POULTRY DISEASES


Poultry diseases
Viral diseases
Infectious
Bronchitis (IB)

IBV:

An acute, highly contagious respiratory disease of chickens. All ages infected; particularly a problem in laying flocks.

Chicks - growth suppression & predisposition to other diseases.

Hens - variable production loss and affects egg quality.

Turkeys resistant.

The virus is acquired following inhalation or direct contact with contaminated poultry, litter, equipment. **Vertical transmission within the embryo have never been reported**. But virus may be present on the shell surface of hatching eggs via shedding from the oviduct or alimentary tract.

(**IB**) regarded as one of the most important poultry diseases, causes **high morbidity** in all ages of chickens and **high mortality** in chicks **less than 4weeks old.**

Avian Infectious bronchitis(IB) was first described and reported by **Schalk** and **Hawn** in (1931) as an apparently new respiratory disease of baby chick in North Dakota,USA. It was later observed to infect all ages, sexes and breeds of chickens.

Since then it has been a cause of serious cause of IB economic loss in chickens. IB is known to be one of the major highly contagious diseases of the respiratory and urogenital tract of chickens.

The coronavirus of the chicken which causes **IB**, is one of the foremost causes of economic loss within the poultry industry, affecting the performance of both meat-type and egg-laying birds.

The virus replicates not only in the epithelium of upper and lower respiratory tract tissues, but also in many tissues along **the alimentary tract** , **kidney**, **oviduct** and **testes**. It can be detected in both respiratory and faecal material

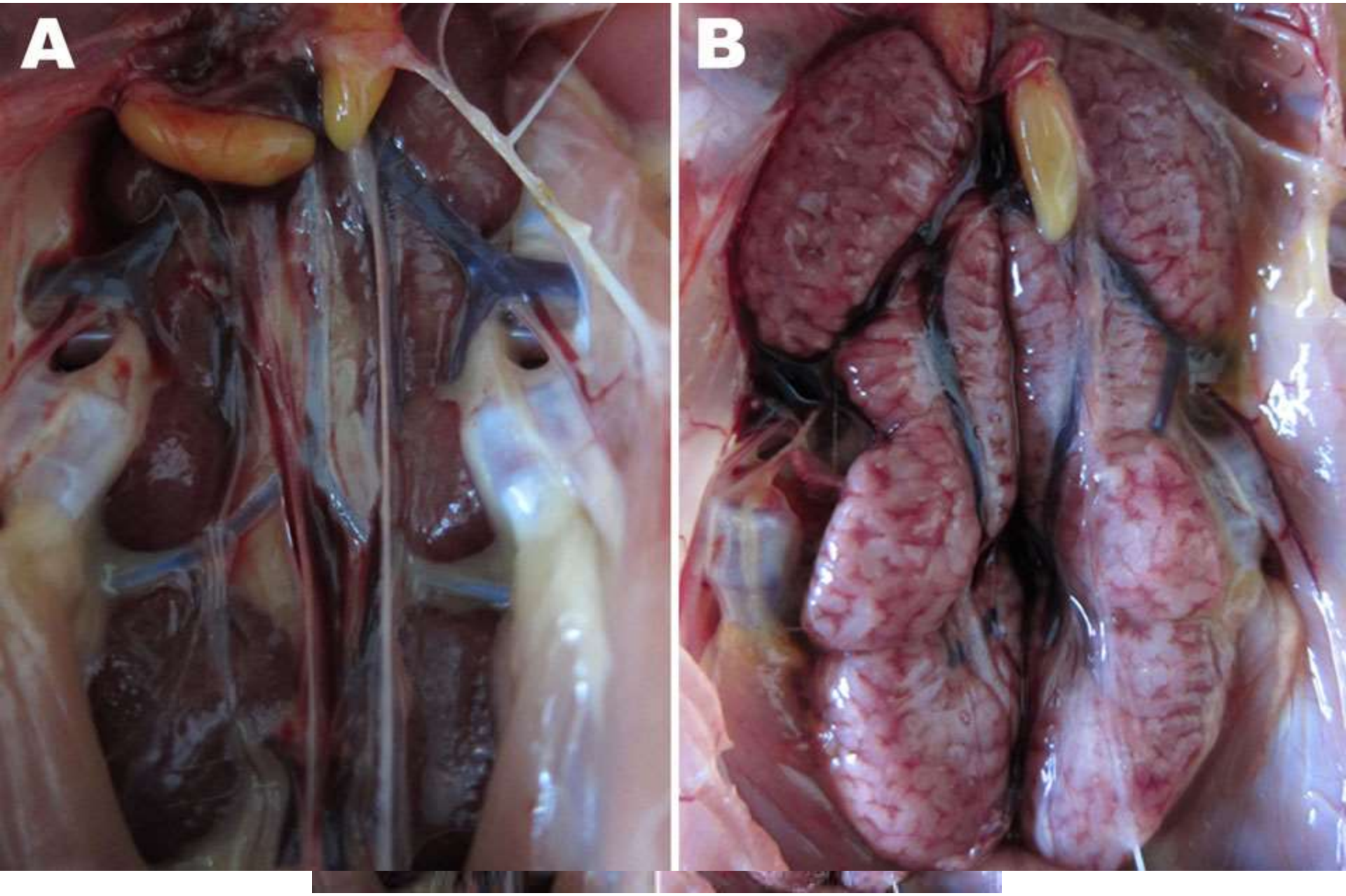
Three form of the disease
Respiratory,
Renal,
Reproductive

1-The respiratory form of the disease is characterized by **generalised distress**, **sneezing**, **coughing**, **tracheal rales** &**excessive mucus production in the bronchi**. In 1940s noted typical respiratory signs regarding of the IB infection.

2-The renal form: The nephrosis syndrome was first reported in Australia and later in USA with IB in the former being more sever.

In 1960s lesion observed in kidney characterized by depression, ruffled feathers, increased of water intake, scouring and wet litter are commonly observed, high mortality (up to 80%).

3-The urogenital form causes **perminant ovirian damage** which leads to precipitous reduction in eggs production of laying flocks..

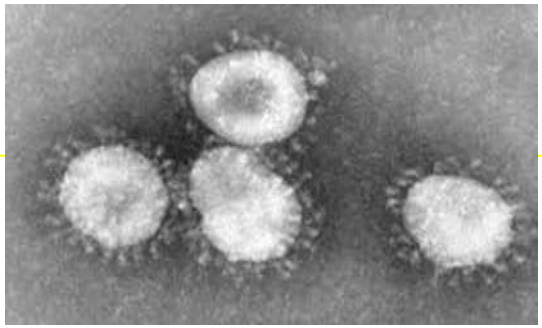

A
B

Incidence & Distribution

IB is distributed worldwide, in US **several serotypes** in addition to the originally identified, **Massachusetts** (Mass) type that being identified in the 1950s, **Mass type** strain have been isolated in Europe &Asia since the 1940s.
Dozens serotypes have been isolates in Africa, Asia, India, Australia, Europe and South America.
-**It is generally accepted that chickens** are **the most important natural host** of IBV, chickens of all ages can be affected. IBV has also been isolated from other species such as pheasants, quail and partridge.
Outbreaks of IB frequently have occurred even in vaccinated flocks.
The virus strains isolated from those outbreaks are often, but not always, found to be serotype distinct from vaccine type.

The disease were reported to occur in Iraq at 1987 and later confirmed by serological and molecular in flocks of Duhok Governorate.

The RT-PCR was used for the 1st time in detection of IBV in Duhok, and was found very efficient in detection of infected chickens. ELISA test was used and found very useful tool in diagnosis of infected chickens.



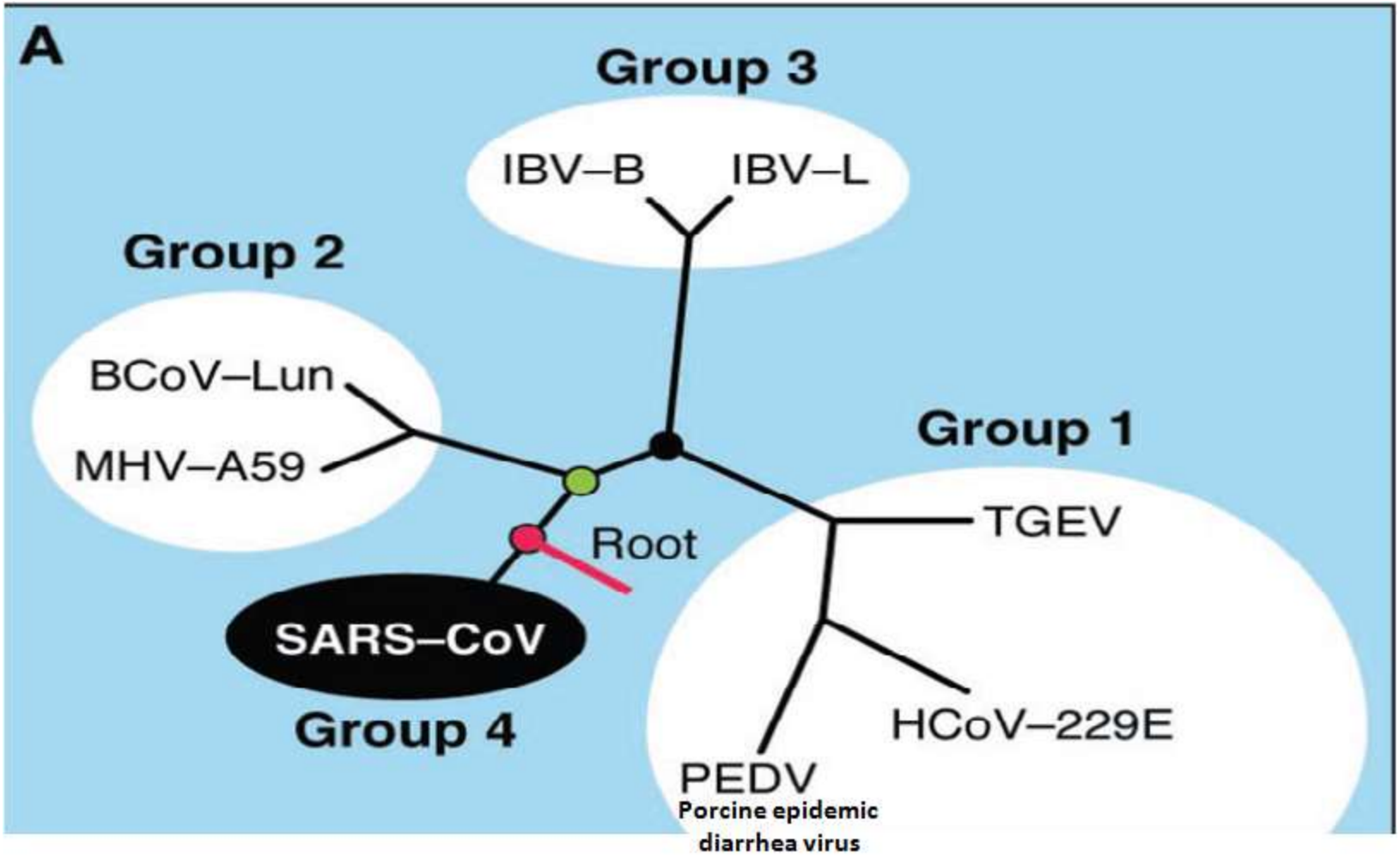
Etiology

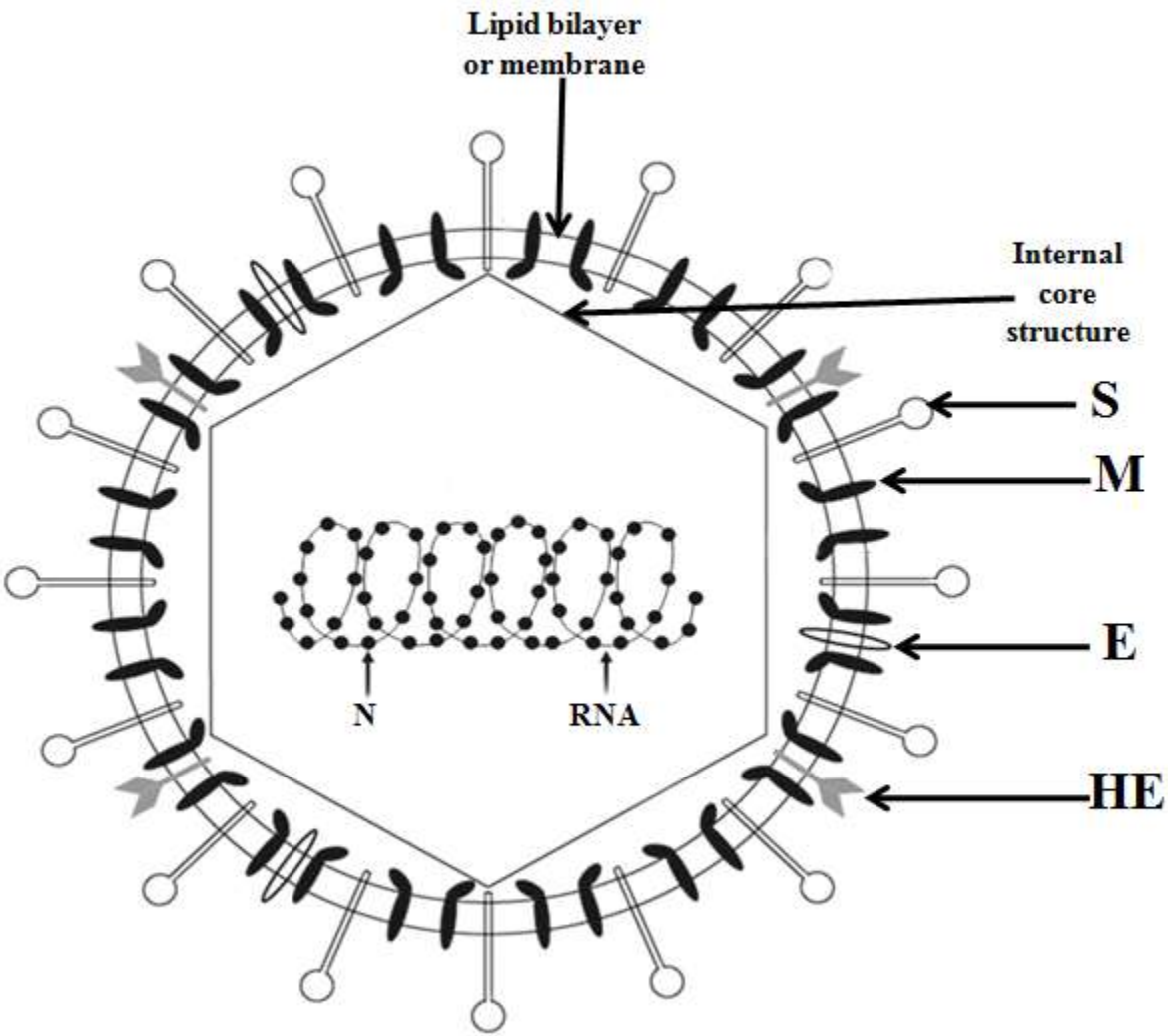
IBV, enveloped virus with spikes, pleomorphic, ss positive sense RNA genome. The virus is a member of the genus Coronavirus, family Coronaviridae, order Nidovirales.

The virus particles has characteristic club-shaped projection (spikes) uniformly distribution on its surface. These projection structure provide them with characteristic, crown-like appearance observed by **EM** which inspired the name of the coronavirus family.

Many serotypes and strains with great antigenic variation have been identified from all over the world and more may found in the future, that lead to high morbidity in all ages of chickens and high mortality in chicks less than 3 weeks old.

