# Safeguarding Egg Yield from Viral Diseases

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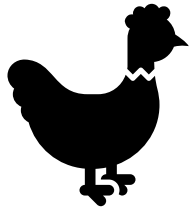
01 Infectious bronchitis

02 E.D.S

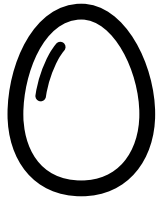
03 Newcastle disease

# Infectious bronchitis

# About the disease

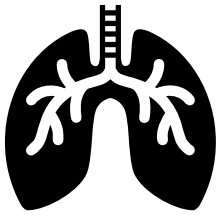


**Affects oviduct of layers & breeder**

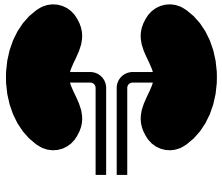


**Egg production losses in layers & breeders may reach to 50%**

# About the disease



**The respiratory form is characterized by up to 100% morbidity, low mortality, except in complicated cases**



**The nephrotropic cases of I.B. were known in 1963 in Australia.**
**Renal form may cause 15–25% mortality**

# 01

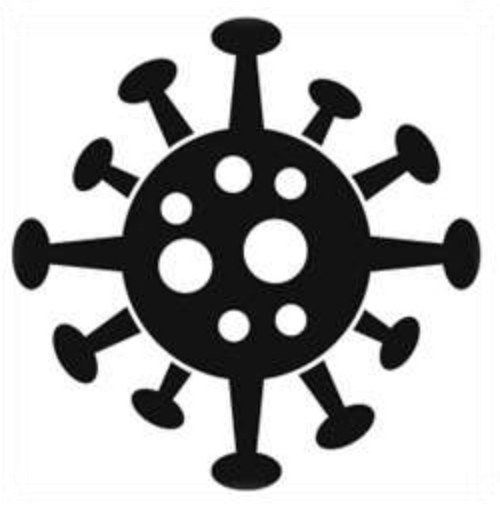
## Causative agent

# Infectious bronchitis virus (I.B.V)

**Coronaviridae family**

↓

**Infectious bronchitis virus (I.B.V)**



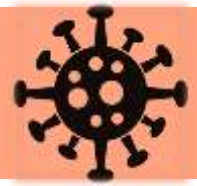
**Coronaviruses**

↓

**3 subgroups**

**All avian coronaviruses are placed in group 3**

# I.B.V



Highly species-specific

Replicates in the cytoplasm of the infected cells

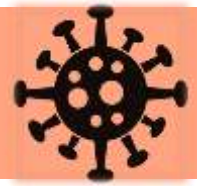
Destroys cilia & localize in subepithelial cells of the trachea

# I.B.V

Highly mutant with several classical & variant strains

Survives outdoors for 12 – 56 days

# I.B.V



Destroyed by heat “56˚C after 15 minutes

Sensitive to common disinfectants
Inactivated by 0.1% sodium hydroxide at 4˚C for 18 h

# 02

## Spread of I.B

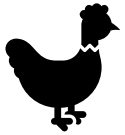
# Routes of spread

**Inhalation**

**Infected feces**

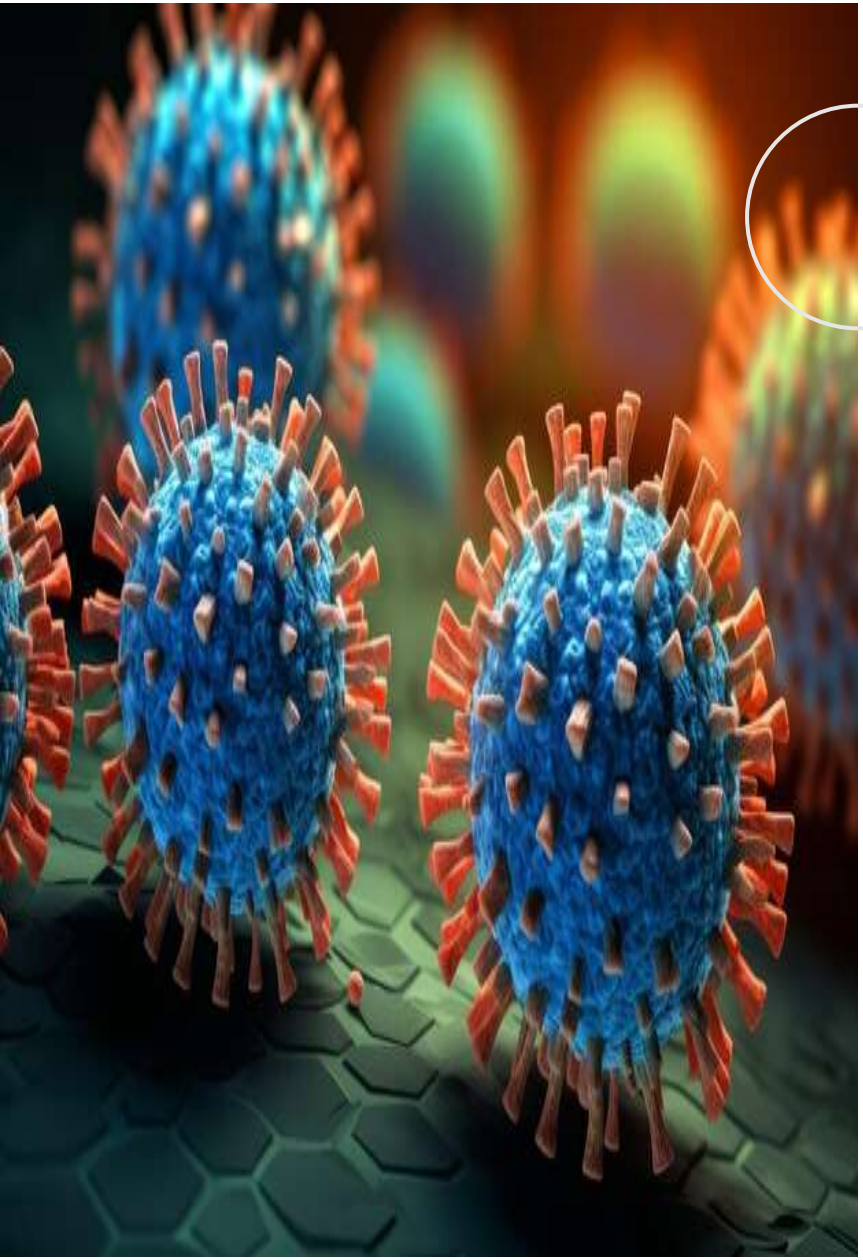
**Cycle from bird to bird**

World-wide spread for a considerable distance



Some birds still carriers for several months

Disease occurs throughout the year



# 03

# Pathogenesis

Incubation period 18–36 hours, although signs may not be evident until 9–10 days P.I.

Attacks the epithelial cells of the upper & the lower respiratory tract

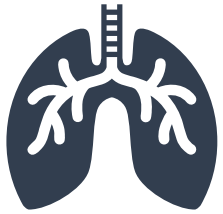
Damages the trachea & the air sacs

Predisposes to secondary bacterial infection

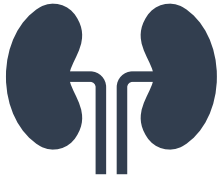
# 04

## Forms of I.B

**Respiratory form**



**Renal form**



**Reproductive form**

## Reproductive form

Flocks are highly susceptible from 17 weeks peak production

In case of infection at point of lay “fully functional oviduct”

In many instance 6–12% subnormal production is recorded

Early infection by I.B (within 2 weeks )lead to permanent damage of the oviduct ,poor laying capacity.

Severe drop in egg production by 25-50% for 6-8 weeks, then return to full lay

Deterioration of internal quality (3-4 weeks after appearance of signs)

Pale eggs appear 2–5 days post exposure to infection

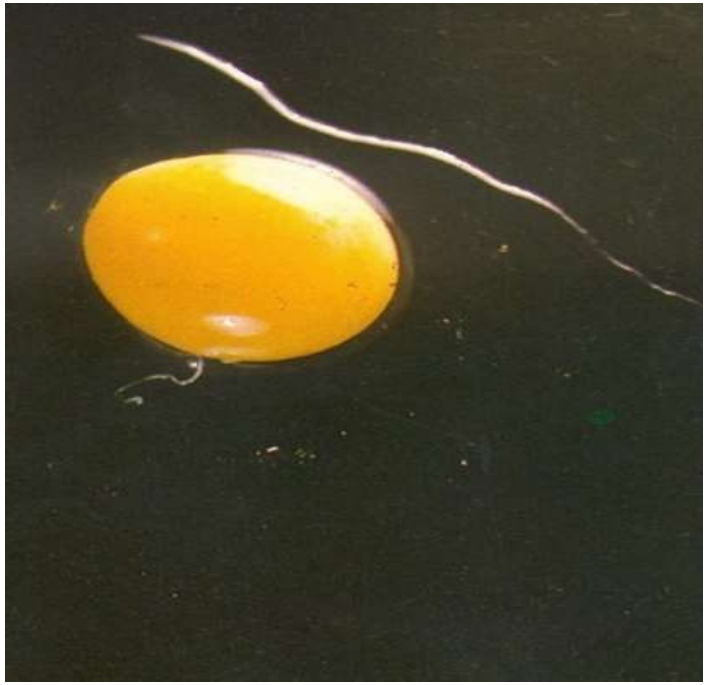
Impacted oviduct, cystic oviduct & ruptured ova

