogenic strains of human influenza



Influenza virions

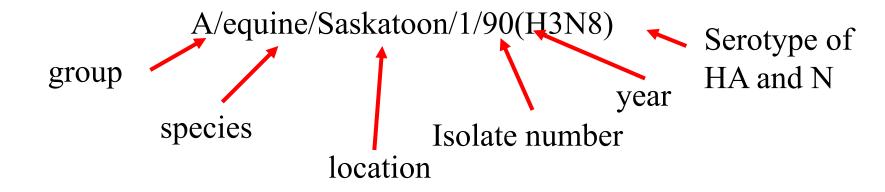




In envelope



Nomenclature



- •A/equine/Prague/1/56(H7N7)
- •A/fowl/Hong Kong/1/98(H5N1)
- •A/swine/Lincoln/1/86(H1N1)



Resistance to physical and chemical action

Temperature: Inactivated by 56°C/3 hours; 60°C/30 min.

pH: Inactivated by acid pH

Chemicals: Inactivated by oxidising agents, sodium dodecyl

sulphate, lipid solvents, B-propiolactone

Disinfectants: Inactivated by **formalin** and iodine compounds

Survival: Remains viable for long periods in tissues, feces and

also in water



HOST Aquatic birds, Poultry birds, Human, Pig, Horse, Seals **TROPISM** Epithelial respiratory cells

TRANSMISSION

Mammals: Respiratory, Zoonosis, animal contact

Birds: Fecal-oral route from contaminated water





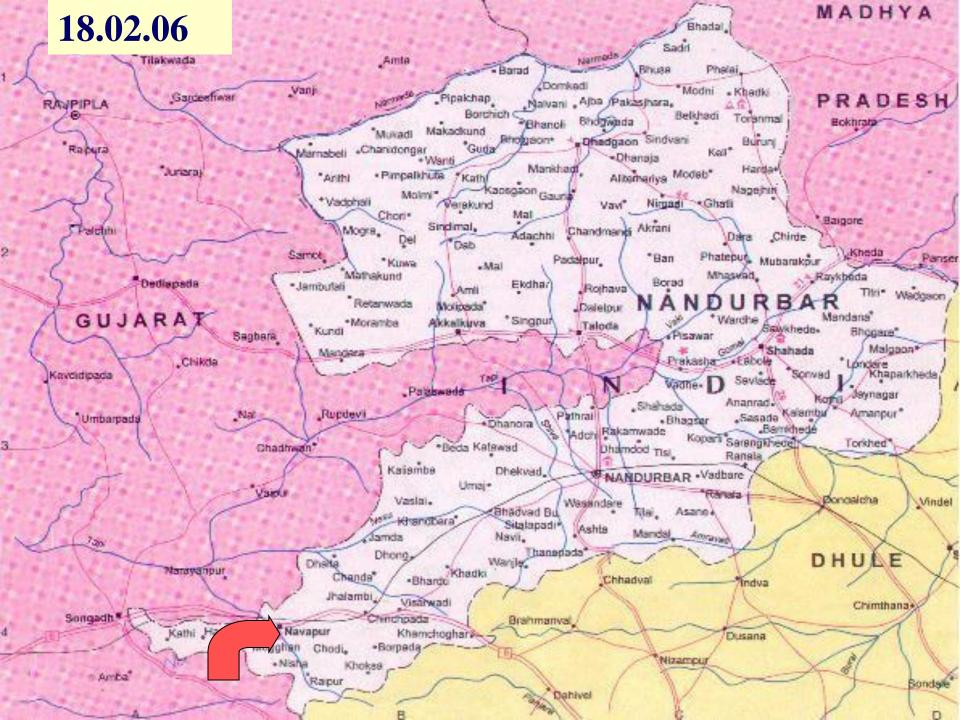
What is Avian Influenza (AI)?

- Avian Influenza is an infectious viral disease of birds caused by type 'A' strains of the influenza virus. The flu virus appears naturally among birds.
- Wild migratory birds such as ducks, geese, gulls and shorebirds are natural carriers of the virus, but are resistant to severe infection from the virus.