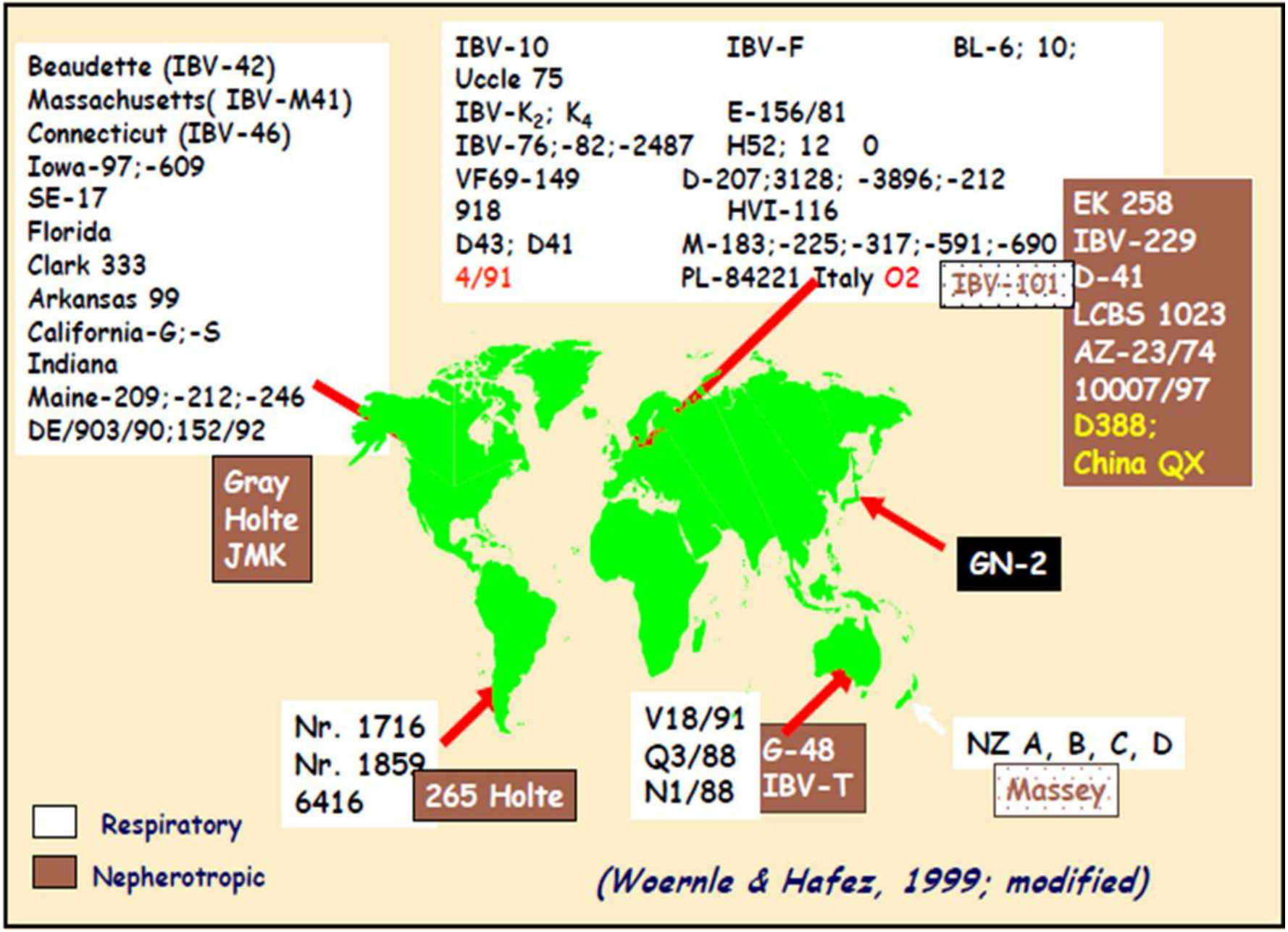
Initially, it was believed that all the isolates belong to a single prototype termed Massachusetts (Mass) serotype mostly isolated from commercial poultry.


A
Group 3
IBV–B
IBV–L
Group 2
BCoV–Lun
MHV–A59
Group 1
TGEV
Root
SARS–CoV
Group 4
HCoV–229E
PEDV
Porcine epidemic diarrhea virus

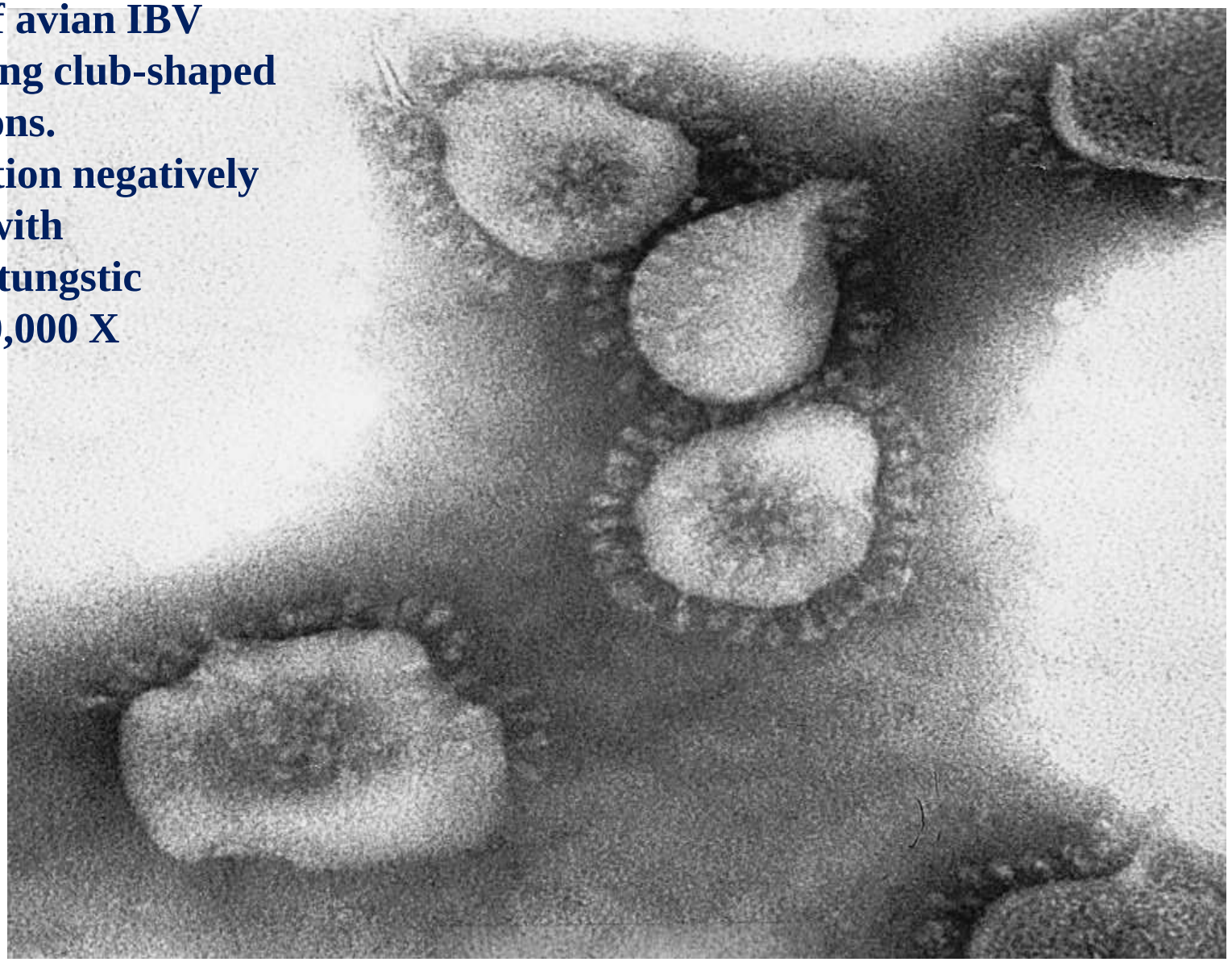


The projection are approximately 20 nm in longand 10 nm wide at their outer edge, with a narrow base. This projections were used for virus attachment to cellular surface proteins, which act as receptors for the virus.

Subsequently, other serotypes were isolated and it is clear now that a considerable number of different serotypes with antigenic and pathogenic differences exist in different parts of the world, also different strains affect different organ systems such as: Respiratory, Renal, Reproductive. Some important field strains are, Massachusetts:
(M41,H 120), Arkansas 99, D 1466, D3128, Delaware, Florida, California, Holte, (4/91also named 793/B and CR88), D388, B1648/D8880, Gray, T-strain, etc, And in more recent times (QX-like IBV), which was associated predominantly with various forms of renal pathology (nephropathogenic strains).


Beaudette (IBV-42)
Massachusetts(IBV-M41)
Connecticut (IBV-46)
Iowa-97;-609
SE-17
Florida
Clark 333
Arkansas 99
California-G;-S
Indiana
Maine-209;-212;-246
DE/903/90;152/92
IBV-10
Uccle 75
IBV-K2; K4
IBV-76;-82;-2487
VF69-149
918
D43; D41
4/91
IBV-F
E-156/81
H52; 12 0
D-207;3128; -3896;-212
HVI-116
M-183;-225;-317;-591;-690
PL-84221 Italy 02
BL-6; 10;
IBV-101
EK 258
IBV-229
D-41
LCBS 1023
AZ-23/74
10007/97
D388;
China QX
Gray
Holte
JMK
GN-2
Nr. 1716
Nr. 1859
6416
265 Holte
V18/91
Q3/88
N1/88
G-48
IBV-T
NZ A, B, C, D
Massey
Respiratory
Nepherotropic
(Woernle & Hafez, 1999; modified)

Virion of avian IBV illustrating club-shaped projections. Preparation negatively stained with phosphotungstic acid. 300,000 X



Variant Strains:

The continuous emergence of new IBV serotypes has complicated the design of appropriate control programs due to the antigenic variation and the **low degree of cross protection** observed among IBV serotypes and has pointed out the necessity for accurate techniques to diagnose and classify this viral agent.

Nucleotide sequencing and subsequent genetic analysis of the **S1** protein gene sequences provides a fast and accurate method to classify and predict IBV serotype, but also a powerful instrument to monitor phylogenetic and epidemiological evolution of IBV subtypes.

Mutation and recombination processes have been demonstrated to be involved in the genetic variation, and therefore in the evolution of IBV, leading to the emergence of new variant strains and giving rise to virus population diversity. The ability of the virus to undergo continuous genomic shift and drift has lead to emergence of several new serotypes especially in the areas of intensive poultry farming.

Strain Classification

Many method are used to differentiated of IBV isolates. More recently **genotype classification** based on features of the **S protein**.
Traditionally , IBV serotype have been define by **VN and HI** test, some laboratory have used monoclonal antibodies that are specific to a given serotype, the antibodies corresponding to epitopes formed by the S1 protein.
These monoclonal antibodies can be used in ELISA which are more econimical than VN assay . However serotype specific monoclonal antibodies are only available for a small number of serotype.
Routinely and more recently, now laboratories are using **PCR** to produced DNA copies of IBV genes.
Sequence analysis of fields strains suggest that the evolution of IBV involved recombination during mixed infections.

Transmission

Horizontally by :

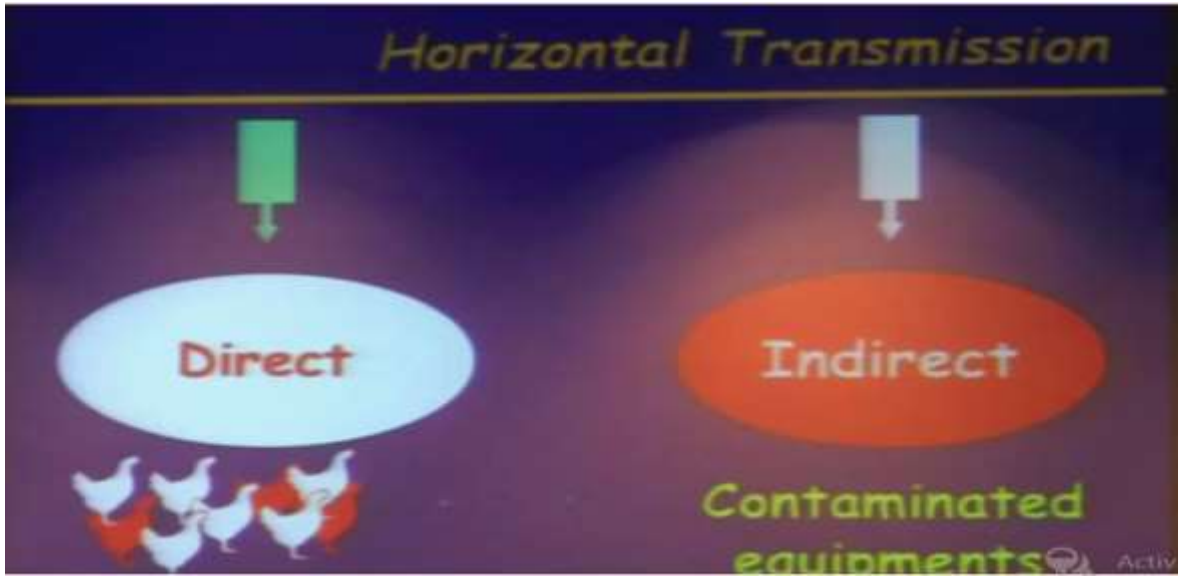
1- Direct contact: Aerosol transmission (sneezing) which was most common.

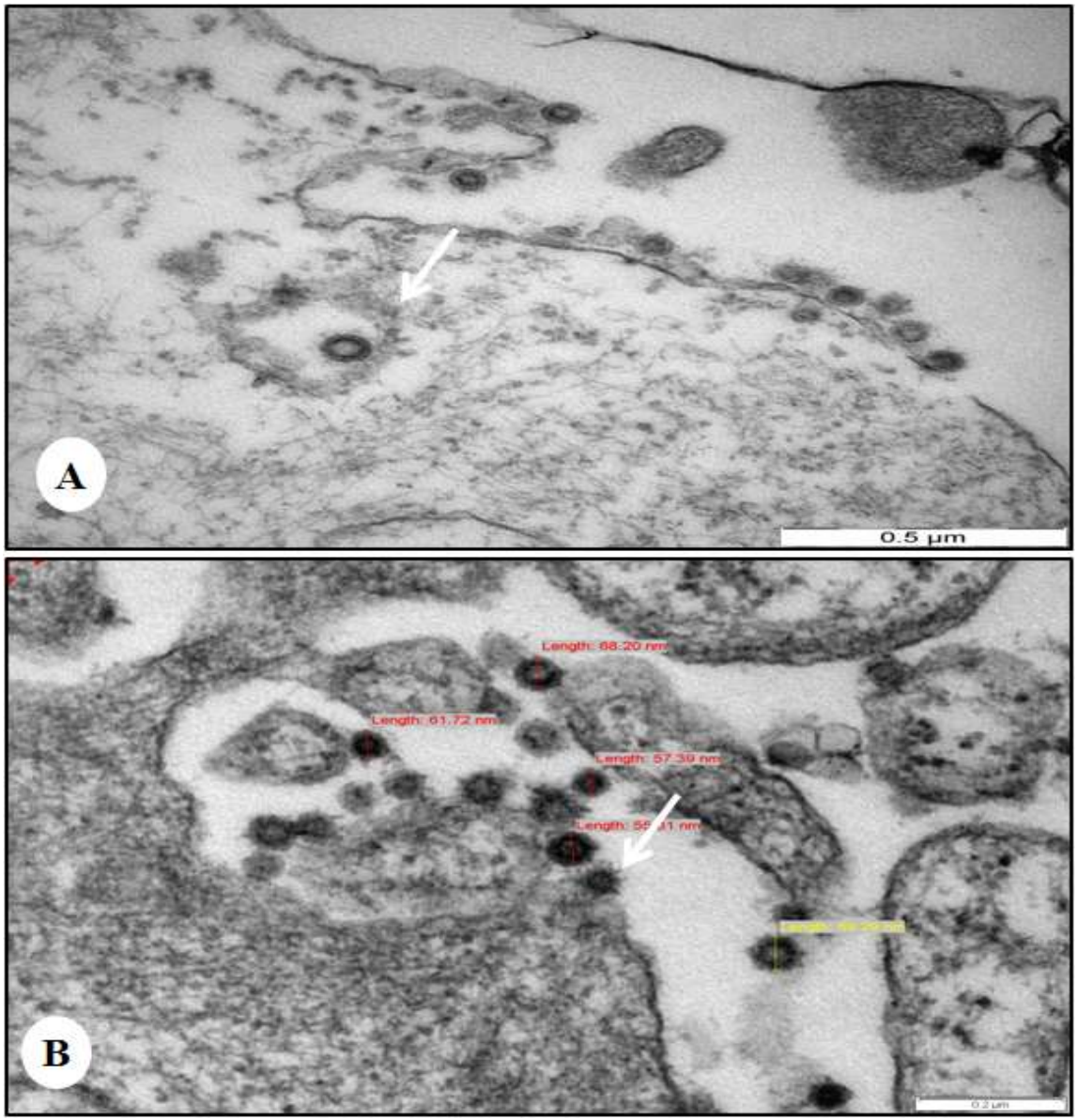
2- Indirect: By Contaminated organic material, drinking water and equipment.

Vertically (from the hen to their progeny through the eggs) has not been shown to be important.

However, surface contamination of eggs with the IBV is a possible by which the virus can spread in hatcheries.