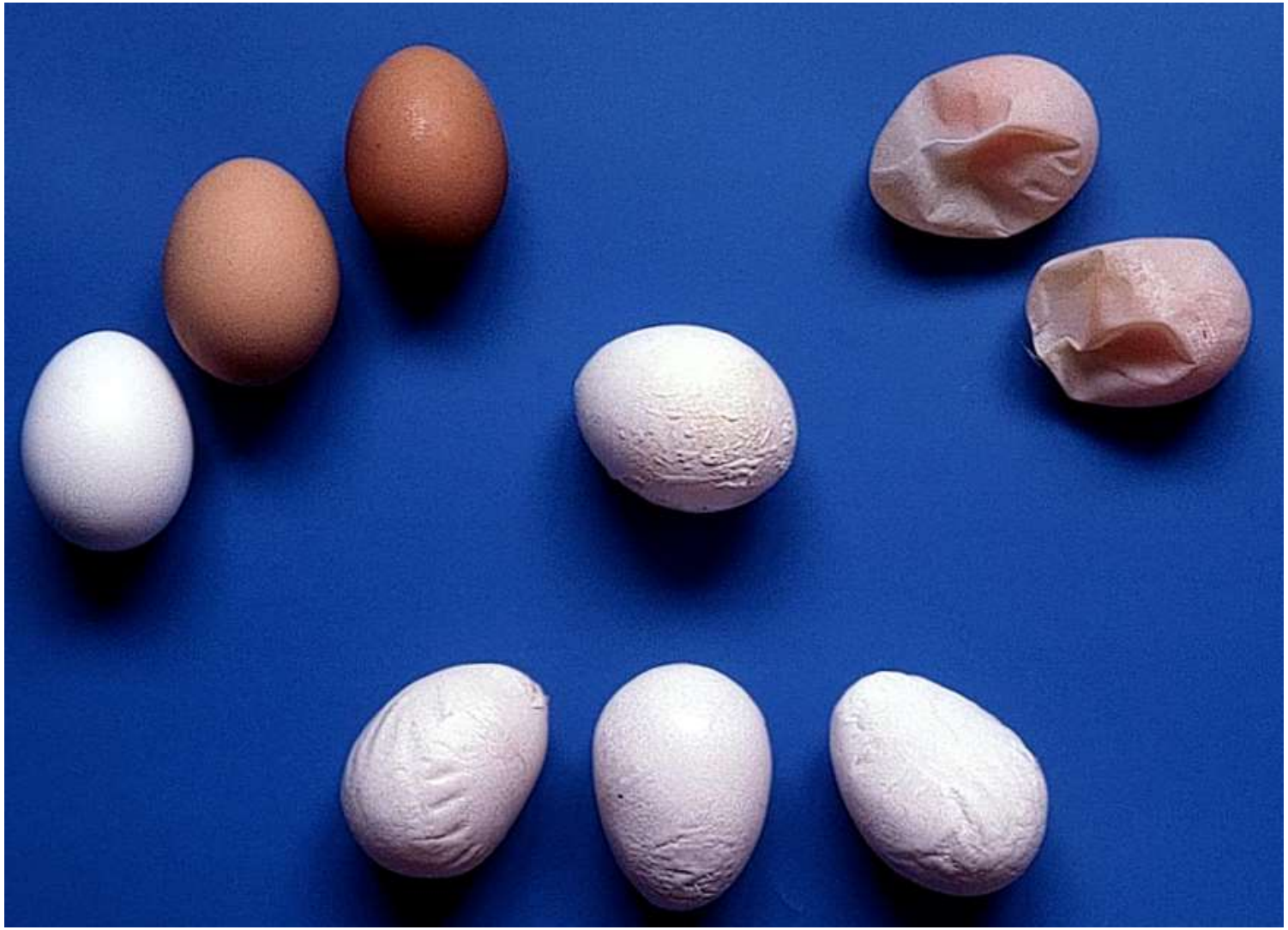
Misshapen, thin shell, shelless eggs & watery albumen

Yolk frequently present in abdominal cavity

Flaccid ovarian follicles

Hypoplastic or cystic oviduct





# 05

# Protection against I.B

# Protection against I.B

**Protection**

**Immunity**

**Vaccination**

# Protection

There is no cross-protection between different serotypes

Some IB serotypes have cross-protective ability against other serotypes (Protectotype)

When two different I.B serotypes are used, immunity develops against both, & some other serotypes

# Immunity

Maternal immunity protect for 2-3 weeks

M.A. reduce severity of vaccinal reaction

Vaccination at day one dose not interfere with M.A

Humoral antibodies appear 10-14 days post-vaccination

# Immunity

Antibodies can be detected 7-14 days post-infection, or vaccination

Respiratory protection continue for 3-4 weeks or more after infection, or immunization

Polyvalent vaccine produce severe P.V.R

Birds usually develop airsacculitis after vaccination

# Vaccination

Purpose of vaccination

Types of vaccines

P.V.R

# Types of vaccines

Vaccination for broilers

Vaccination for layers, breeders

# Vaccination for broilers

Monovalent (Mass.) by coarse spray at the hatchery

Bivalent (Mass. + variant) in areas where the variant is present

Second dose at 14 days by spray or eye drop

**Vaccination for layers, breeders**

**Several live attenuated vaccines during rearing**

**Inactivated vaccine at least 2 times before production**

# Factors exaggerating P.V.R

Fine spray administration

Long intervals between live vaccinations

High level of ammonia & dust

Immunosuppression, other respiratory diseases “H9, ND.”

# Factors exaggerating P.V.R

- Vaccination of M.G, M.S positive birds
- Strong vaccine (H52) used as a primary
- Poor vaccination technique

# Egg drop syndrome (EDS 76)

# Three groups of adenoviruses are widely spread among avian species

# Adenoviruses

Non-enveloped DNA viruses

Propagated in T.C, produces inclusion bodies in nuclei & CPE

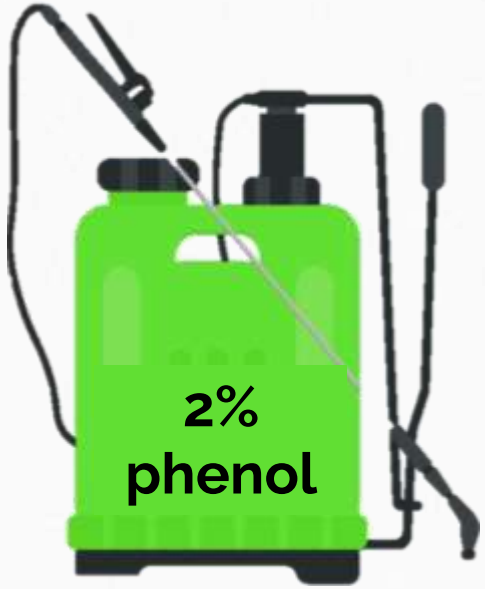
Some viruses cause stunting in chicken embryos.

Some serotypes agglutinate RBC of rats, sheep, poultry, and share a common group of C.F antigen

# Adenoviruses

**Highly resistant to disinfectants**


**3-9**



**2% phenol**

**70 ° c for 30 min**

# The three important groups

CELO
(Chicken Embryo Lethal Orphan)

Quail bronchitis

Inclusion body hepatitis
(I.B.H)

Group 2

Marble spleen of pheasants

Turkey Haemorrhagic Enteritis
(T.H.E)

Group 3

E.D.S

# 5 genera , 3 of them cause diseases in avian species

| | | |
|---|---|---|
| Avi adenovirus (conventional) Group I | Si adenovirus Group II | At adenovirus Group III |
| I.B.H H.H.S | T.H.E | E.D.S |

A disease of breeders & layers characterized by

loss of eggshell pigmentation

Thin and soft shell

Shell-less egg

Causative agent

Adenovirus group III one serotype
(natural adeno of ducks, geese)

# Transmission

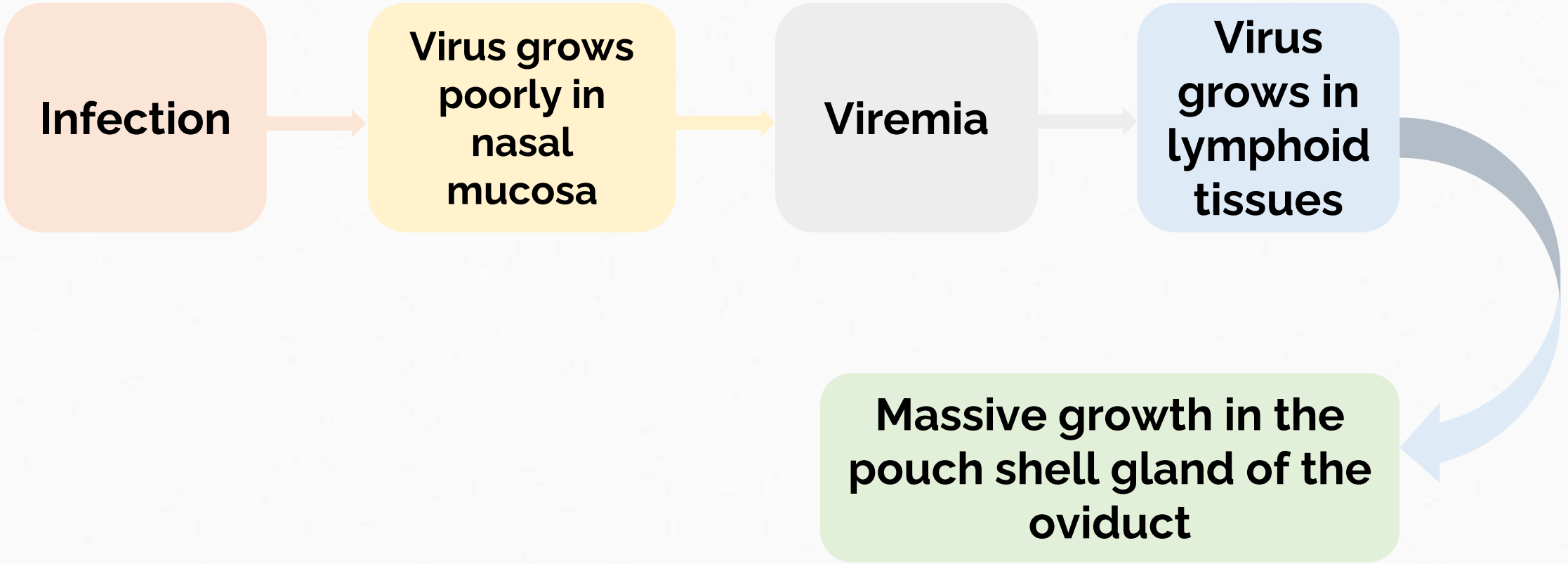
Vertical transmission (latent infection)