HISTORY

- First noticed in Italy in the year 1878 killing a large number of birds
- The disease was named as fowl plague
- The causative agent as a virus was established in the year
 1901
- Relationship between human influenza A virus established in 1955

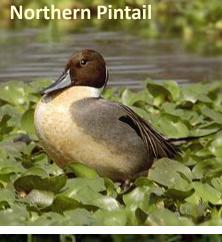




Avianinfluenza

Transmission

- Aerosol
- Contaminated water & food
- Inanimate Objects,
- Workers
- Migratory Birds

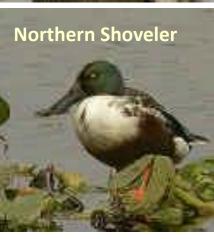






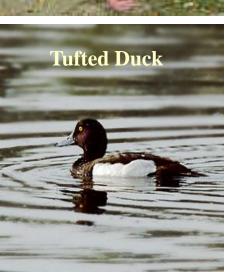












- •Migratory water fowls, most notably wild ducks are natural reservoirs of AIV
- ■The virus usually does not cause clinical disease in wild birds (with exceptions)



Clinical Signs

- Clinical signs are dependent on the virulence of the infecting virus and the species infected.
- In outbreaks of Highly Pathogenic Avian Influenza (HPAI) mortality can be up to 100%.
- Low Pathogenic Avian Influenza (LPAI) in chickens may even go unnoticed.

Symptoms of HPAI in chickens

Sudden Death
Depression

If survives for 48 hours:

Decreased Appetite
Cessation / drop in Egg Production
Swollen Blue Combs and Wattles
Coughing, Sneezing, respiratory distress
Lacrymation, edema of head, face and neck
Cyanosis of unfeathered skin - Comb,
Diarrhea











Symptoms of LPAI in chickens

Mild respiratory disease

Sinusitis, Depression

Decreased egg production



Material Collection

- Throat swab or cloacal swab
- Serum





Diagnosis

1. Isolation of Virus

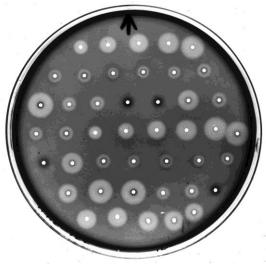
- 9-11 day-old embryonated chicken eggs (SPF)
- Cell cultures (Chicken embryo fibroblast)
- Demonstration of hemagglutination
- Strain virulence evaluation: intravenous pathogenicity index (IVPI) in 4-8 week-old chickens



Diagnosis

2. Serological tests

Hemagglutination inhibition tests (subtype specific serum)
Agar gel immunodiffusion
ELISA (Influenza A nucleoprotein)
Immunofluorescence technique
Single Radial Hemolysis





Diagnosis

3. Molecular Techniques

- Subtype specific Polymerase Chain Reaction
- Multiplex PCR
- Real time PCR
- RT-PCR



Differential diagnosis

- Acute fowl cholera
- Velogenic Newcastle disease
- Infectious laryngotracheitis
- Infectious Bronchitis



Principle of Vaccination

Effective HPAI Vaccine-

- Not only Protect against disease but also prevent shedding of virus.
- Vaccine with closer antigenic Match.
- Only in High Risk Areas/Endemic.





Avian Influenza Vaccines Available Abroad

Inactivated Adjuvanted Whole Virus Vaccine
Homologus (INTERVET)
Heterologus

DIVA Based Vaccines

Recombinant Vaccines

- Recombinant fowl pox-vectored vaccine that co-expresses the HA and NA of the A/goose/Guangdong/1/96 virus Merial
- A recombinant LaSota strain of Newcastle disease virus (NDV) expressing an H5 HA insert

List of Vaccine Manufacturers

Vaccination Schedule for Nobilis Influenza H5*

- a. Dosage: 0.5 ml per dose in birds older than 3 weeks of age, 0.25 ml per dose in younger birds.
- Administration: subcutaneously in the lower back of the neck or intramuscularly in older birds.
- c. Emergency Vaccination Schedule:

Primary vaccination administered to all poultry irrespective of age.