IB is highly contagious so the incubation period is relatively short, 18-36 hours. Disease spreading through entire flock **within one or two days**. (positive-sense genome)

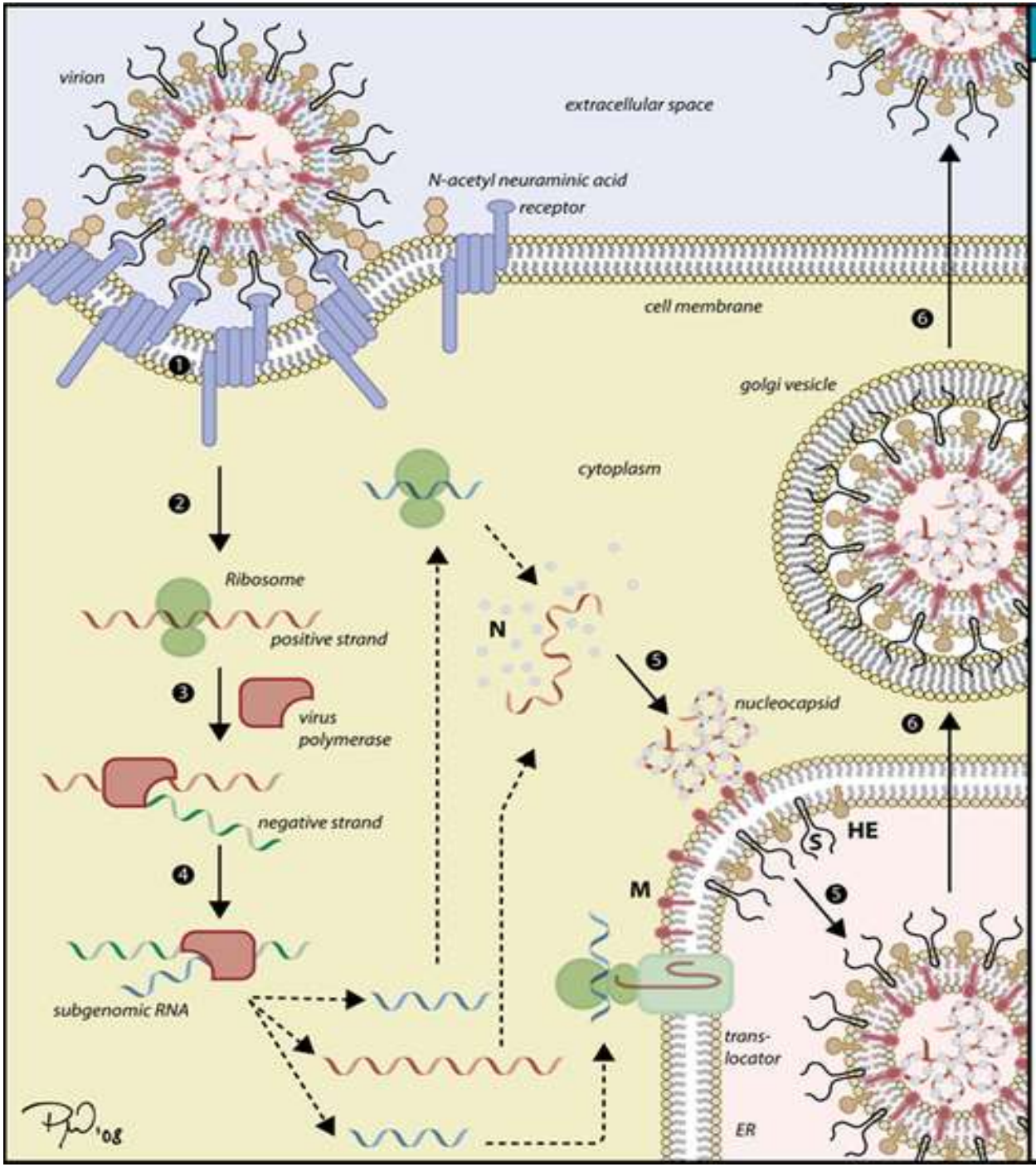



A
0.5 µm
Length: 68.20 nm
Length: 61.72 nm
Length: 57.39 nm
B

Virus replication

The virus replicates in the cytoplasm of infected cells, five subgenomic messenger RNAs being produced by a discontinuous transcription mechanism, three of these mRNA 2, 4 and 5 are responsible of the S, M and N virion proteins respectively ,while the other two m RNAS 1 and 3 encode other proteins respectivly.

New virus starts to appear 3-4 hours after infection with **maximum output per cell being reached within 12 hours** at 37 C . Virion are assembled at internal membrane (e,g, Golgi appartus not at cell surface).



Replication of Coronavirus

1 With their S-protein, coronaviruses bind on cell surface molecules such as the metalloprotease »amino-peptidase N«. Viruses, which accessorily have the HE-protein, can also bind on N-acetyl neuraminic acid that serves as a co-receptor.

2 So far, it is not clear whether the virus get into the host cell by fusion of viral and cell membrane or by receptor mediated endocytosis in that the virus is in-corporated via an endosome, which is subsequently acidified by proton pumps. In that case, the virus have to escape destruction and transport to the lysosome.

3 Since coronaviruses have a single positive stranded RNA genome, they can directly produce their proteins and new genomes in the cytoplasm. At first, the virus synthesize its RNA polymerase that only recognizes and produces viral RNAs. This enzyme synthesize the minus strand using the positive strand as template.

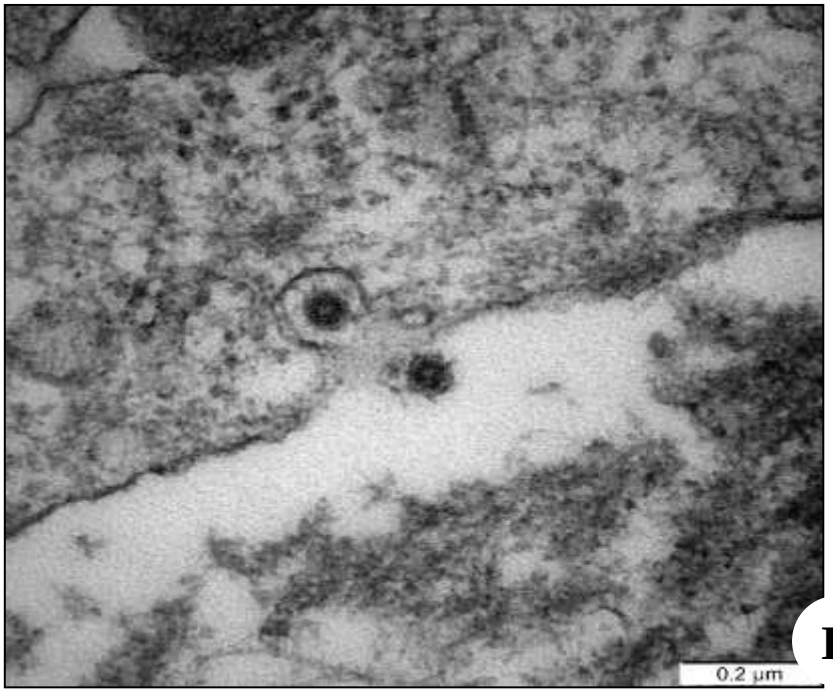
4 Subsequently, this negative strand serves as template to transcribe smaller subgenomic positive RNAs which are used to synthezise all other proteins. Furthermore, this negative strand serves for replication of new positive stranded RNA genomes.

5 The protein N binds genomic RNA and the protein M is integrated into the membrane of the endoplasmatic reticulum (ER) like the envelope proteins S and HE. After binding, assembled nucleocapsids with helical twisted RNA budd into the ER lumen and are encased with its membrane.

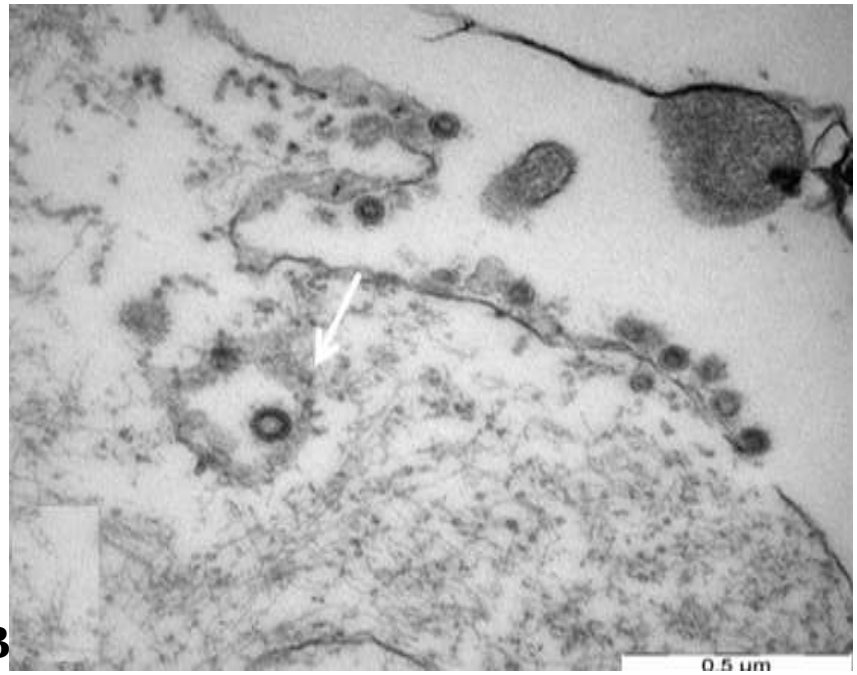
6 These viral progeny are finally transported by golgi vesicles to the cell membrane and are exocytosed into the extracellular space.

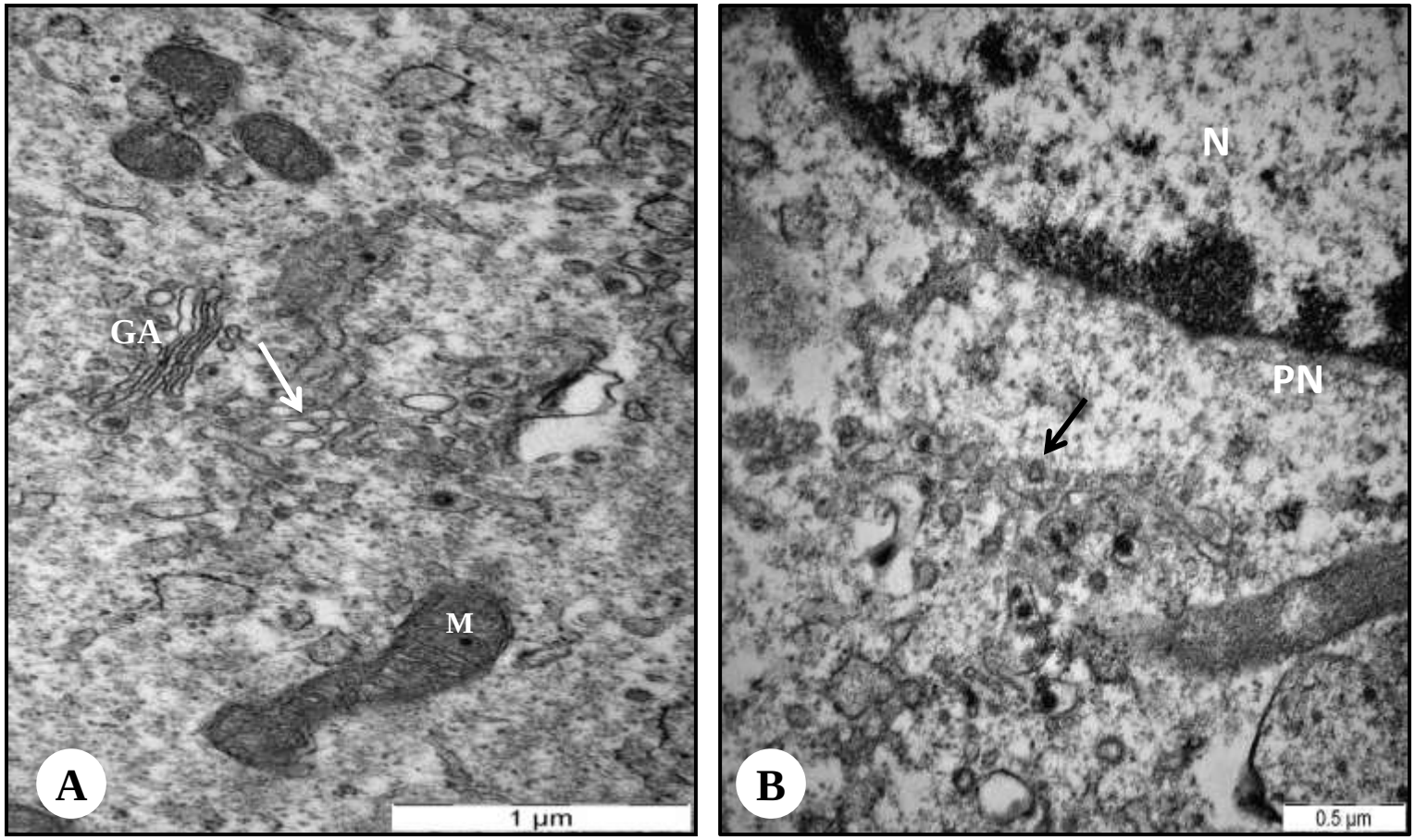
Not drawn to scale! Not all cellular compartments and enzymes are shown. Colors: positive strand RNA (red), negative strand RNA (green), subgenomic RNAs (blue).

Based on: Lai MM, Cavanagh D (1997). The molecular biology of coronavirus. Adv. Virus Res (48) 1-100.



B




GA
M
A
1 µm
N
PN
B
0.5 µm

Economic Important

In broilers, producer losses from IB occur due to poor growth and feed conversion, secondary bacterial infections that require antibiotic treatment and increased condemnations at slaughter.

Layers and Breeders

When birds are infected in the first few days of life with a very virulent IB virus:

1- Permanent damage in the oviduct may occur.

2-These birds may mature like normal hens but produce no eggs, so-called false layers.

3-Drops in eggs production.

4-Poor quality eggs.

5-Production often does not return to pre-infection levels.

Resistance to chemical and physical agent

More strains of IBV are inactivated after 15 min at 56°C and after 90 min at 45°C, long –term storage of IBV at -20 C should be avoided.

Able to withstand pH ranges of pH 2 - 12 depending on **the strain**, **temperature** and **time of exposure**.

The virus was sensitive to most common disinfectants.

Infectious allantoic fluid lyophilized sealed under vacunm and stored in refrigetor has remained for at least 30 years .

Attenuwated vaccines are lyophilized in the presence of sucrose or lactose to presence potency and extend shelf life.

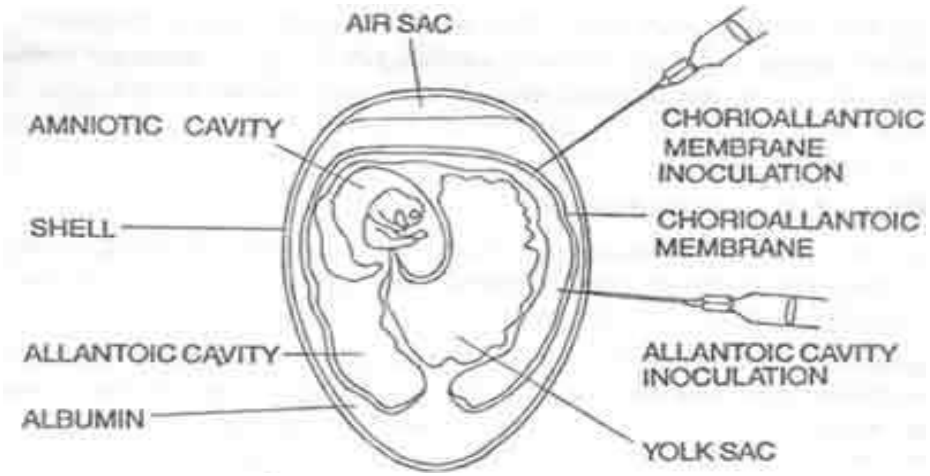
Laboratory host system

In vitro cultivation of avian IBV

A- Eggs inoculation

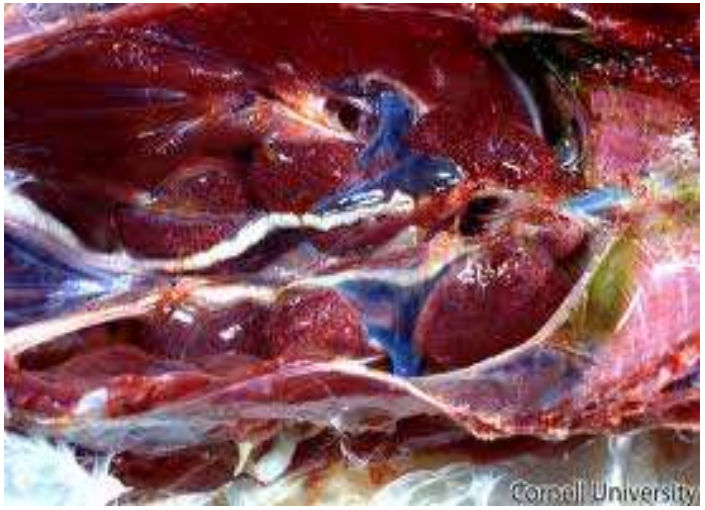
Most isolates of IBV replicate well in the developing chicken embryo following inoculation of the allantoic cavity; The allantoic cavity of a 10 to 11 day-old chicken embryo is a route of choice for inoculation of the virus because of the simplicity of the operation and its sensitivity.