Live vaccines prepared from duck embryos

Contaminated needles, insects

Feces do not contain virus; droppings do because exudate of oviduct contain virus

# Pathogenesis



# Pathogenesis

Both normal, affected eggs contain virus externally & internally for next 2 weeks

Hatched chicks from infected eggs do not develop antibody, but are latently infected

At the peak of production, the virus reactivates again

# Clinical signs

Infections in waterfowl are usually asymptomatic

Egg losses are more severe in floor birds

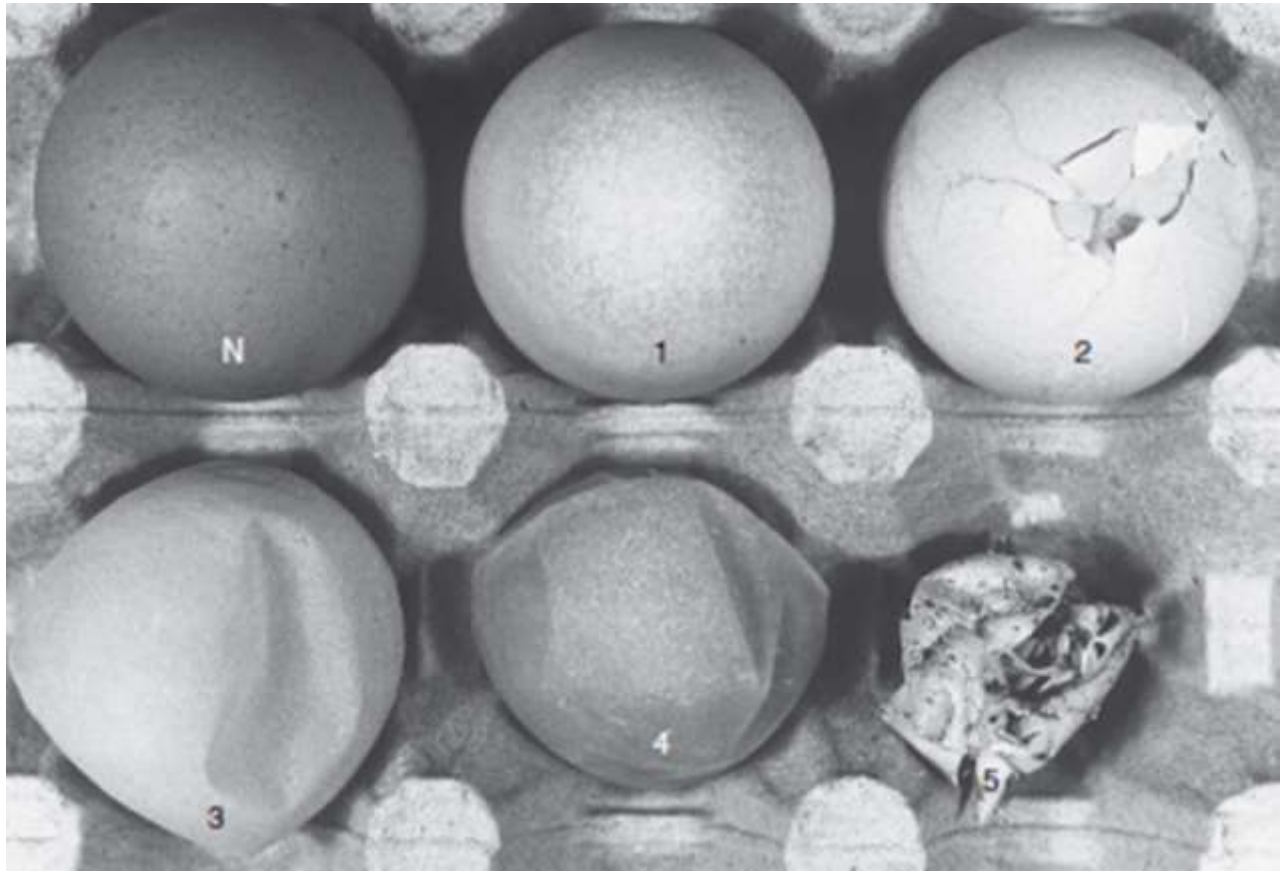
Losses continue 4-10 weeks , may reach up to 50 %

# Clinical signs

- Loss of shell strength & pigmentation
- Thin shell, soft shelled, shell less eggs
- Mineral deposition of the shell
- Birds are usually healthy, rarely depressed for 48 hours
- Diarrhea due to excess oviduct secretions

# Vaccines 

Inactivated, used by the IM route before laying（ at 14-18 weeks ）


N
1
2
3
4
5

# Newcastle disease

# New castle disease (Avian Pneumo-encephalitis)

Acute, Contagious disease of many kinds of Poultry, Wild and Cage birds

Suspected in any flock in which sudden death , high mortality following severe depression, inappetence , respiratory or enteric signs , drastic drops of production

Mortality may be high in absence of any signs

# Etiology

1

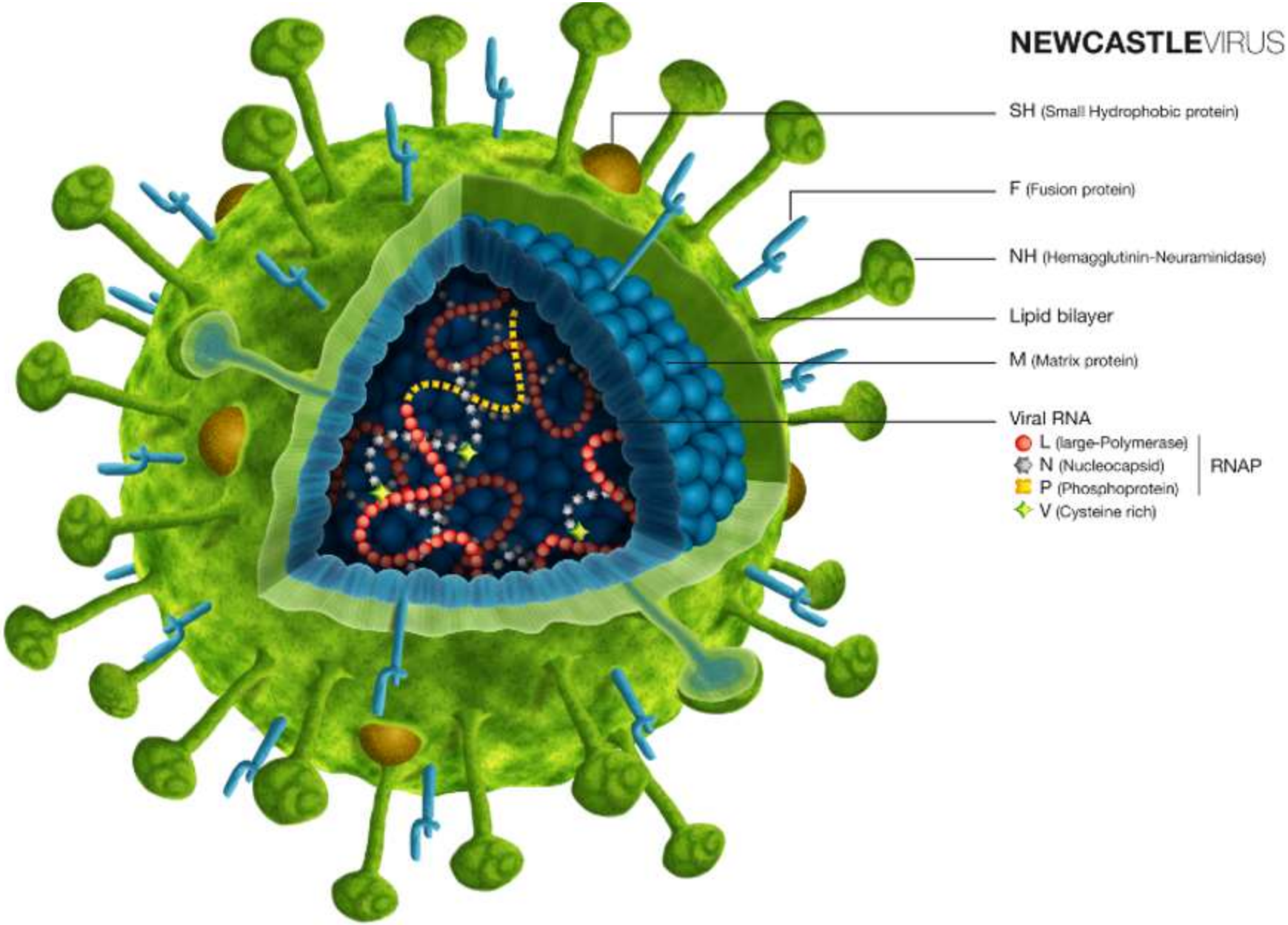
New castle disease virus

Avian Avulavirus

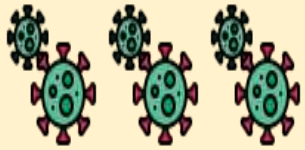
Single stranded enveloped RNA VIRUS belongs to (PMVI)