Booster vaccination administered 4 – 6 weeks later.

(If the primary vaccination was administered to birds younger than 3 weeks of age a third vaccination is recommended at 16 – 18 weeks of age)

d. Vaccination of Replacement Flocks:

Vaccination schedule is dependant on perceived risk of infection.

In <u>high risk</u> areas (active infection) primary vaccination (0.25 ml) is recommended at day old to establish immunity as early as possible.

Two booster vaccinations (0.5ml) are recommended at 4-6 and 16-18 weeks of age.

In areas with high infection pressure revaccination at midlay may be indicated.

^{*} The vaccination schedule for Nobilis Influenza H5, which is mentioned by Intervet in product related information, is based on existing registrations. However in the light of recent

Limitations to vaccination



In India- Vaccination not permitted / Not recommended

- Expensive
- No cross protection between 16 H subtypes
- Possible creation of reassortant virus-Update the vaccine annually.
- Two doses of 10ug.
- BSL3 Containment facilities for production of vaccine.
- Whole vaccine virus s/b preferred.
- Vaccine induced antigenic drift.
- Efficacy of vaccine in ducks.

Inactivated H5 and recombinant vaccine licensed in the U.S. for emergency in HPAI outbreaks



Human vaccine

Each dose contains neuraminidase and 15 µg of each of the following strains:

A/New Caledonia/20/99 (H1N1) A/Wisconsin/67/2005 (H3N2) B/Malaysia/2506/2004



Chemotherapy

- Prevent membrane fusion
 - Amantidine (Symmetrel)
 - Remantidine (Flumadine)
- Neuraminidase inhibitors
 - Zanamivir (Relenza)
 - Oseltamivir (Tamiflu)



Control Strategy



- 1) Biosecurity and quarantine
- Rapid Diagnostics and surveillance
 High level of true surveillance to detect the emergence of antigenic variants.
 (1st Week)
- 3) Elimination of infected poultry or controlled marketing of convalescent poultry. Culling of infected poultry reduces the viral load-& likelihood of transmission to human
- 4) Decreasing host susceptibility to the pathogen by vaccination- Vaccination to reduce the re-invasion of the virus in endemic areas.
- 5) Combined Antiviral therapy.
- 6) Education of personnel, owners, and villagers on disease transmission, prevention and control.
- 7) Political commitment & determined implementation.
- 8) Planning, communications, and preparation.



Swine InfluenzaPorcine Influenza

History

First report was observed in USA in 1918 Subtype- H1N1 Pigs as major reservoir

Host

Pigs of all age, Turkeys, Human

Transmission

Aerosol and direct contact with infected animals. Recovered animals sheds the virus for long time



Swine Influenza Porcine Influenza

Pathogenesis

Incubation period: 1-3 daysAfter entry virus multiplies in the mucosa of respiratory tract.

Develops rhinitis - May progress to Bronchopneumonia

Clinical signs

Severe paroxysms of coughing, dyspnoea, anorexia, oculo-nasal discharge, rise of temperature. Recovery after 5-7 days Secondary bacterial infection-Haemophilus suis Lesions include-Emphysema, hyperplasia of bronchial epithelial cells.





Laboratory diagnosis

Material collection: pharyngeal or nasal swab (50% GPB)

Isolation of virus-ECE-Allantoic cavity route-Confirm by HI test

Neuraminidase inhibition test

Prevention and control

Quarantine Symptomatic treatment

Inactivated vaccine available-No satisfactory protection



Swine Influenza

Human

Symptoms include fever, cough, sore throat, chills, weakness and body aches. Children, pregnant women and the elderly are at risk from severe infection.