Titaration of IBV in embryonating eggs gives higher titers 10- to 100 fold than in tissue cultures.



The lesions caused by IBV on chicken embryos are **curling of the embryo** with a wry neck, deformed feet compressed over the head and retardation of embryo growth to half the normal size. The thickened amnion membrane which is dry and fibrotic, adheres closely to the embryos and thus restricts its movement.

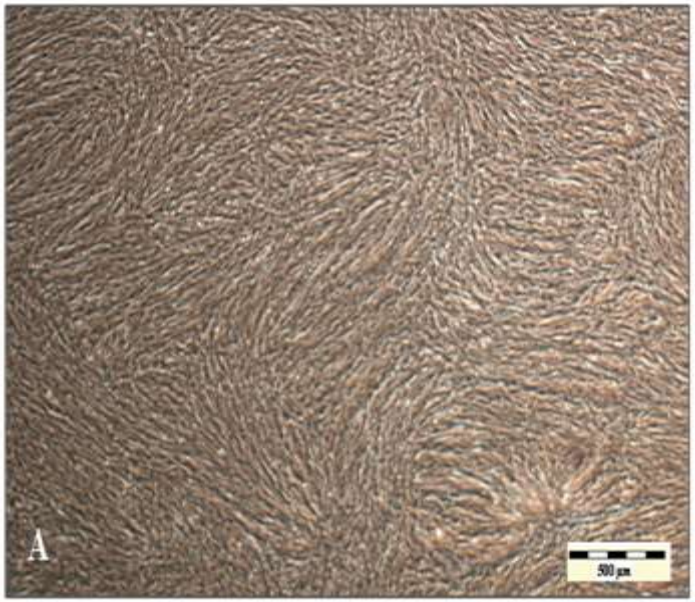
The stunted embryos eventually give rise to the retention of the urates in mesonephros.

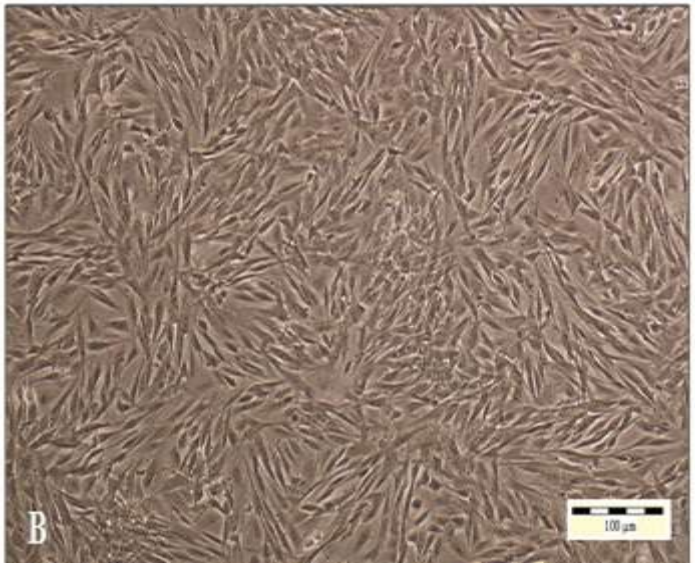


In vitro cultivation of avian IBV

B- Cultivation of IBV in cell culture

IBV requires initial isolation and adaptation to embryonated chicken eggs before cultivation can be done in cell culture.
Avian cell system are the only cell line in which IBV replicated and produced CPE(cytopathic effects).
The Beaudette-M41 and Lowa 79 strains of IBV have been propagated successfully in the **African green monkey (vero cell line),** which has been used for many fundamental studies of IBV.
Also **chick embryo kidney (CEK) cells** have been used most successfully. Staining culture showed CPE characterized **by plaques formation** that could be seen after the first passage.
Adaptation of some strains to CEK is facilitated **by prior embryo passage.**





(A) Uninfected control CMS cells monolayer. (B) Cytopathic effect of IBV isolate of the 3rd passage at day 5 pi.

Pathogenesis

It is generally considered that the **chicken** is the only bird that is naturally infected by IBV and in which the virus causes disease.

Infectious bronchitis virus can replicate in tissues of **the respiratory tract**, **intestinal tract**, **kidneys**, and the **oviduct.**

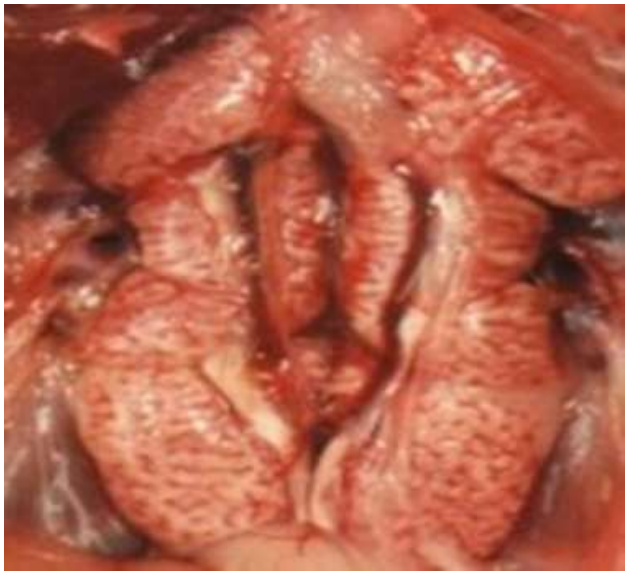
The virus has also been shown to replicate in the **Harderian gland** after eye drop inoculation.

IBV initially infects and replicates in the upper respiratory tract causing the loss of protective cells lining the sinuses and trachea.

Within three days of the infection, virus titres are maximal in the **nose and trachea** and remain for other two to five days

After a brief viraemia, the virus can be detected in other **non-respiratory organs** such as kidneys, reproductive tract, and caecal tonsils. (in kidneys, causing minor and major nephritis),

Renal damage associated with different IB strains is an increasingly important feature of IB infections, especially in broilers



Age of Host Commonly Affected

All ages are susceptible, but the disease is most severe in baby chicks, causing some mortality . As age increases, chickens become mo re resistant to the nephritogenic effects, oviduct lesions, and mortality due to infection.

Incubation Period

The incubation period of IB is 18—36 hours, depending on dose and route of inoculation. Chickens exposed to an aerosol of undiluted infective egg fluid regularly have tracheal rales within 24 hours. Naturally occurring spread requires about 36 hours or more .

Clinical signs:

A-Respiratory disease

The virus is highly infectious, presumed to spread by aerosol. Several serotypes can co-circulate in a region. Young birds are depressed and huddle under the heat source. ruffled feathers, respiratory distress including such as gasping, coughing, tracheal rales, nasal discharge.

As serotypes cross-protect poorly, chickens can be productively infected several times. The first recognized and most conspicuous signs are:

1- The respiratory signs, hence the name Infectious Bronchitis.

Respiratory disease is the most frequently observed syndrome caused by IBV .

In broiler chicks of between 2-6 weeks of age, the main clinical signs seen are

2- Difficulty in breathing (gasping)

3- Tracheal rales

4- Coughing and sneezing with or without nasal discharge

.5- A generalised weakness is observed, accompanied by depression. Feed consumption and body weight are markedly reduced

6-. Clinical signs in uncomplicated infections can be of short duration, commonly lasting less than seven days.

B-Reproductive disorders

Infectious bronchitis virus infection at a young age and after maturity can both lead to reproductive problems in hens.

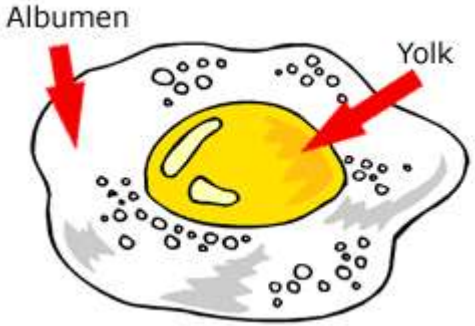
1-A decline in egg production usually follows within 7 to-12 days. The severity of the decline in egg production varies according to the stage of lay at infection and the strain of virus involved. Typically, these declines are between 3% and 10%, but reductions of up to 50% have also been observed. In some flocks, a decline in egg production will be the only feature.

2- However, in many flocks the decline in egg production is also associated with eggs of smaller size

3- Inferior shell and internal egg quality, seen as soft-, pale-shelled and misshapen eggs

4- Eggs with thin albumen. Flocks usually return to near normal production within **one week**, but occasionally six to eight weeks might elapse before a return to normal production.

In many instances, production levels remain subnormal, usually 6% to 12% below pre-infection levels. On post-mortem examination, the oviduct length may be reduced and ovarian regression is noticed in some birds. If IBV infection occurs when chicks are less than two weeks of age, permanent damage of the oviduct may result, leading to poor laying capacity.



C-Infectious bronchitis virus nephritis

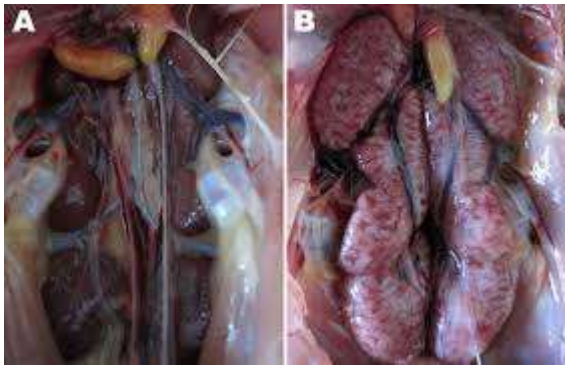
The nephritic form of IB:

1- Is characterized by mild and transient respiratory signs followed by

2-depression, ruffled feathers, reluctance to move

3-excessive water intake, rapid weight loss and diarrhoea. Characteristically, wet litter is present.

4-Death occurs four to five days after infection and ceases by day 12 after infection. On necropsy, carcasses are dehydrated and dark in colour, kidneys are enlarged and may be pale or marbled, and deposits of urates may be present in the ureters of some chicks.



IB - Clinical signs

False Layer

Respiratory
Kidney damage

Production Respiratory

Early infection

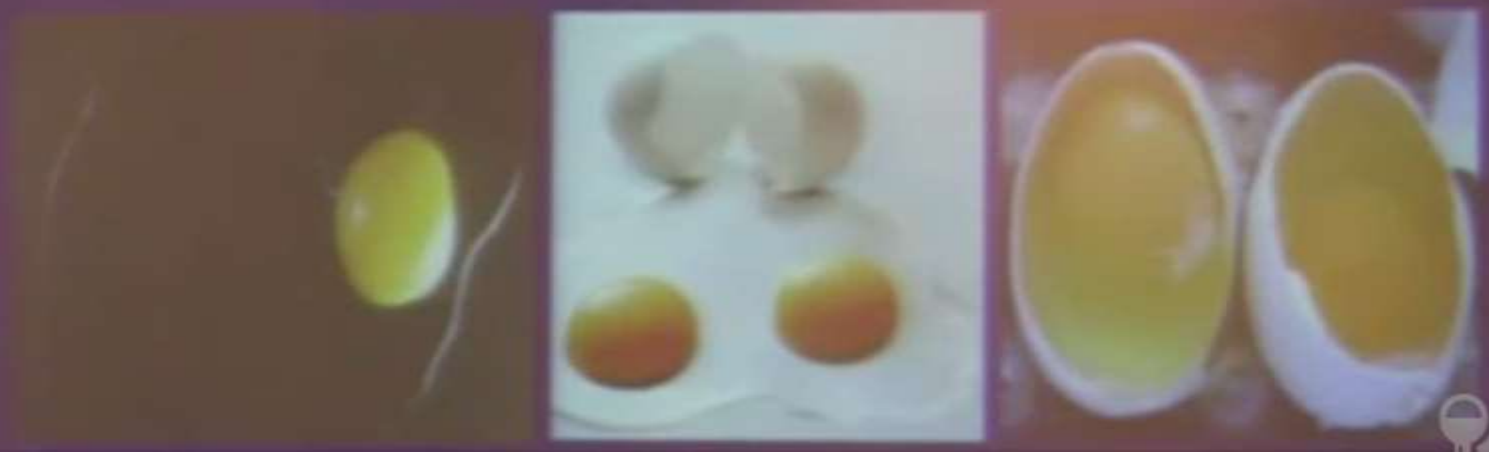
Rearing

Production
> 18 weeks

IB - Clinical signs - Layer & Breeder

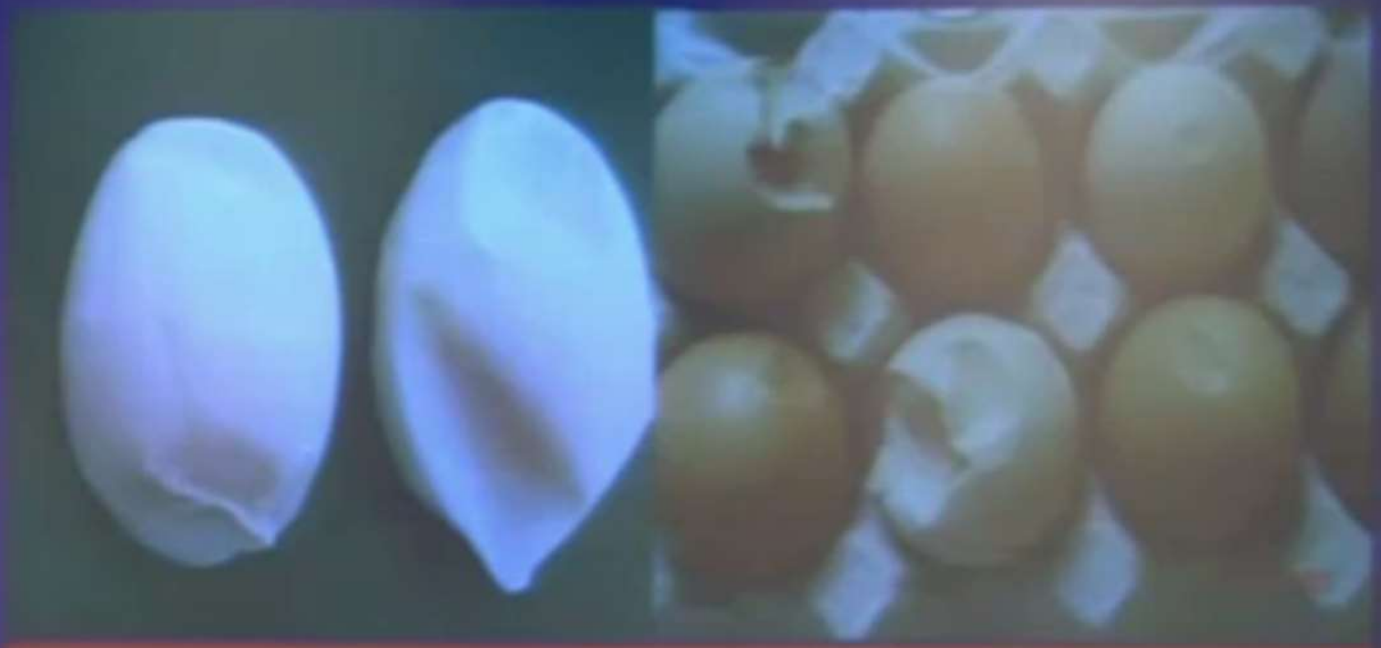
Reduced internal egg quality (thinning of the thick albumin)

"watery whites"