Genus Orthoavulavirus

Subfamily Avulavirinae

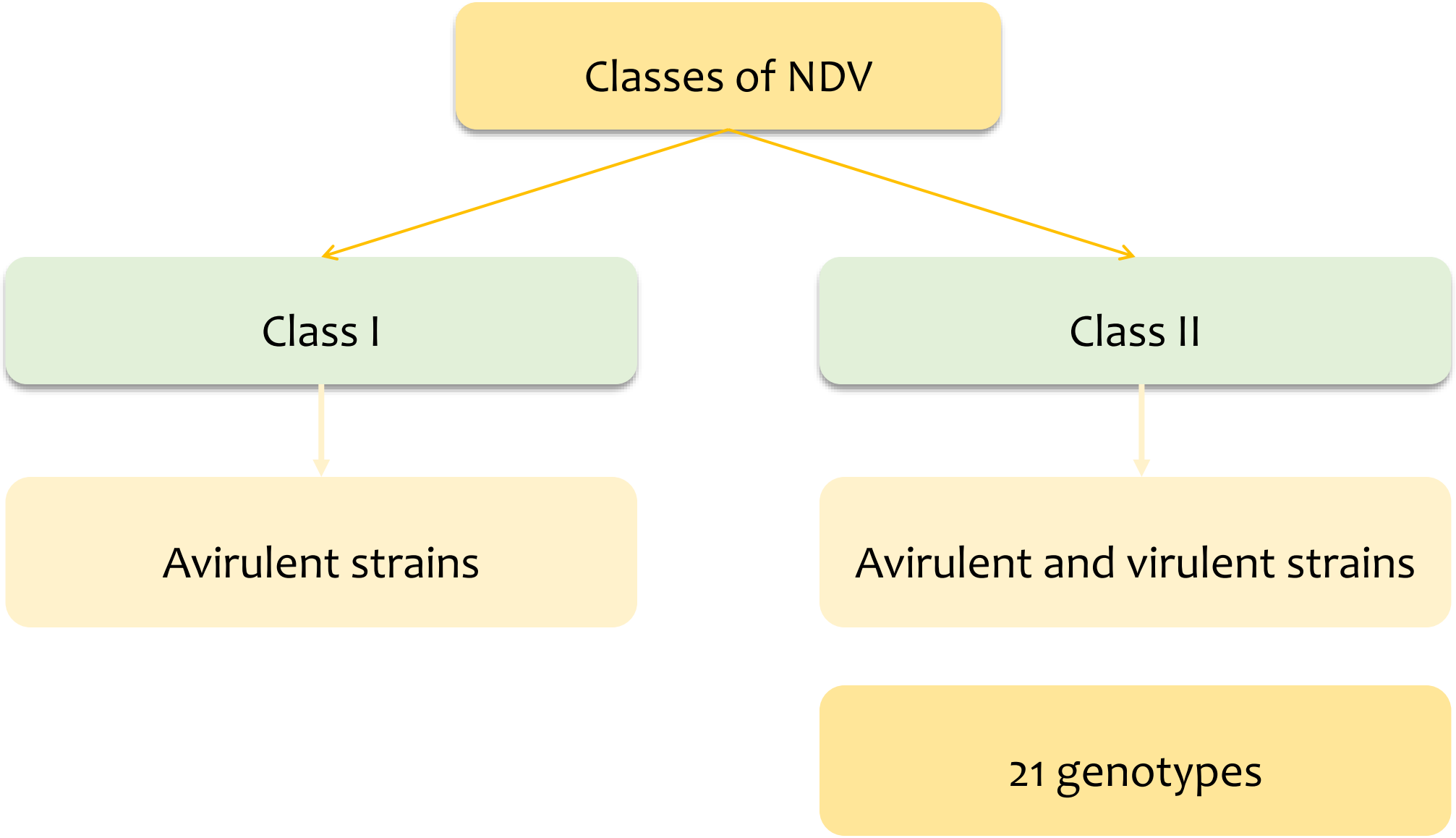

NEWCASTLEVIRUS
SH (Small Hydrophobic protein)
F (Fusion protein)
NH (Hemagglutinin-Neuraminidase)
Lipid bilayer
M (Matrix protein)
Viral RNA
L (large-Polymerase)
N (Nucleocapsid)
P (Phosphoprotein)
V (Cysteine rich)
RNAP

Replicates in the cytoplasm



Aldehyde & chlorine sensitive

Agglutinates avian RBCs


Classes of NDV
Class I
Class II
Avirulent strains
Avirulent and virulent strains
21 genotypes

Class II

21 genotypes

Genotypes V,VI &VII

Widely circulating in the world

Genotype VII

Many recent epidemics in Asia , Africa , the Middle east & S.America

Genotypes II,VI

Identified in Europe & S.America

# Pathotypes 2

Viscerotropic Velogenic

Neurotropic Velogenic

Mesogenic NDVs

Lentogenic NDVs

Asymptomatic Enteric NDVs

Viscerotropic Velogenic NDVs

Causes acute lethal infections , hemorrhagic lesions of the intestinal tract

Neurotropic Velogenic NDs

Causes high mortality following respiratory & nervous signs

| | |
|---|---|
| Mesogenic NDVs | Causes respiratory, and sometimes nervous signs |

| | |
|---|---|
| Lentogenic NDVs | Cause mild, or in apparent respiratory infections |

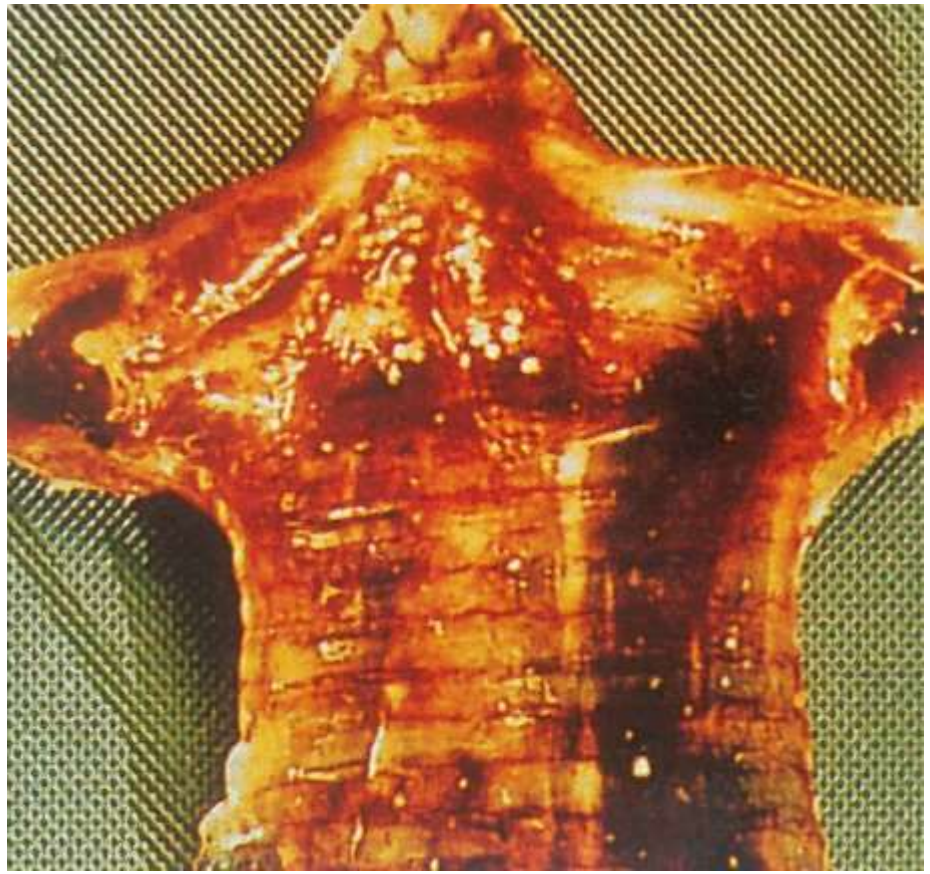
Asymptomatic Enteric NDVs

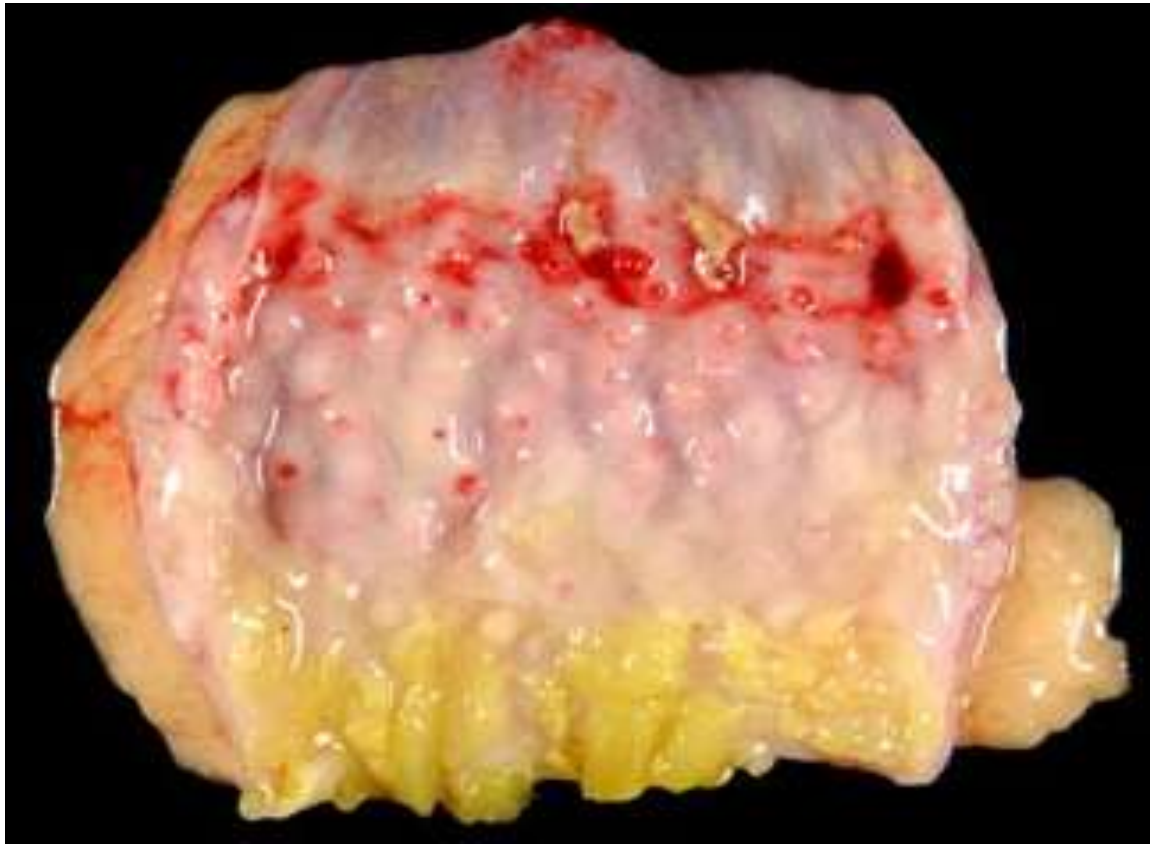
Causes avirulent infections in which replication appears to occur in the gut