People may experience:

Pain areas: in the muscles

Cough: can be dry

Gastrointestinal: diarrhoea, nausea, or vomiting

Whole body: chills, fatigue, or fever

Also common: headache, shortness of breath, or

sore throat

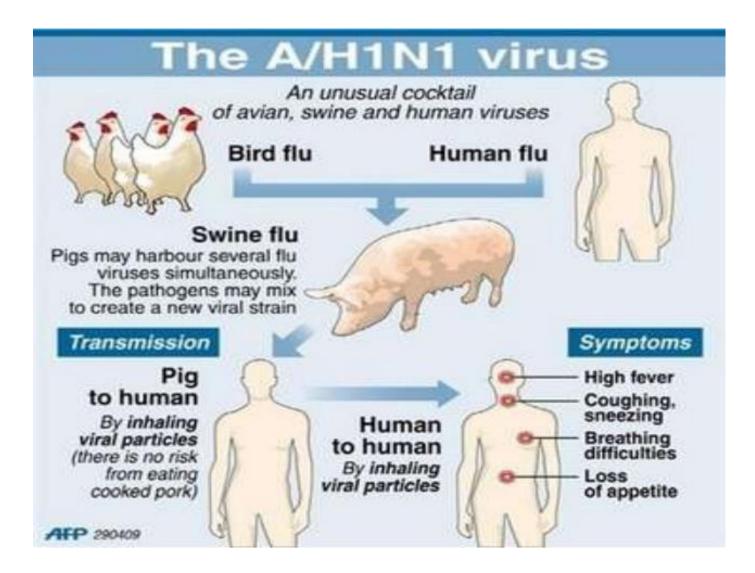
Transmission

The main route of swine flu virus spread between humans is exposure to the virus when someone infected sneezes or coughs, and the virus enters one of the potential mucous surfaces, or when a person touched something infected with the virus and subsequently touch their nose











Swine Influenza

- Prevent membrane fusion
 - Amantidine (Symmetrel)
 - Remantidine (Flumadine)
- Neuraminidase inhibitors
 - Zanamivir (Relenza)
 - Oseltamivir (Tamiflu)





History

First report was observed in Sovinova, Czechoslovakia in 1956 Subtype- H7N7, H3N3 –Florida, USA (1963) World wide in distribution

Host

All breeds and all ages of Horses, donkeys, mules are susceptible

Transmission

Aerosol and direct contact with infected animals.

International spread- Transport of horses for racing and breeding purpose



Pathogenesis

Incubation period: 1-3 days After entry virus multiplies in the mucosa of respiratory tract.

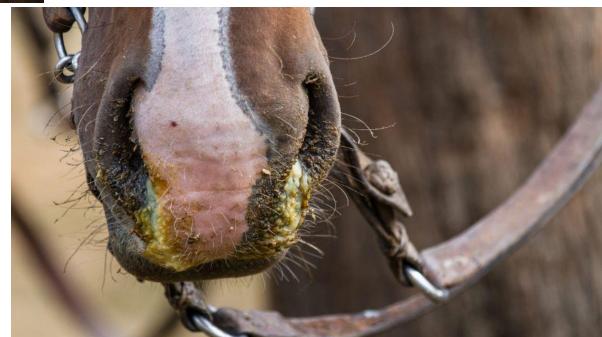
Develops rhinitis - May progress to Bronchopneumonia

Clinical signs

Coughing, dyspnoea, reddening of nasal mucosa, anorexia, oculonasal discharge, sudden rise of temperature. Swelling of pharyngeal lymph node, Recovery after 5-7 days Secondary bacterial infection-Mucopurulent nasal exudate Catarrhal bronchopneumonia









Laboratory diagnosis

Material collection: pharyngeal or nasal swab (50% GPB)

Isolation of virus-ECE-Allantoic cavity route-Confirm by HI test Neuraminidase inhibition test

Prevention and control

Quarantine and Biosecurity at stud farms

Equine influenza Bivalent inactivated vaccines available

Two doses at a interval of 3-4 weeks









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