IB - Clinical signs - Layer & Breeder

Drop in egg shell quality for
6 - 8 weeks /or months

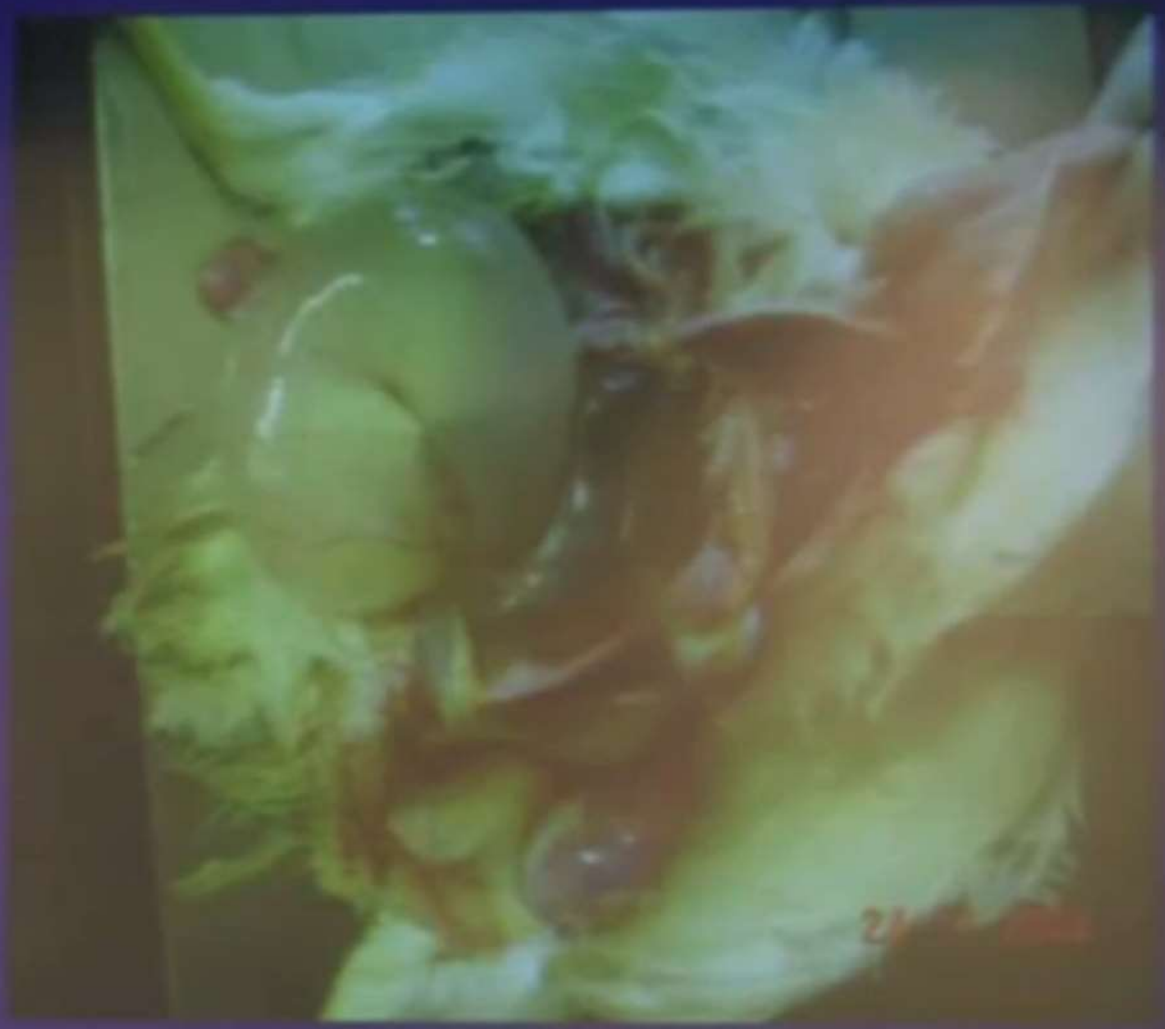


Thin shell

IB - Clinical signs - Layer & Breeder

False Layer ?

IB - Clinical signs - Layer & Breeder



Morbidity and mortality

All birds in the flock become infected, but mortality is variable depending on **virulence of the infecting serotype**; **age**; **status of immunity**, either maternal or active; **and stresses** such as cold or secondary bacterial infections.

In broiler flocks, morbidity is virtually 100%, whereas mortality is usually low.

Early reports describe mortalities of 30% to 50%, and these were almost certainly due to mixed infections with other infectious agents such as E. coli or Mycoplasma. In younger chicks affected with IB nephritis, mortalities of up to 35% are common

Gross Lesions:

Post mortality, lesions are found in the **respiratory tract** and **urogenital tract.**

Renal damage associated with different IB strains is an increasingly important feature of IB infections, especially in broiler.

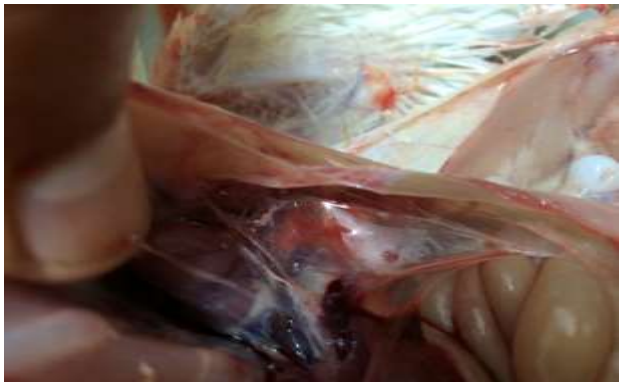
Respiratory

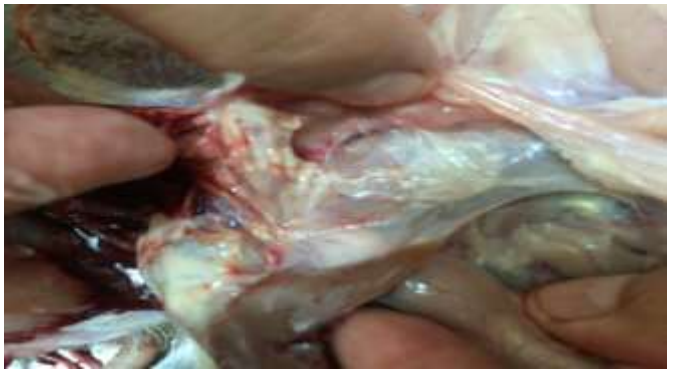
1-Infected chickens have serous, catarrhal, or caseous exudates in the trachea, nasal passages, and sinuses,

2- Air sacs may appear cloudy or contain a yellow caseous exudate.

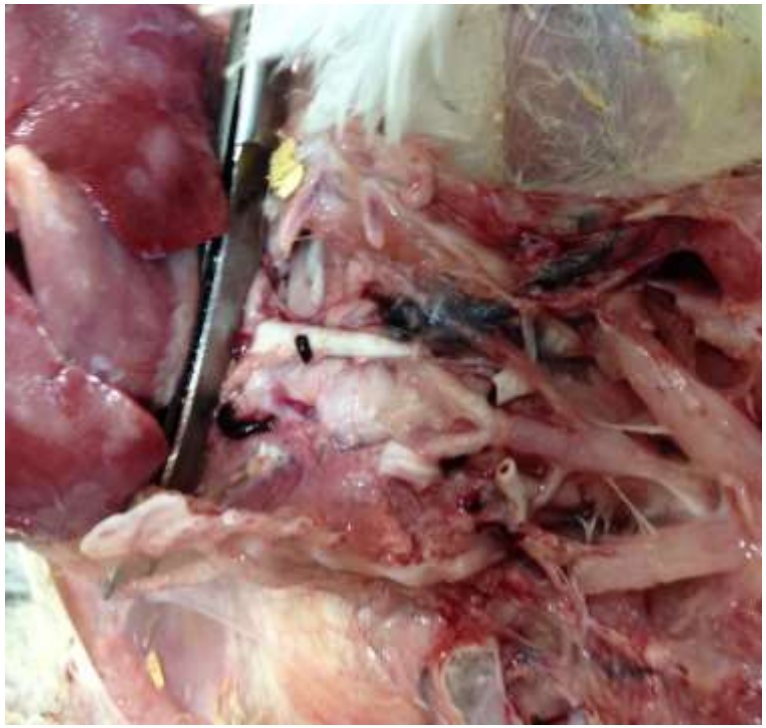
3-A caseous plug may be found in the lower trachea or bronchi of chicks that die.

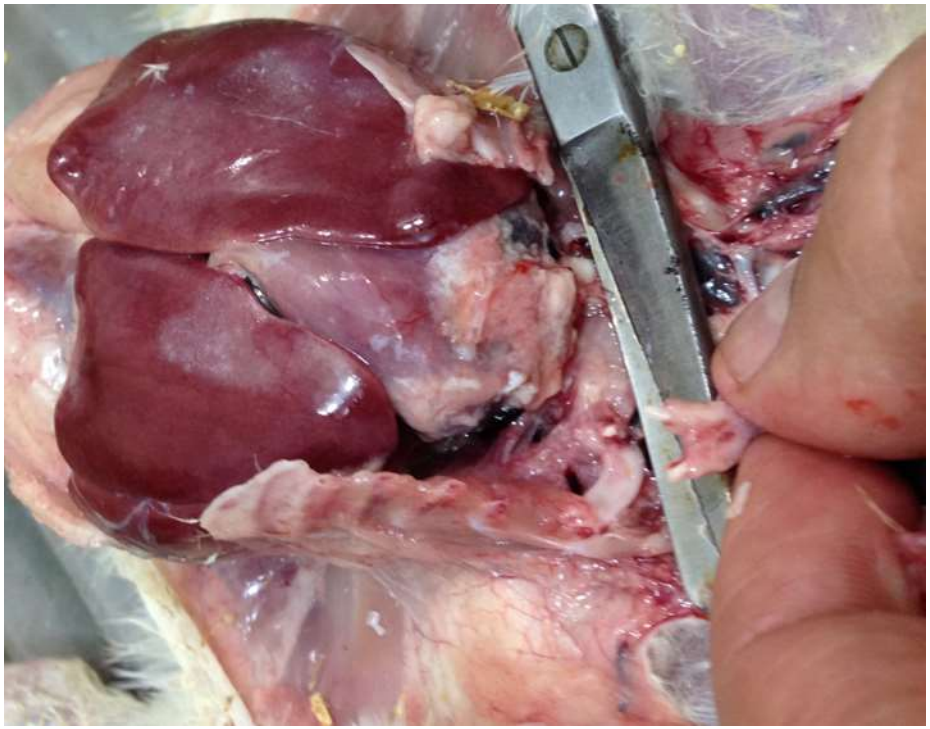
4- Small areas of pneumonia may be observed around the large bronchi.





flocks(the most prominent lesion in respiratory disease infected flocks is severe exudation in trachea, which leads to tubular cast formation in the tracheal bifurcation and extending to the lower bronchi

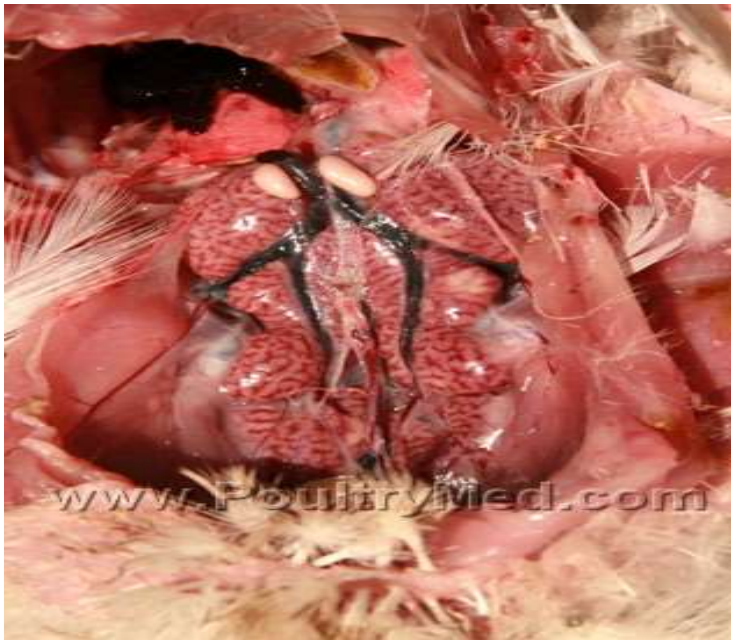


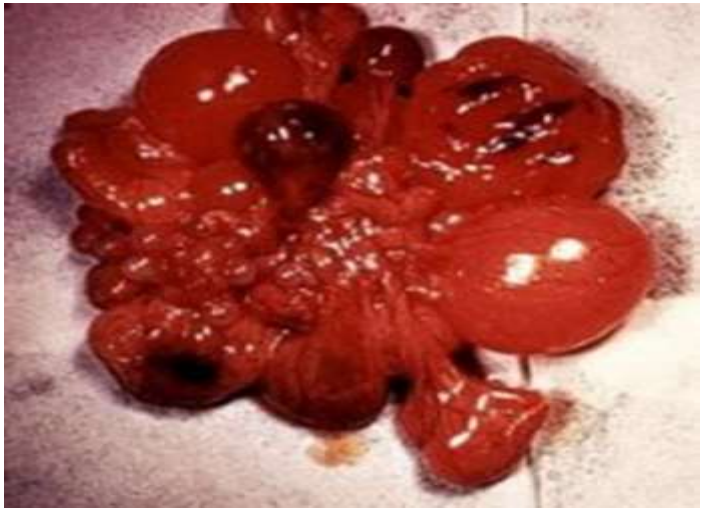


Urogenital

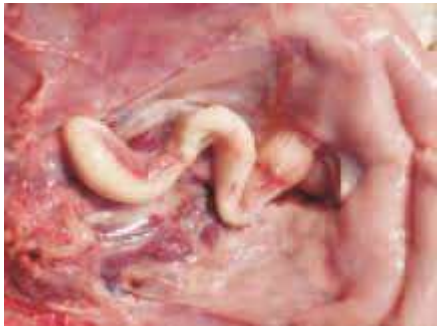
1- Swollen, pale kidneys, with distended tubules and ureters containing urate crystals in nephropathogenic cases.

2- Fluid yolk material may be found in the abdomen of the birds in production (egg peritonitis). Degeneration of the ovary and swollen of the oviducts.

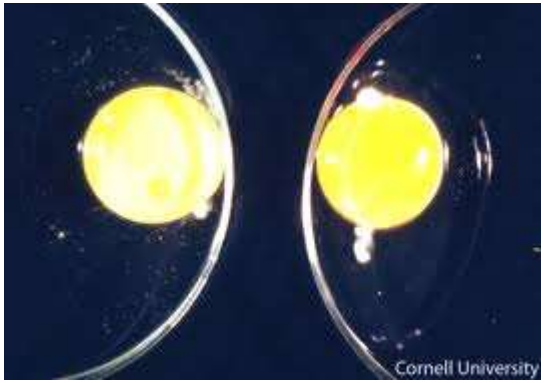




The most noted lesions associated with the infection of pheasants by coronaviruses in the field are visceral urate deposition (“gout”) and urolithiasis with gross swelling of the kidneys, which are pale.



In laying hens the virus causes glandular hypoplasia in the oviduct that leads to reduction in the synthesis of albumin proteins, especially ovomucin, lysozyme and other major proteins which constitute the structural matrix of the thick albumen, and lead to ‘watery-whites’

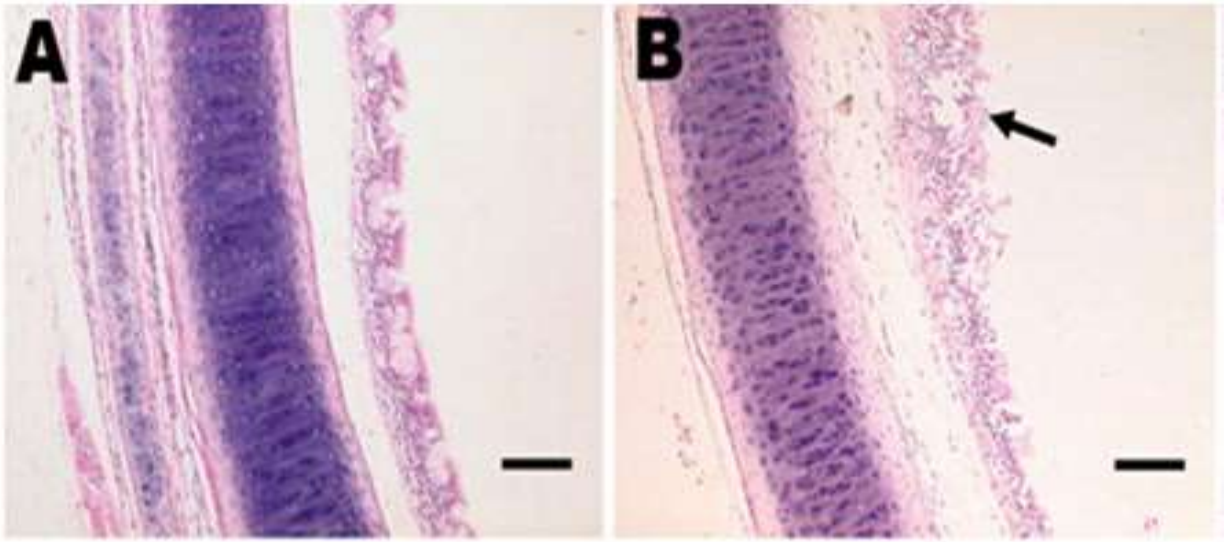


Histopathology

In chickens with **respiratory disease**, the main histological lesions are found in **the trachea.**

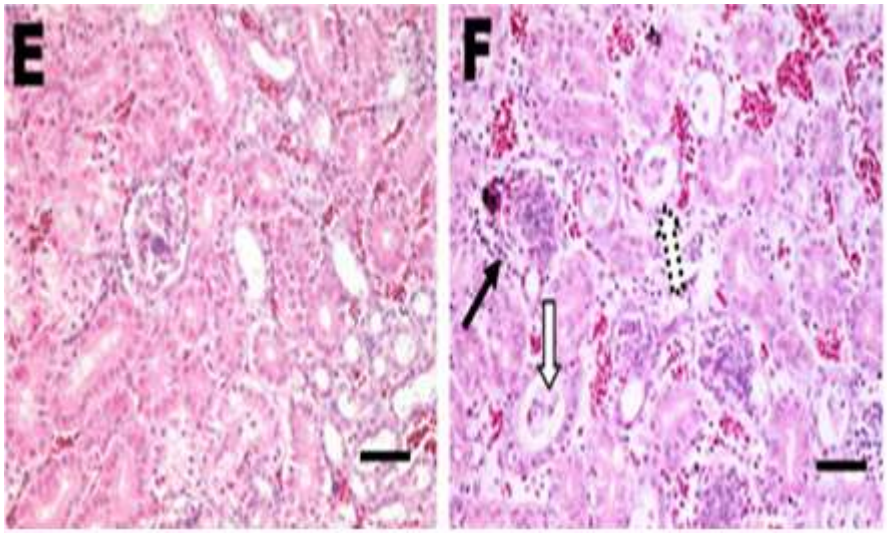
However, these lesions are not pathognomonic for IBV. The virus replicates in ciliated epithelial cells of the trachea, causing loss of cilia, rounding and sloughing of epithelial cells, and minor infiltration of heterophils and lymphocytes within 18 hours of infection.