# Clinical picture

3

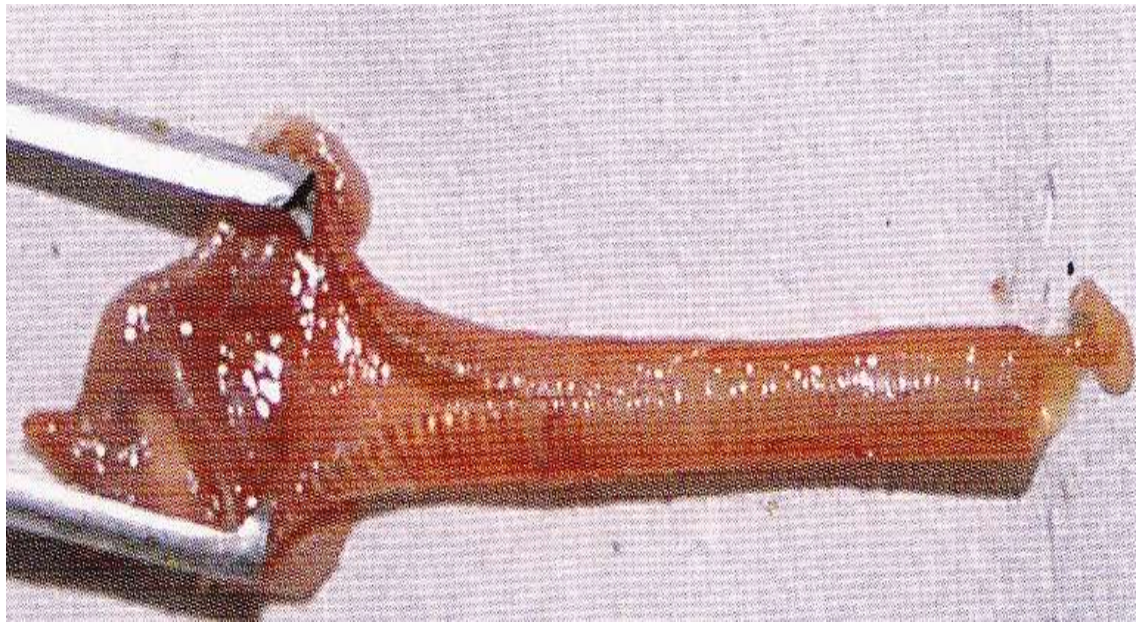
| Clinical signs | Velogenic | | Mesogenic | Lentogenic | Apathogenic |
|---|---|---|---|---|---|
| | Viscera | Neuro | | | |
| Diarrhea | +++ | _____ | _____ | _____ | _____ |
| Respiratory | _____ | +++ | ++ | (+) | _____ |
| Nervous | (++) | +++ | (++) | _____ | _____ |
| Drop of eggs | +++ | +++ | ++ | (+) | _____ |
| Morbidity | +++ | +++ | ++ | (+) | _____ |
| mortality | +++ | ++ | + | (+) | _____ |