In the oviduct, the height of the epithelial cells is reduced, and this is accompanied by a reduction or complete loss of cilia. The epithelial cells, especially the goblet cells, become cuboidal.

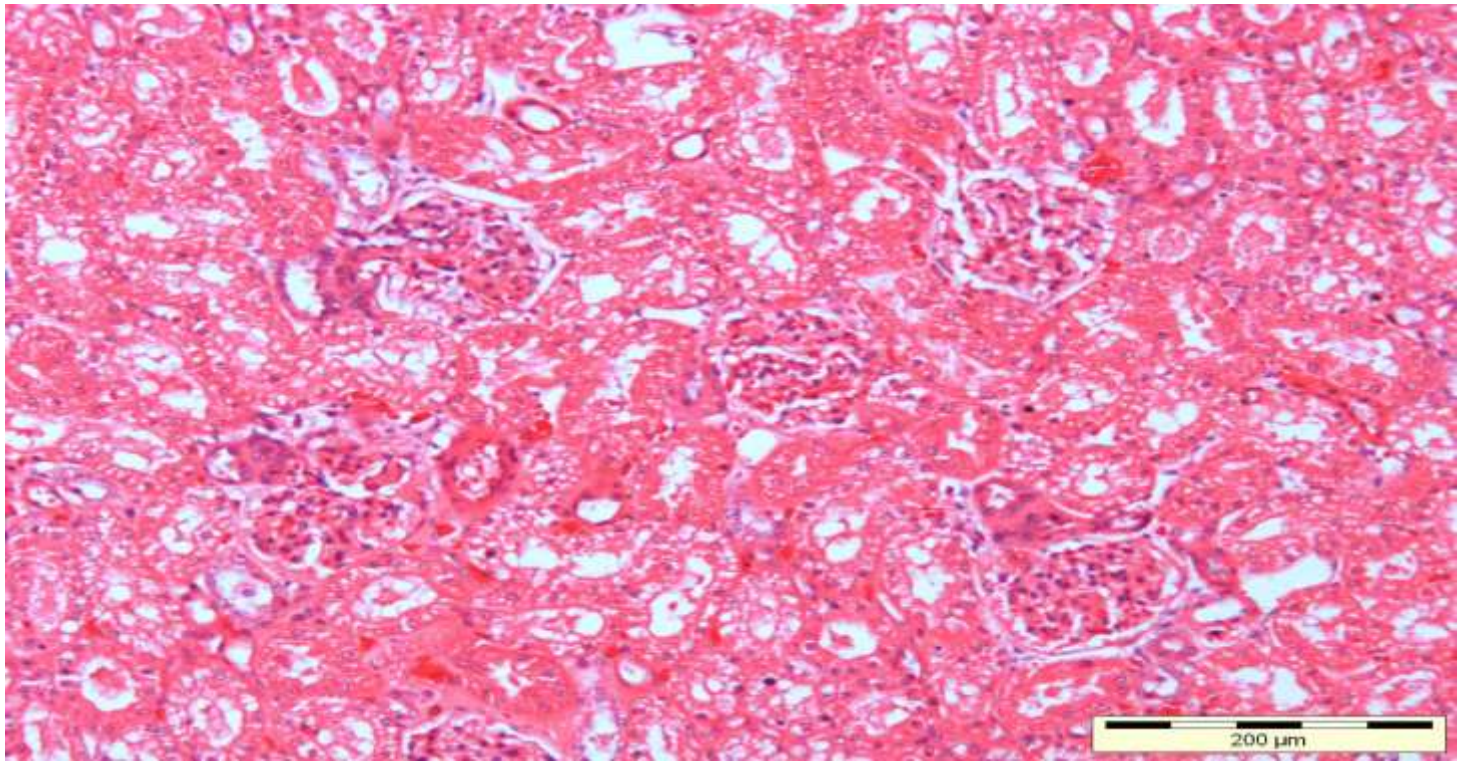


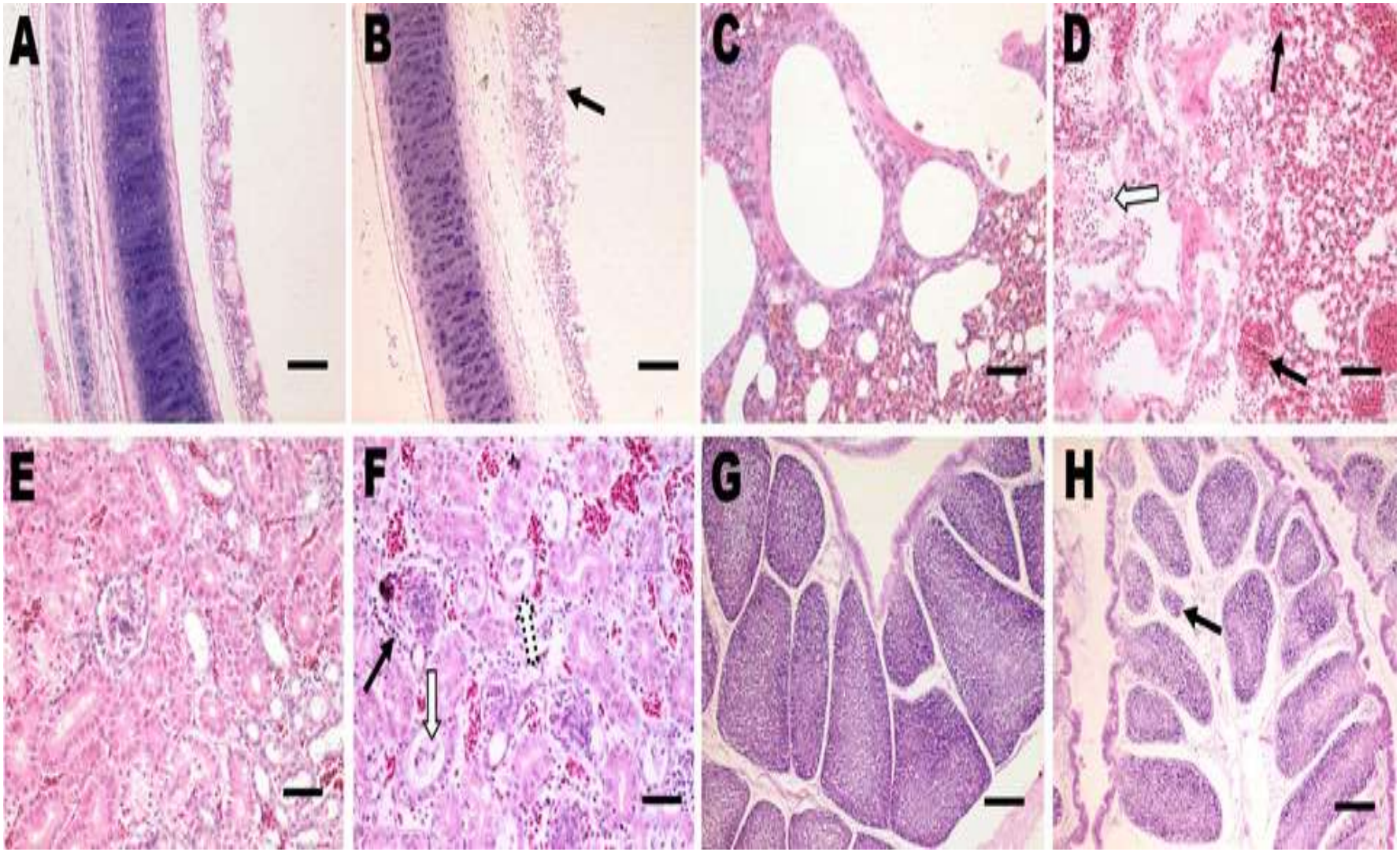
Histopathology

The route of spread to the kidney tissues is unknown. In the kidney, cytopathic changes become apparent, initially in the **tubular epithelium**. As a result, an interstitial inflammatory response is generated and polymorphonuclear leukocytes can be seen, first in the medullary region and then in the cortex. Extensive tubular degradation and necrosis follow and these are prominent at between 5&10 days after infection. Focal areas of uric acid precipitation appear in the kidney and may also accumulate in the ureters .



The main changes in the **kidney** appeared that most of the renal tubular epithe- lium undergo either acute cellular swelling (ballooning), hydrodegeneration due to the accumulation of multiple clear water vacuoles with the cytoplasm leading to the narrowing of the lumen and giving the tubules star like shape appearance. Other tubules showed advance coagulative type necrosis characterized by increased in acidophilia of the cytoplasm with absence of nuclear staining.




A
B
C
D
E
F
G
H

Diagnosis

Diagnosis of infectious bronchitis on the basis of clinical sings alone is very difficult.


?
What is the Problem?

Diagnosis

Problems related to diagnosis

*_ Clinical signs
*_ Gross lesions

not specific

Diagnosis:

Traditionally, virus isolation is the first step in the detection of IBV. **The trachea is a primary target for IBV isolation**, especially within the first week of infection. One week after clinical signs have appeared, the possibilities of isolating IBV are higher in **cloacal swabs or caecal tonsils, kidney or oviduct** than in the trachea.

IBV can be isolated in different biological systems such as **embryonated eggs** and **chicken tracheal organ cultures.**

Virus isolation is a sensitive technique but can be extended, time consuming should be passage through embryonated eggs or tracheal organs several times.

Currently, the most common techniques utilize the RT-PCR and amplify a genomic region conserved across viral strains.

Diagnosis:

Alternatively, a specific IgM ELISA has been used to detect recent IBV infections, because of the limited lifespan of this antibody isotype. All IBV serotypes have both group and type-specific epitopes the latter are present in the S1 protein.

In general the serological tests (ELISA, Haemagglutination inhibition, immunofluorescence and immunodiffusion) bind to group, as well as to type-specific, antigens, so do not differentiate serotypes. Other disadvantage is that the method requires reference strains and sera. That disadvantage can be avoided using monoclonal antibodies, but they are limited and do not covers the full range of IBV serotypes.

The steps below were used to identify the IBV which included:

1- Haemagglutination and Haemagglutination Inhibition Tests

2-Confirmation of Infectious Bronchitis Virus by Antibody- Based Methods

3-Virus Neutralization (VN)

3- Indirect Enzyme-linked immunosorbent assay (ELISA):

4- Agar gel immunodiffusion (AGID)

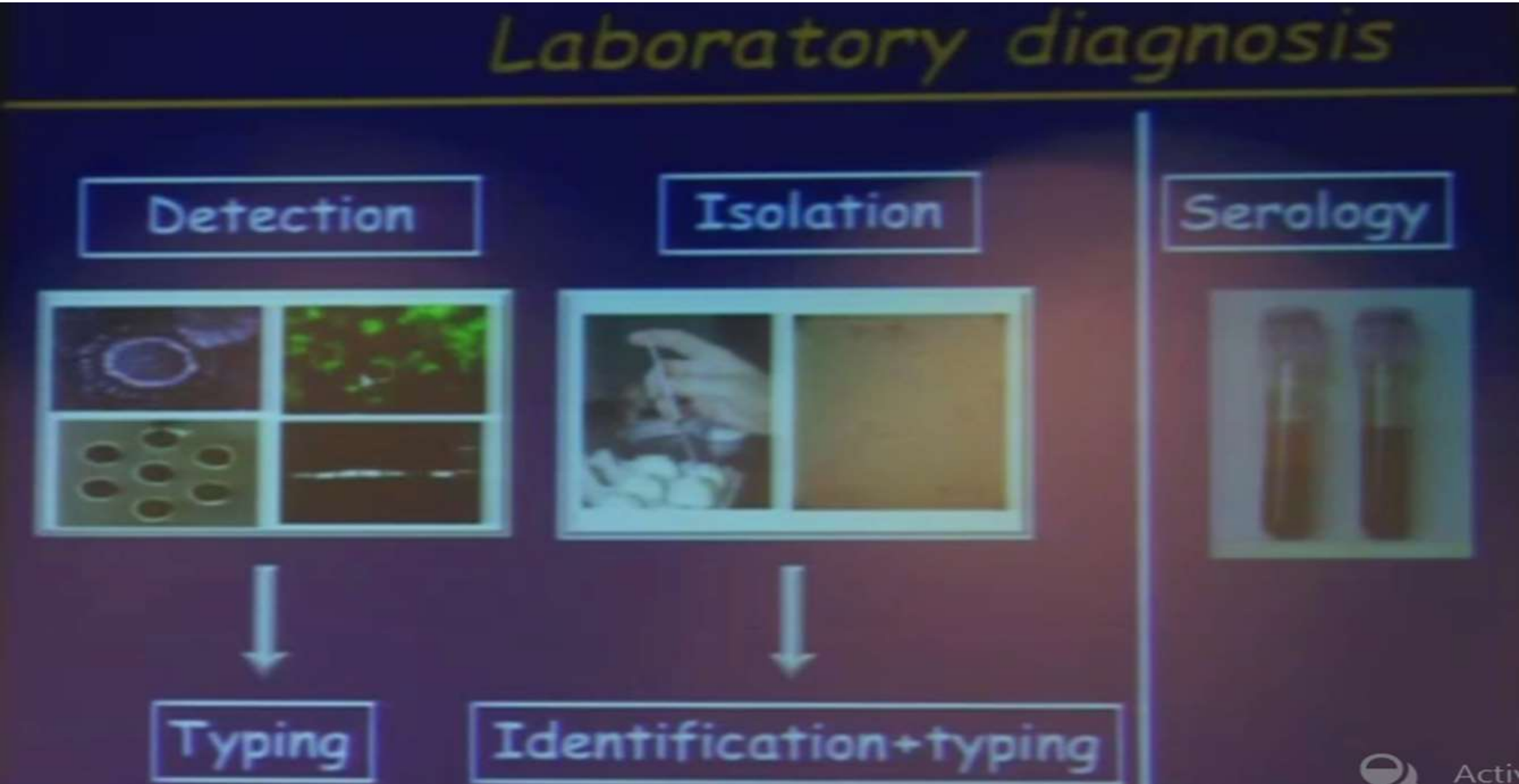
5-Confirmation of Infectious Bronchitis Virus by Nucleic Acid-Based Methods by using RT-PCR method.