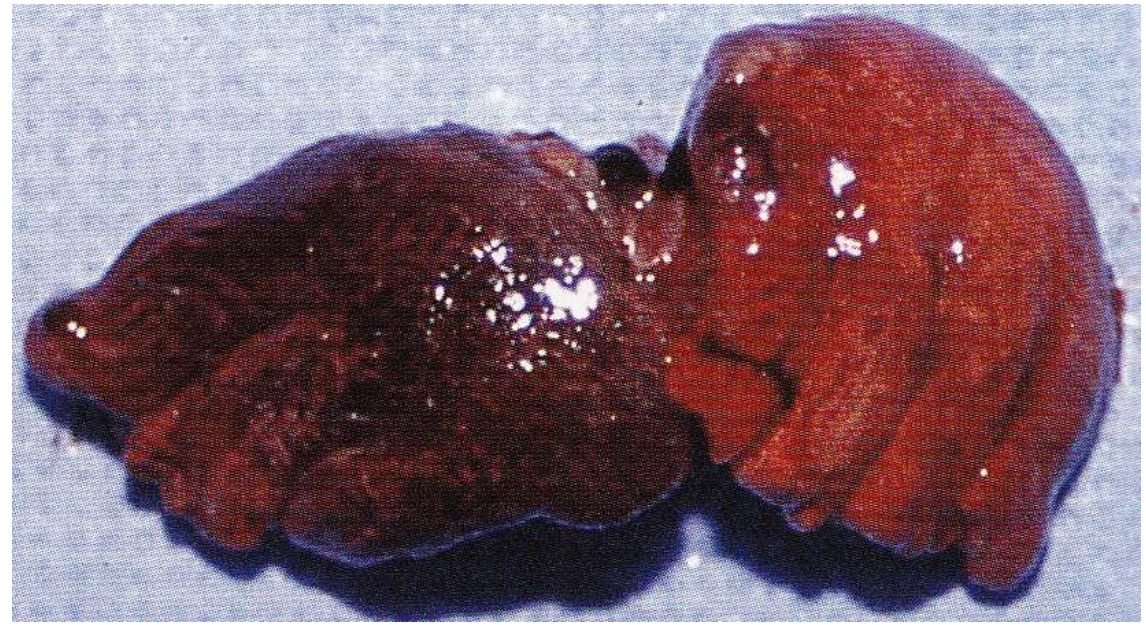


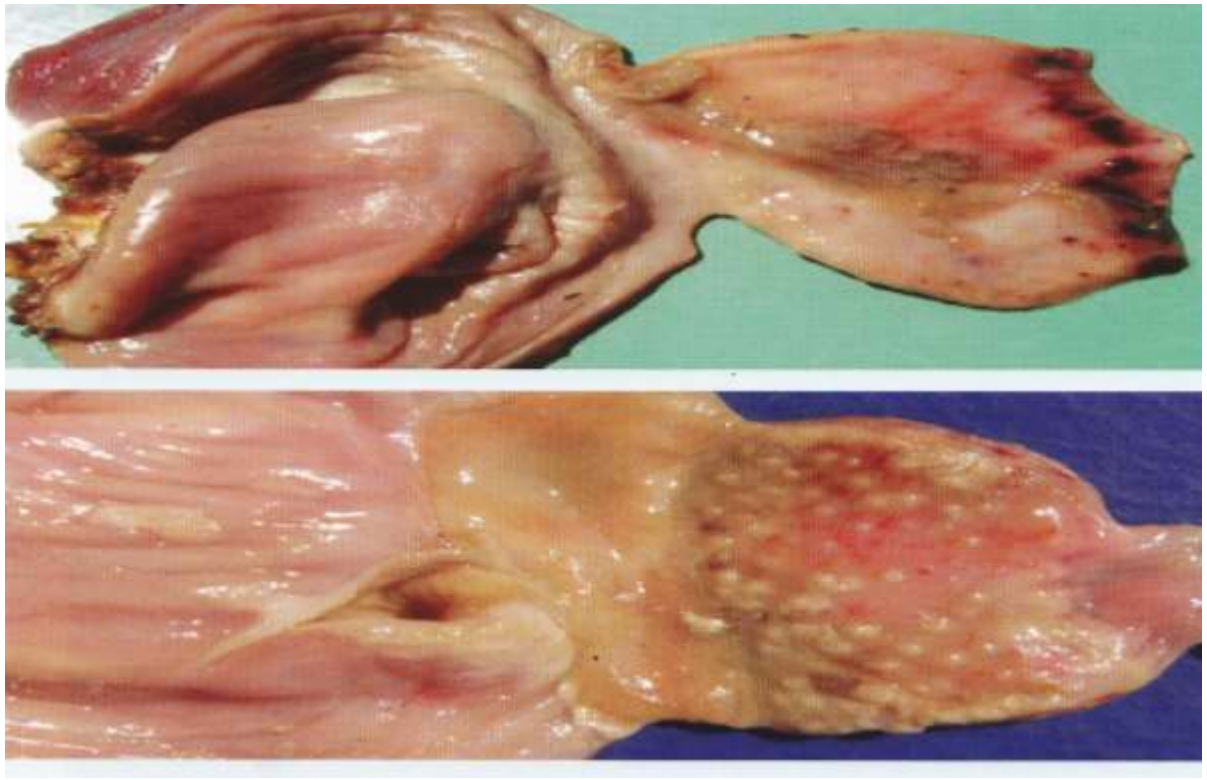


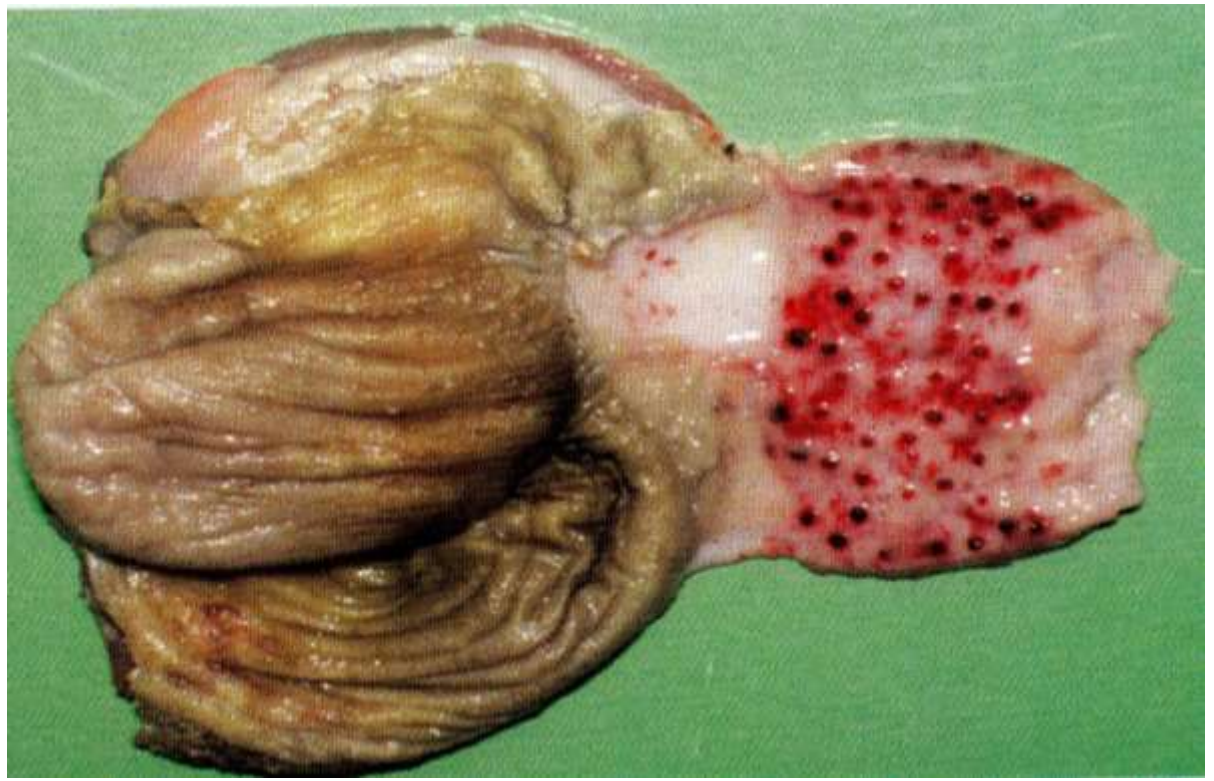

a

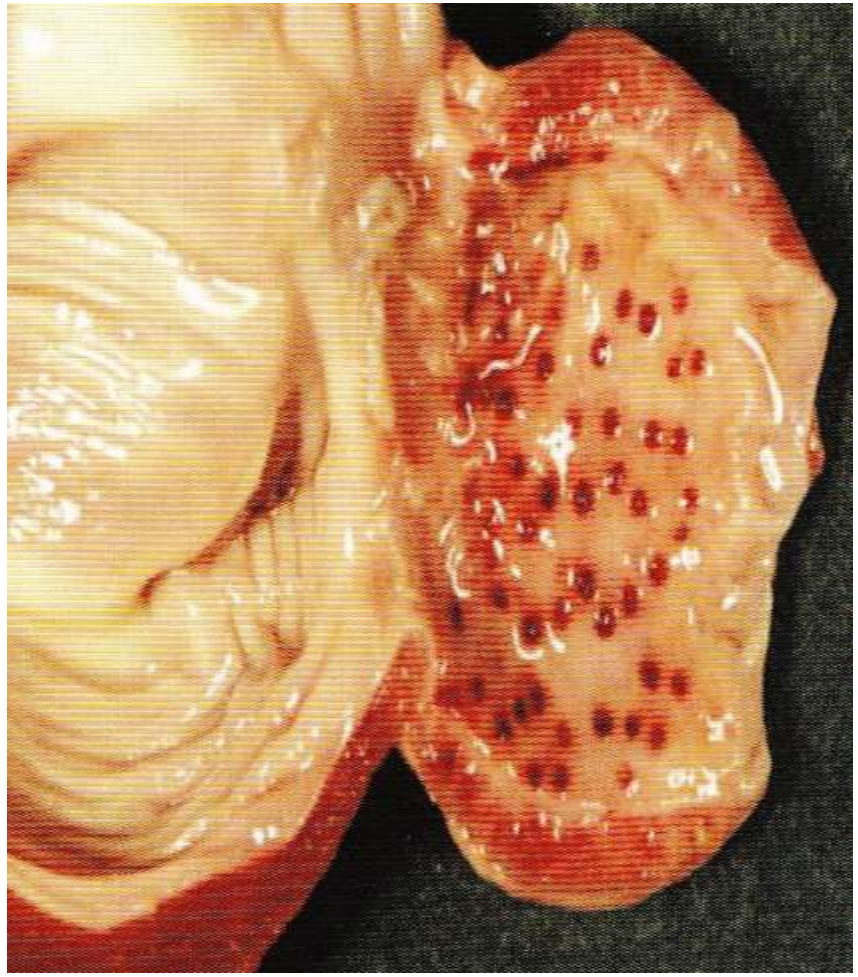

b

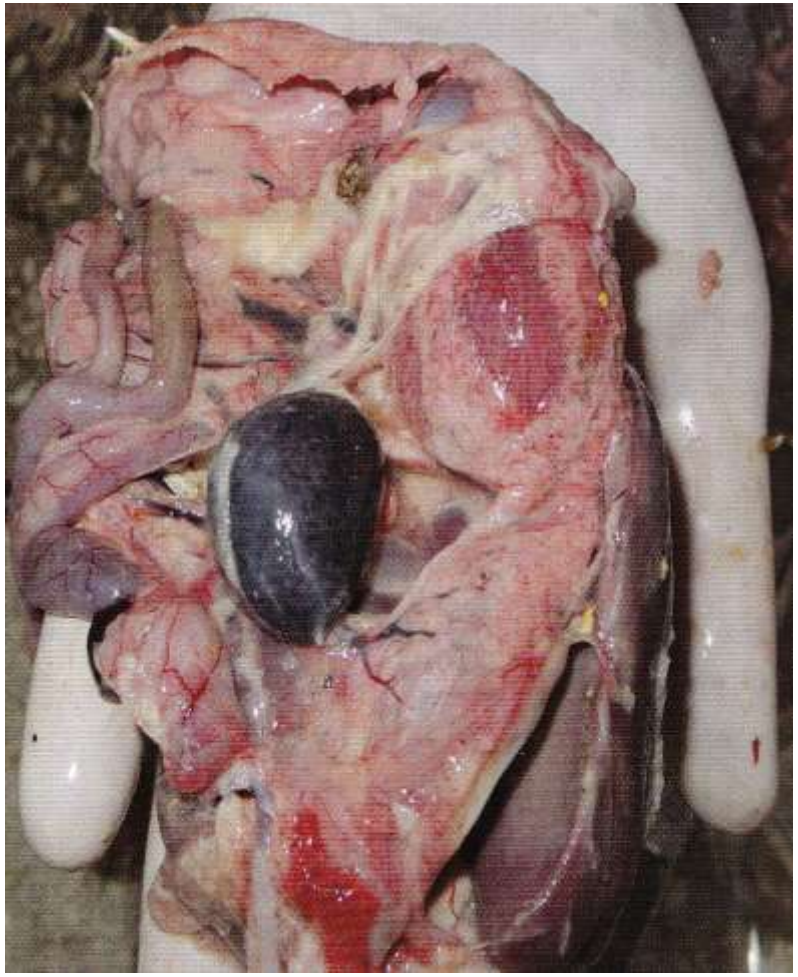