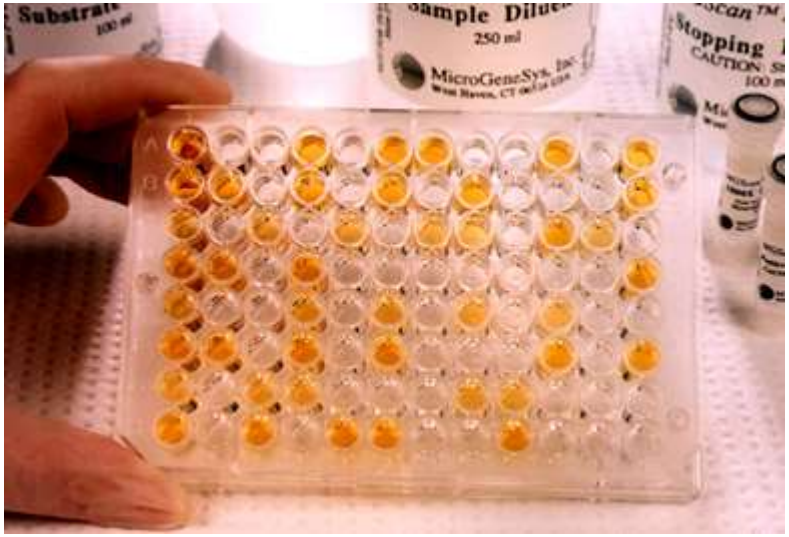
Problems related to isolation including. short duration of shedding. several passages are mostly required and high cost.

Diagnosis

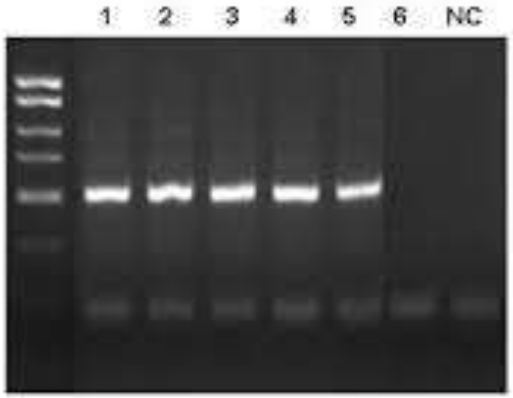
Detection of IBV

using RT-PCR exhibited higher sensitivity than virus isolation and can be used as a rapid diagnostic method in the field, as well as in serotype differentiation and epidemiological studies.

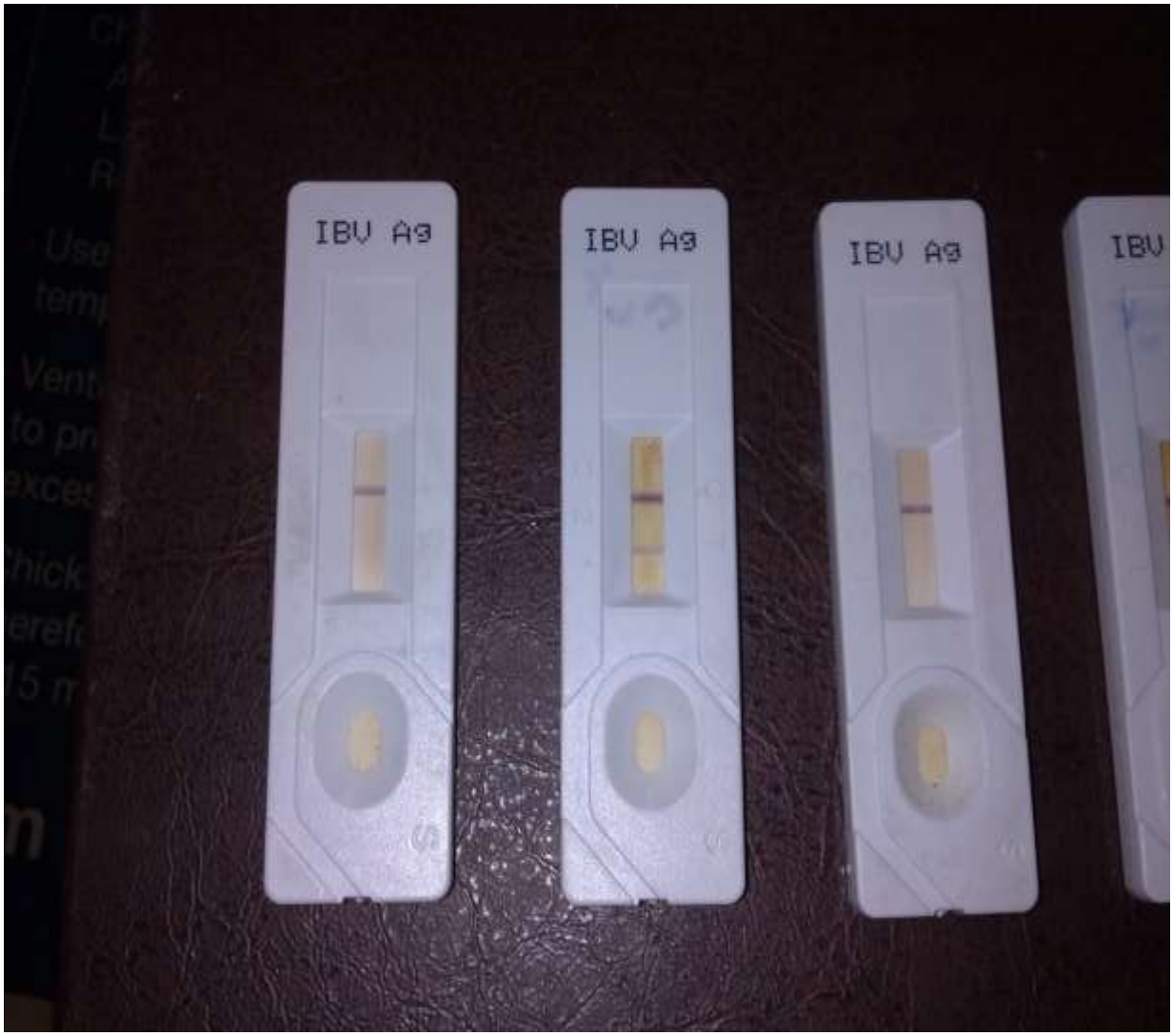



250 ml
MicroGeneSys, Inc.


BioTek



http://www.eppendorf.sh.cn


IBV A3
IBV A3
IBV A3
IBV

Differential Diagnosis

Infectious bronchitis may be similar to other acute respiratory diseases such as:

Newcastle disease (ND), infectious laryngeotracheitis, low pathogenicity avian influenza, infectious coryza and **EDS.**

- **Newcastle disease** caused by **velogenic viscerotropic** or neurotropic strains of paramyxovirus type 1 produces much higher mortality than IB.
- **Lentogenic ND infections with pneumotropic strains and low pathogenicity strains of avian influenza produce mild to moderate respiratory disease with low mortality and. thus, may resemble IB.**
- **ILT** tends to spread more slowly in a flock, but respiratory signs may be more severe than with IB, histological the nucleic inclusion bodies formation with ILT without formation in IB.
- **Infectious Coryza** can be differentiated on the basis of facial swelling that occurs only rarely in IB.
- Production declines and shell quality problems in flocks infected with the **(EDS)** adenovirus are similar to those seen with IB, except that internal egg quality is not affected in the case of EDS.

Immunity:
Active Immunity

Chickens just recovered from the natural disease are resistant to challenge with the same virus (homologous protection), but the extent of protection to challenge with other IBV strains (heterologous protection) varies.

Factors that complicate studies of the mechanism and duration of immunity to IB **are the multiple serotypes** that are recognized the variation in virulence observed among strains and the different manifestations of IBV infection for which protection may be needed.

More than 26 serotypes of IBV have been reported throughout the world, there wide and variable tropism for tissues challenge vaccine strategies, **one vaccine may not cross protect completely**. Vaccination is only partially successful due to continual emergence of antigenic variants and requires the application of multiple vaccines at many sites due to the simultaneous presence of multiple antigenic types

Immunity

Challenge of vaccinated birds with **homologous** virus results in much lower titers of recovered challenge virus, and for a shorter period, than in unvaccinated birds.
When the challenge virus is of a **heterologous** type, the challenge virus may replicate to high titers and cause clinical disease.

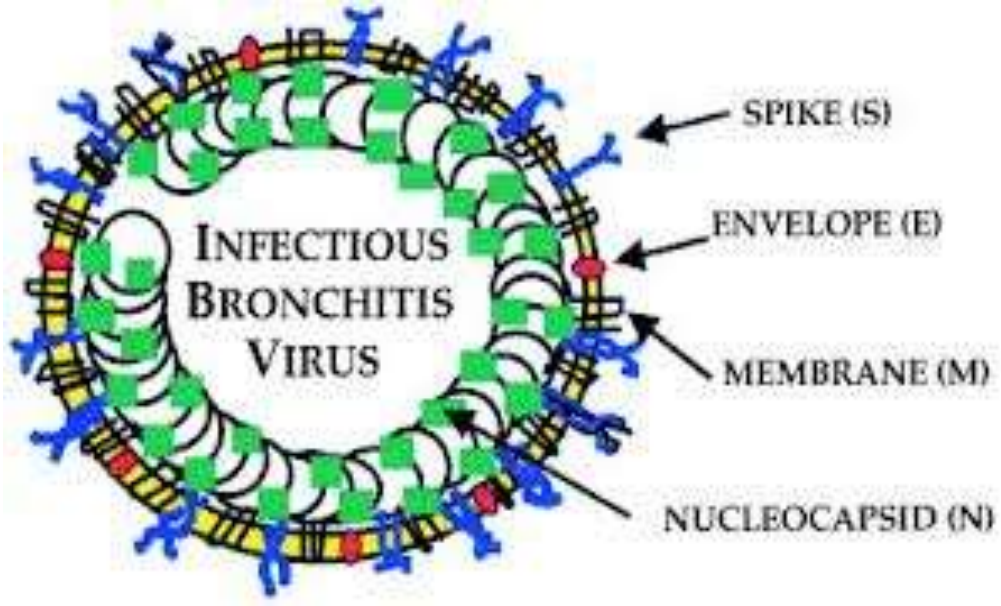
Protection against mortality from nephritis is important as evidence of satisfactory vaccinal immunity in which nephritis is a major clinical problem.

Evidence exists that the S1 glycopolypeptide is primarily responsible for the induction of the VN and HI antibodies and that it plays a major role in the induction of protective immunity, the knowledge of the mechanism of protection against clinical disease is incomplete.

The role of local antibody in preventing reinfection is also unclear. Some studies have reported neutralizing antibody in nasal secretions to play a role in preventing re-infection and that the Harderian gland contributes to local immunity.

Diseases caused by coronaviruses such as IBV are more frequent in winter, with a unique ability to establish persistent infections in a minority of infected animals. **Both live and inactivated IB vaccines are used extensively, the latter requiring priming by the former. Their effectiveness is diminished by poor cross-protection.**

The nature of the protective immune response to IBV is poorly understood. Evidence exists that the S1 glycopolypeptide is sufficient to induction of protective immunity. There is increasing evidence that only a few amino acid differences amongst S proteins are sufficient to have a detrimental impact on cross-protection.



Passive Immunity

Passive Immunity against IBV

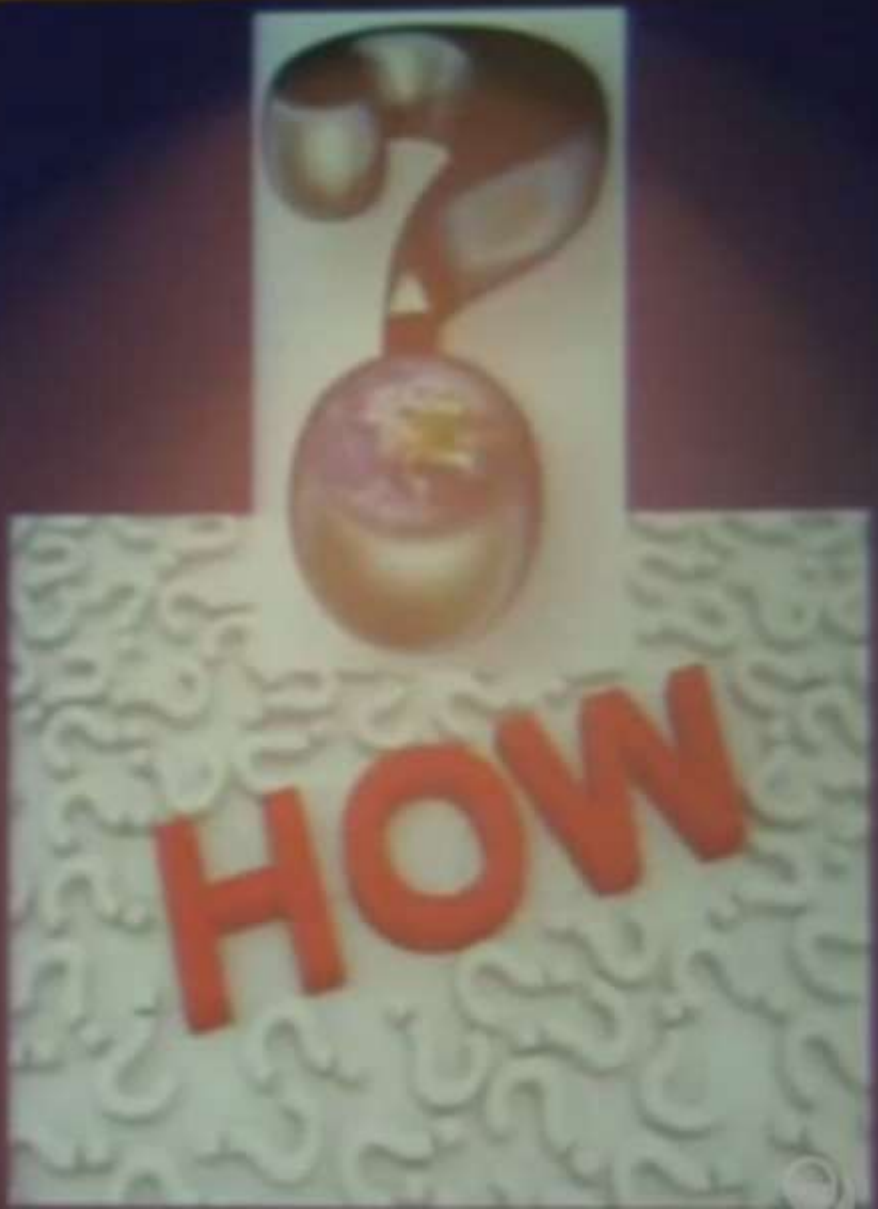
Maternal-derived antibody (MDA) can be transferred via the yolk sac to neonatal chicks. This antibody (largely IgG) can interfere with the immunity generated by live vaccines of the same strain.

MDA produced by vaccination of breeders can provide protection of their chicks against IBV challenge until one week of age, but may not prevent viral infection of the respiratory system.

Chicks hatched with high levels of maternal antibody had excellent protection (>95%) against IBV challenge at day 1 of age, but not at 7 days (<30%).

Prevention and control of IBV

Disease Prevention and Control



Disease Prevention and Control

*- Biosecurity
*- Sanitation (C&D)
*- Monitoring & early detection

Disease Prevention and Control

Vaccination

PREVENTION

The extent to which infection is an economic problem will depend on many factors, including the **strain of virus**, **age of chicken** at infection, **nutrition,** and **the environment** both within the poultry house e.g. ammonia levels, and outside e.g. temperature.

In areas where there are many poultry farms, it is virtually impossible to keep chickens free of IBV.

Biosecurity is likely to be insufficient, as the virus is spread readily. Consequently vaccination is commonly practised. Whilst the humoral response to IB vaccination has been measured for many years, very little is known about the cellular immunity induced by IB vaccines or field strains.

None of the countries which have an Intensive poultry industry are free from IBV. **Although attempts have been made, at the regional level, to keep flocks free from IBV, none have been successfu**l.

Vaccination

The only practical means of controlling IB is vaccination, which is routinely used throughout the intensive poultry industry. The following factors are a feature of IB vaccination:

a) Vaccinal immunity is not long-lasting and re-vaccination is necessary.

b) The selection of an appropriate antigenic type for the region is important, given the existence of wide antigenic variation.

c) Timing and method of vaccine application will vary for different flocks and may require adjustment according to practical experiences.

Type of vaccine used and effectiveness in protecting agains infection and disease

Live vaccine

- **Replicated in the respiratory tract**
- **Stimulate local, humoral and cellular immunity**

Inactivated vaccine

- **Need live vaccine priming**
- **Stimulate uniform and persistent titres**

Live vaccines

Live vaccines are in widespread use. These vaccines represent IBV strains that have been passaged in embryonated chicken eggs to achieve a reduction in virulence for the respiratory tract. Consequently, depending on the level of attenuation, IBV vaccines can be either **mild or virulent**.

Live IBV vaccines are administered by either **coarse spray, aerosol or drinking water,** depending upon the vaccine used. **Vaccination of day-old chicks at the hatchery, with vaccines of low virulence, is practised in most countries since this is a simple way of handling the birds.**

More **virulent vaccines** are used for booster vaccination at approximately 7 to- 10 days, **usually in the drinking water**.