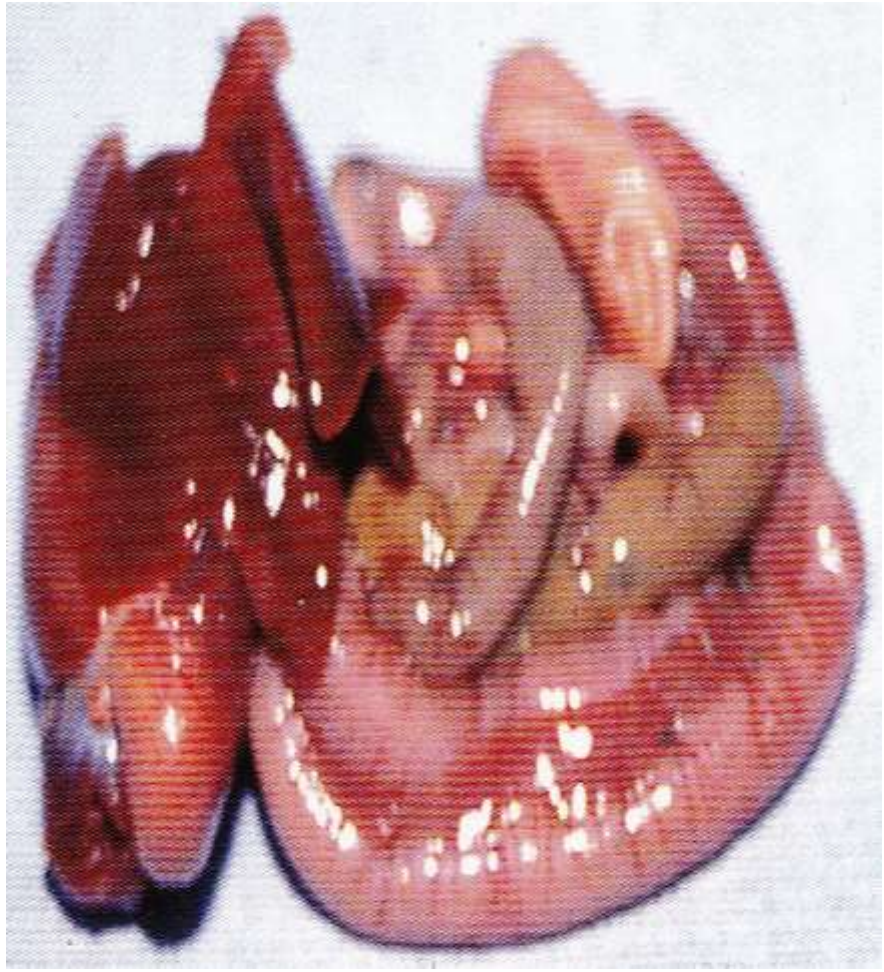


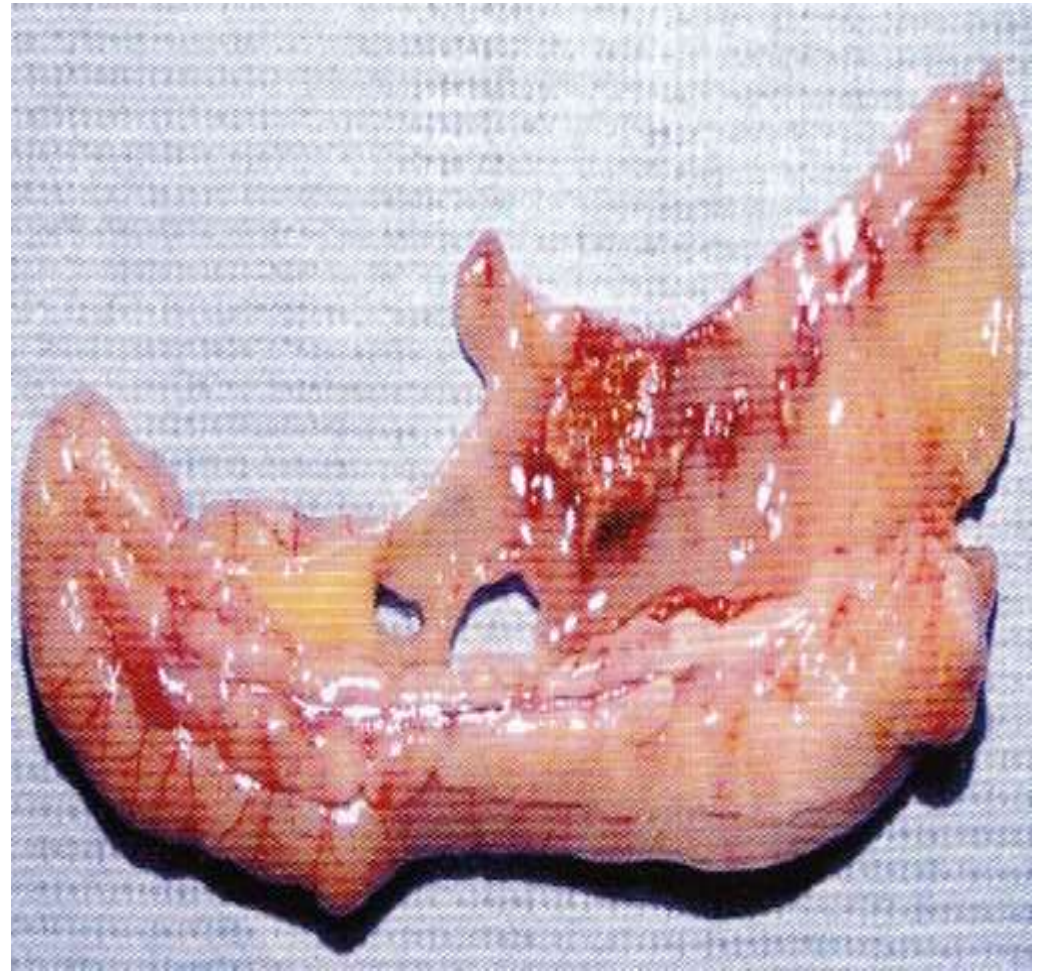


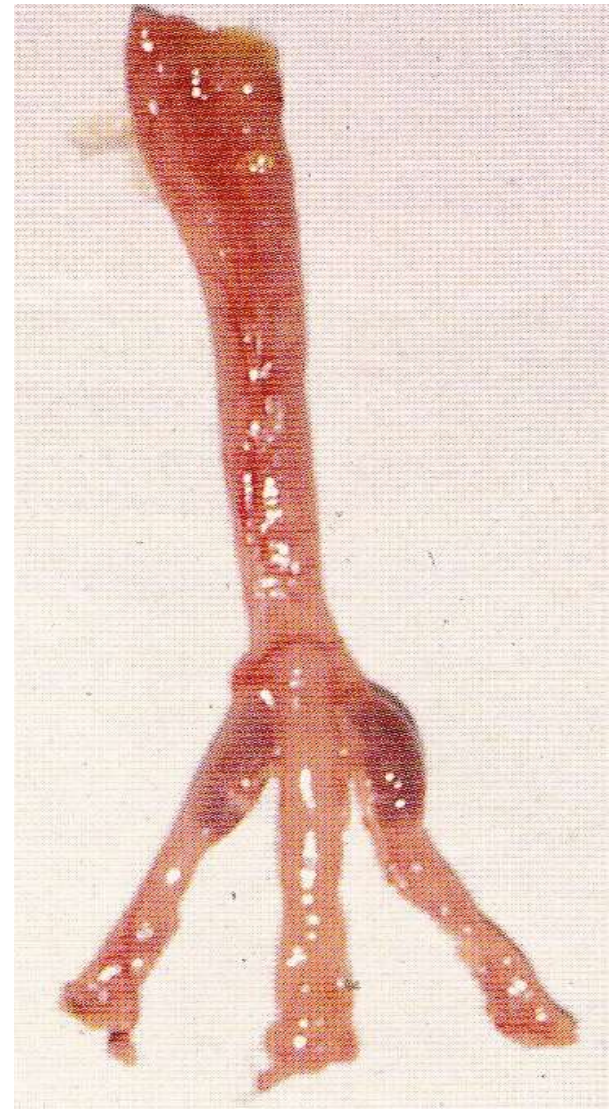


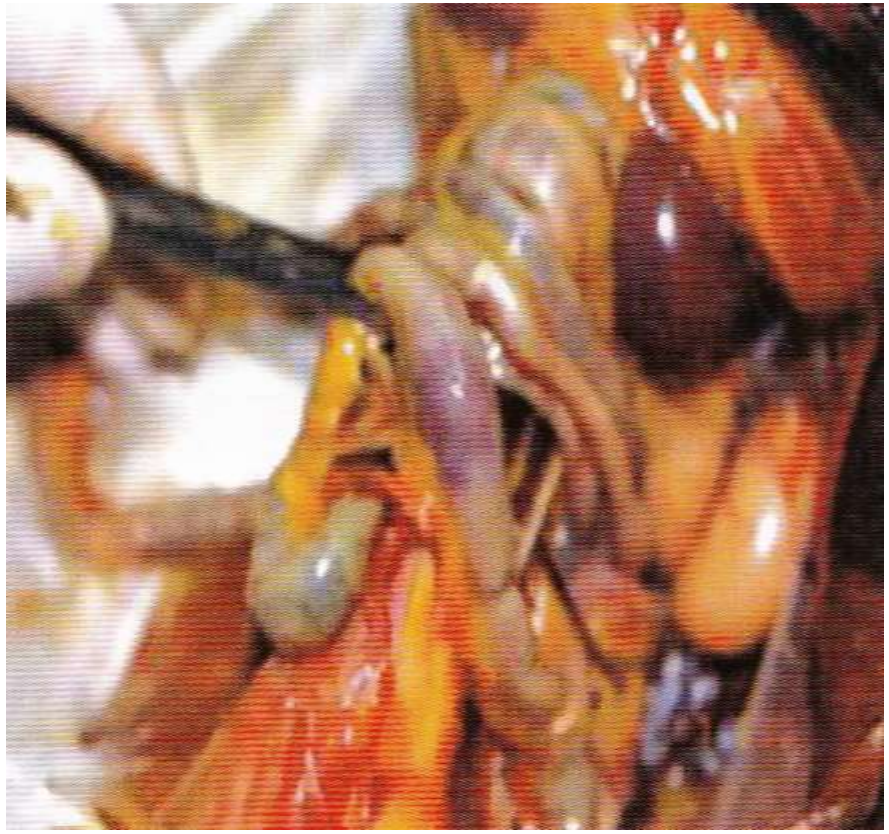


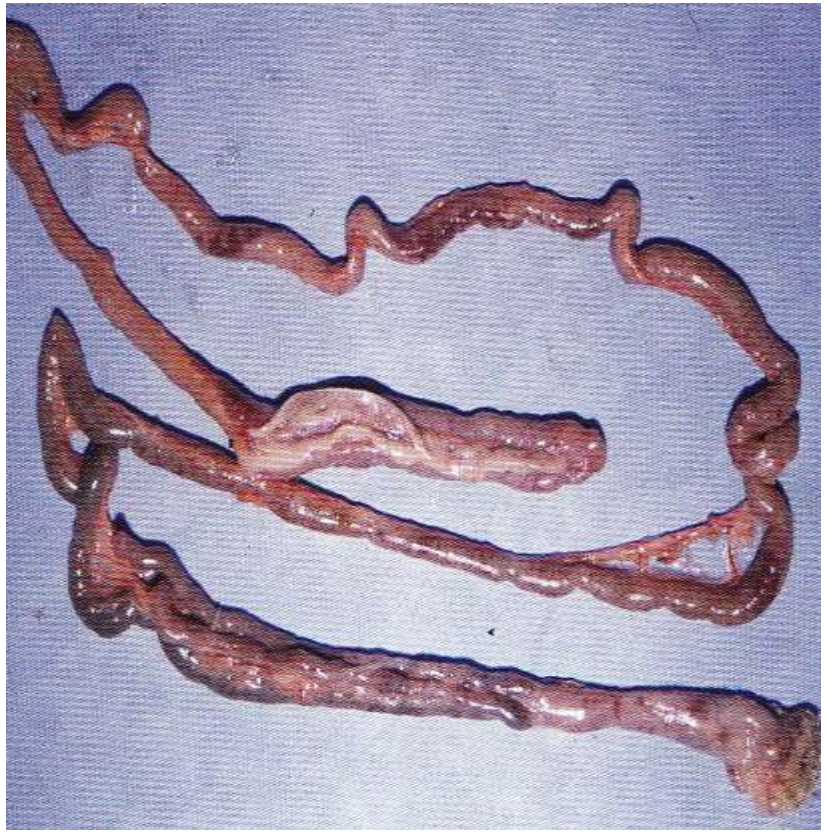