Vaccines of low virulence are suitable for chicks with a **lower level of maternal** immunity.

The advantage of these vaccines is that they do not cause the respiratory reactions or lead to a decline in growth rate, which can occur with vaccines of greater virulence.

The disadvantage of milder vaccines (**low virulence**)is that the level of immunity provided is low, and is only sufficient to protect the respiratory tract.

Often these vaccines will not protect tissues such as the kidney and oviduct against challenge with strains which are nephropathogenic or pathogenic for the reproductive tract.

Some live vaccines have a significant degree of residual virulence and a tendency to increase air sacculitis, particularly in adverse environmental conditions

Vaccines

Despite the widespread use of vaccines, IB continues to be one of the most economically important diseases of poultry worldwide

BECAUSE

The RNA-dependent RNA-polymerase present in in IBV, required for copying of the viral genome, is mainly responsible for the extremely high mutation rate. This enzyme does not have proofreading capability. So, when a mistake in copying the genome is made, the enzyme cannot go back and fix it. This high mutation rate creates a diverse population of virus particles. This adaptation is evident clinically as variant viruses or emerging new serotypes of the virus.

So, IBV continues to be one of the major pathogens of chickens throughout the world.

The control of IBV by vaccination is hampered because of the appearance of new emerging serotypes, which are only weakly affected by vaccination against the original serotypes. Although homologous live vaccines for IB are better than heterologous vaccines in controlling the disease, it is still recommended to develop the vaccines from different local strains to control IB.

Many antigenic types of IB vaccines are available and this is a major problem for many poultry growers. The choice of vaccines will decide the success of vaccination and should be based on prior information as to which **antigenic types are prevalent in the region.**

The vaccines used most frequently are based on **Mass strains**, examples of these are **M41, Ma5, H52 and H120**.

Other monovalent vaccines for regional use are also available, such as Conn 46, Ark 99, Florida, JMK, 4/72, D247, etc. Often two different antigenic types of vaccines are required.

Live vaccine strains from other parts of the world should not be used or introduced if prevailing endemic strains are of a different serotype or genetic lineage.

Inactivated vaccines

Inactivated vaccines are intended for use in layers and breeders. The vaccines are administered by subcutaneous inoculation at thirteen to eighteen weeks of age, to pullets which have been previously primed with live attenuated vaccines.

Inactivated vaccines provide **high and uniform levels of antibodies** that persist for longer periods than those induced by live vaccination.

These high levels of antibodies are particularly useful in providing protection for the internal organs by preventing spread of the virus.

In layers and breeders, inactivated vaccines provide protection against reductions in egg production, which might not always be afforded by live vaccination.


IB + ND + ED


ND BROILER

In addition, in breeders, progeny chicks will be protected by maternally transferred antibodies.
Progeny chicks that originate from breeders vaccinated with inactivated vaccines have high and uniform maternal antibody levels in comparison to broilers from dams vaccinated with live vaccines only.
Most of the inactivated vaccines are of one type, Mass M41; however, bivalent vaccines that incorporate additional variant antigens may also be necessary .
Inactivated vaccines are produced from IBV-infected allantoic fluid, which is inactivated and usually formulated as oil emulsion vaccine. The disadvantage of such vaccines is the expense.

Live vaccine should contain not less than $10^{3.5}$ EID50 per dose per bird until the expiry date indicated, and not less
Than $10^{2.5}$ EID50 per dose per bird after incubation at 37°C for 7 days at the time of issue.

Causes of the vaccinal break

Management

Bird

Vaccine

Infectious pressure

vaccine failures:

1-Vaccine preparations should be appropriately stored at all times and not used after the expiry date.

-2-The efficacy of vaccines will decline if stored for long periods at room temperature and if diluted in an inappropriate solution.

3-Vaccine failure may also result if the chosen vaccine is not protecting against the prevalent antigenic type; if the vaccine is too mild or too virulent; or if the vaccinating dose per chick is too low due to excessive dilution of the vaccine.

4-The route of vaccine application will affect the level and the duration of immunity as well as the vaccine reaction. The chosen route will depend upon previous experience or on the degree of clinical reaction in the flock.

Inactivated vaccines, if applied correctly, perform well in most situations. The lack of efficacy of inactivated vaccines may be due to inappropriate priming or because challenge strains are of a variant serotype which is not included in the common inactivated IB vaccines.

Headache or tired ?

Introduction

Duck Hepatitis (DH) is a highly contagious disease of young ducklings 1-28 days of age, characterized primarily by hepatitis. It can be caused by any of three different viruses namely duck hepatitis virus types 1, 2 and 3. The more common and internationally widespread is duck hepatitis virus (DHV) type 1, an enterovirus, which causes a highly lethal, acute, contagious infection in ducklings under 6 weeks of age and, frequently, under 3 weeks of age. The disease is rarely seen in ducklings over **4 weeks of age**. The onset of the disease is very rapid, it spreads quickly through the flock and may cause up to 90% mortality.

Sick ducklings develop spasmodic contractions of their legs and die within an hour in a typical "arched-backward" position. The liver is enlarged and shows hemorrhagic spots.

Nature of the disease

DVH is caused by three different viruses.

1-The most severe and widely distributed virus, duck hepatitis A virus **(DHAV) 1** (formerly called DHV-1), belongs to the Picornaviridae, and causes disease in ducklings before 6 weeks old.

2-The second viruses are duck astrovirus (formerly known as **DHV-2)**, which causes disease in ducklings between 6 and 10 weeks old

3-The third-**DHV-3** caused by another virus unrelated to DHV-1 and DHV-2 which causes milder disease.

It is RNA virus. It is propagated in 9 - 11 day old chicken embryo through allantoic sac causing death with stunting and edema of dead embryos.

The virus grows in cell culture of chicken and duck embryos origin with cytopathic effect. It is resistant to ether and chloroform. Relatively heat stable, and capable of survival for long periods under usual environmental conditions. Highly resistant to environmental conditions. The virus has not haemagglutinating affinity

Transmission

The disease is very contagious transmitted by infected ducks and other waterfowl and spreads rapidly, the virus excreted by faeces and transmitted by direct contact between birds or through fomites such as brooders, water, feed, equipment.

Recovered animals can shed the virus for up to 8 weeks.

No egg transmission.

Clinical signs

1-DHV-1 causes the most severe disease.

2-The incubation period lasts 1 to 2 days .

3-The onset and spread of the disease are very rapid, with practically all mortality occurring within 3-4 days.

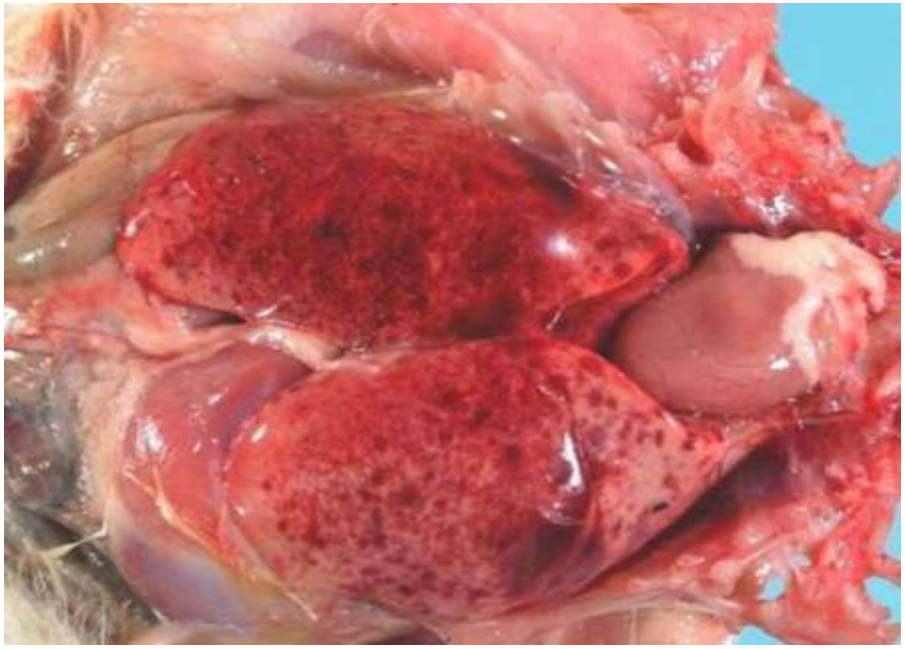
4- At first, birds stop moving, lethargy, anorexia and squat down with eyes partially closed. Birds fall on their sides, kick spasmodically(opisthotonos) with both legs, and die with heads drawn back. Death occurs within hours or so after signs are noted.

5- Morbidity is often 100% and mortality reaches 80%.

6-Disease is less severe in ducks older than 7 weeks.

Post-mortem findings

The liver is enlarged with haemorrhagic lesions (petechia, ecchymosis) and decolouration. The spleenenlarged and swelling with some Congestion of renal blood vessels may also be apparent.



Differential diagnosis

Duck virus enteritis

Coccidiosis

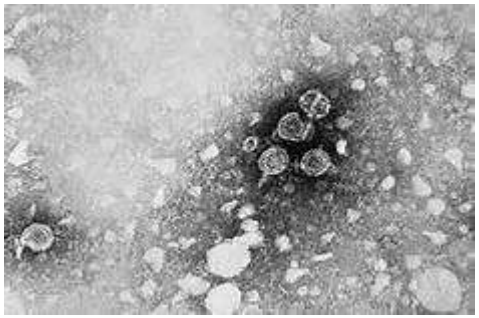
Mycotoxicosis

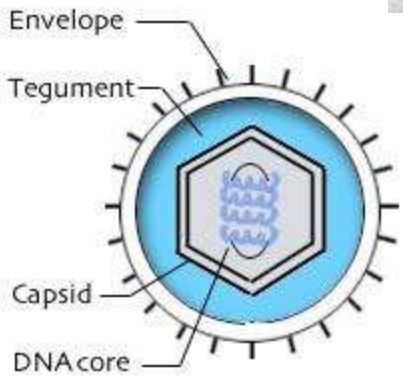
Pasteurella anatipestifera

Control / vaccines

If accidentally introduced, strict isolation and control of rats are necessary measures to control DHV. Rats have been described as a reservoir and control of this pest on arrival should be systematic

Vaccination against DHAV-1 and DHV-3 is possible using live attenuated vaccines. A killed vaccine is also available against DHAV-1.





http://stdgen.northwestern.edu/stdgen/bacteria/hhv2/herpes.diogram.jpg

Introduction

Duck Hepatitis (DH) is a highly contagious disease of young ducklings 1-28 days of age, characterized primarily by hepatitis. It can be caused by any of three different viruses namely duck hepatitis virus types 1, 2 and 3. The more common and internationally widespread is duck hepatitis virus (DHV) type 1, an enterovirus, which causes a highly lethal, acute, contagious infection in ducklings under 6 weeks of age and, frequently, under 3 weeks of age. The disease is rarely seen in ducklings over **4 weeks of age**. The onset of the disease is very rapid, it spreads quickly through the flock and may cause up to 90% mortality.

Sick ducklings develop spasmodic contractions of their legs and die within an hour in a typical "arched-backward" position. The liver is enlarged and shows hemorrhagic spots.

Nature of the disease

DVH is caused by three different viruses.

1-The most severe and widely distributed virus, duck hepatitis A virus **(DHAV) 1** (formerly called DHV-1), belongs to the Picornaviridae, and causes disease in ducklings before 6 weeks old.

2-The second viruses are duck astrovirus (formerly known as **DHV-2)**, which causes disease in ducklings between 6 and 10 weeks old

3-The third-**DHV-3** caused by another virus unrelated to DHV-1 and DHV-2 which causes milder disease.

It is RNA virus. It is propagated in 9 - 11 day old chicken embryo through allantoic sac causing death with stunting and edema of dead embryos.

The virus grows in cell culture of chicken and duck embryos origin with cytopathic effect. It is resistant to ether and chloroform. Relatively heat stable, and capable of survival for long periods under usual environmental conditions. Highly resistant to environmental conditions. The virus has not haemagglutinating affinity

Transmission

The disease is very contagious transmitted by infected ducks and other waterfowl and spreads rapidly, the virus excreted by faeces and transmitted by direct contact between birds or through fomites such as brooders, water, feed, equipment.

Recovered animals can shed the virus for up to 8 weeks.

No egg transmission.

Clinical signs

1-DHV-1 causes the most severe disease.

2-The incubation period lasts 1 to 2 days .

3-The onset and spread of the disease are very rapid, with practically all mortality occurring within 3-4 days.

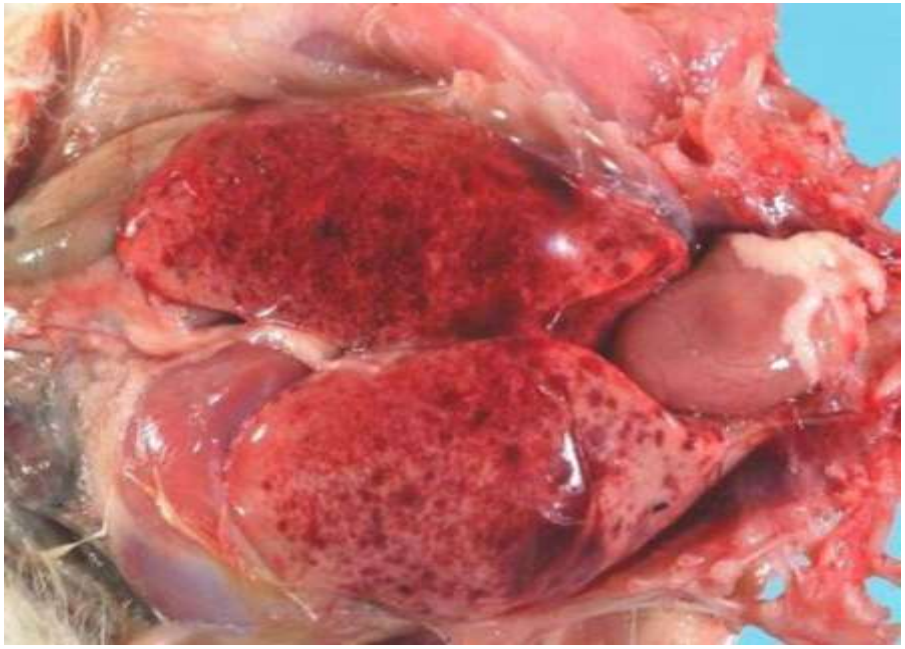
4- At first, birds stop moving, lethargy, anorexia and squat down with eyes partially closed. Birds fall on their sides, kick spasmodically(opisthotonos) with both legs, and die with heads drawn back. Death occurs within hours or so after signs are noted.

5- Morbidity is often 100% and mortality reaches 80%.

6-Disease is less severe in ducks older than 7 weeks.

Post-mortem findings

The liver is enlarged with haemorrhagic lesions (petechia, ecchymosis) and decolouration. The spleenenlarged and swelling with some Congestion of renal blood vessels may also be apparent.



Differential diagnosis

Duck virus enteritis

Coccidiosis

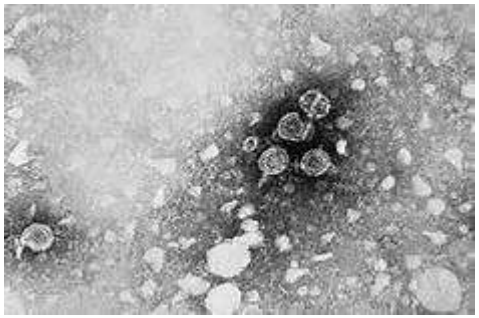
Mycotoxicosis

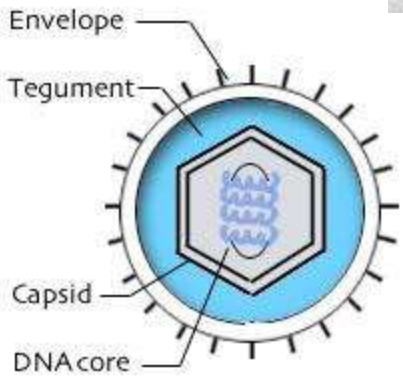
Pasteurella anatipestifera

Control / vaccines

If accidentally introduced, strict isolation and control of rats are necessary measures to control DHV. Rats have been described as a reservoir and control of this pest on arrival should be systematic

Vaccination against DHAV-1 and DHV-3 is possible using live attenuated vaccines. A killed vaccine is also available against DHAV-1.





http://stdgen.northwestern.edu/stdgen/bacteria/hhv2/herpes.diogram.jpg

Golden medal

IB Ma5 day old +Inactivated vaccine and IB 4/91 day 12-14. This is broad protection

Thanks