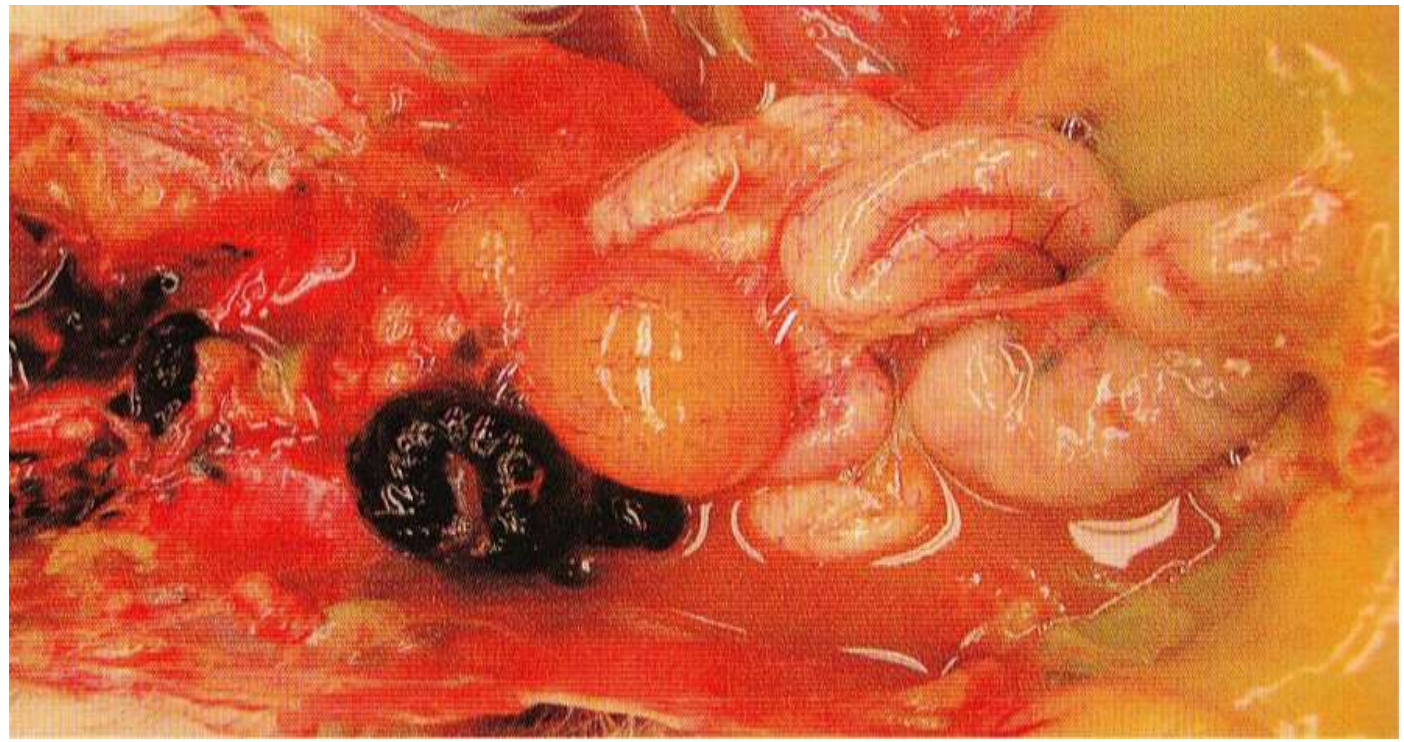


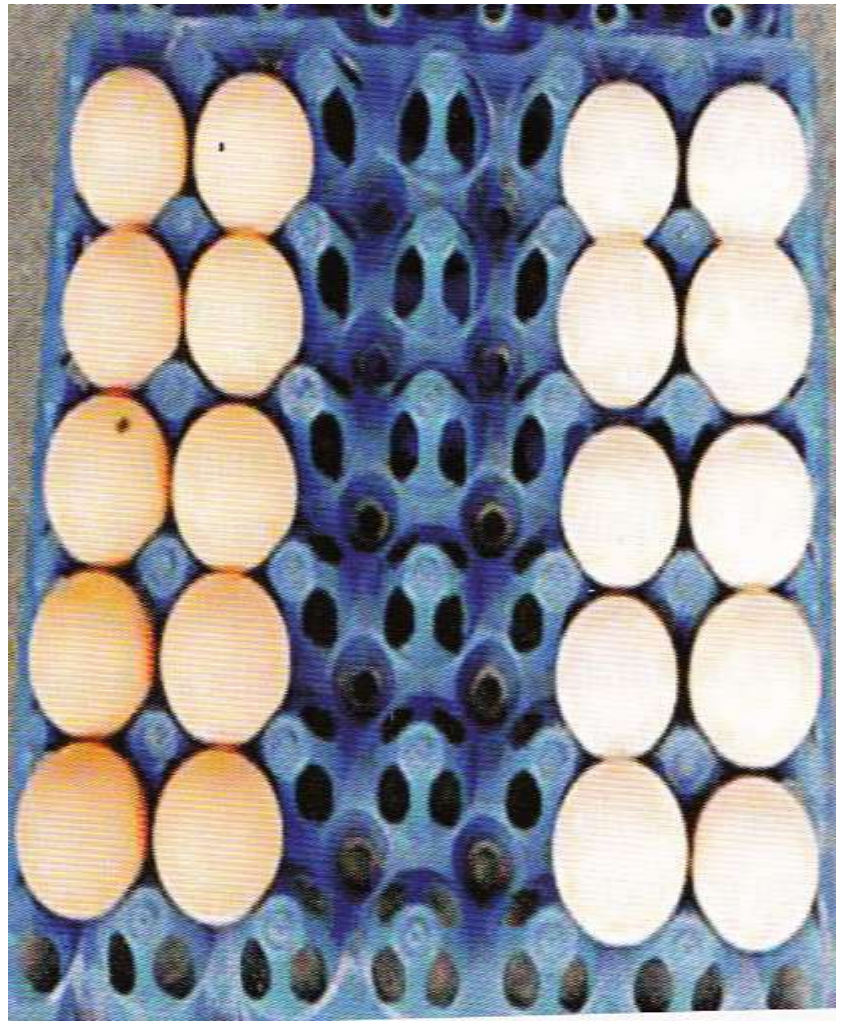


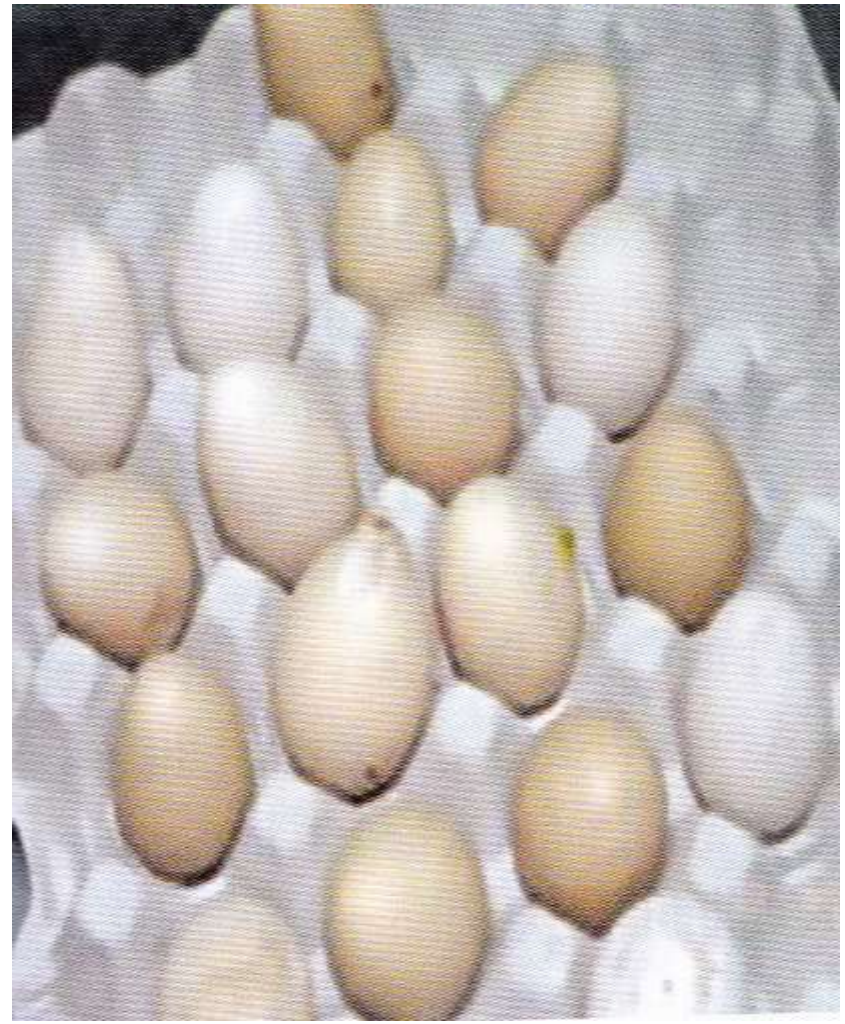










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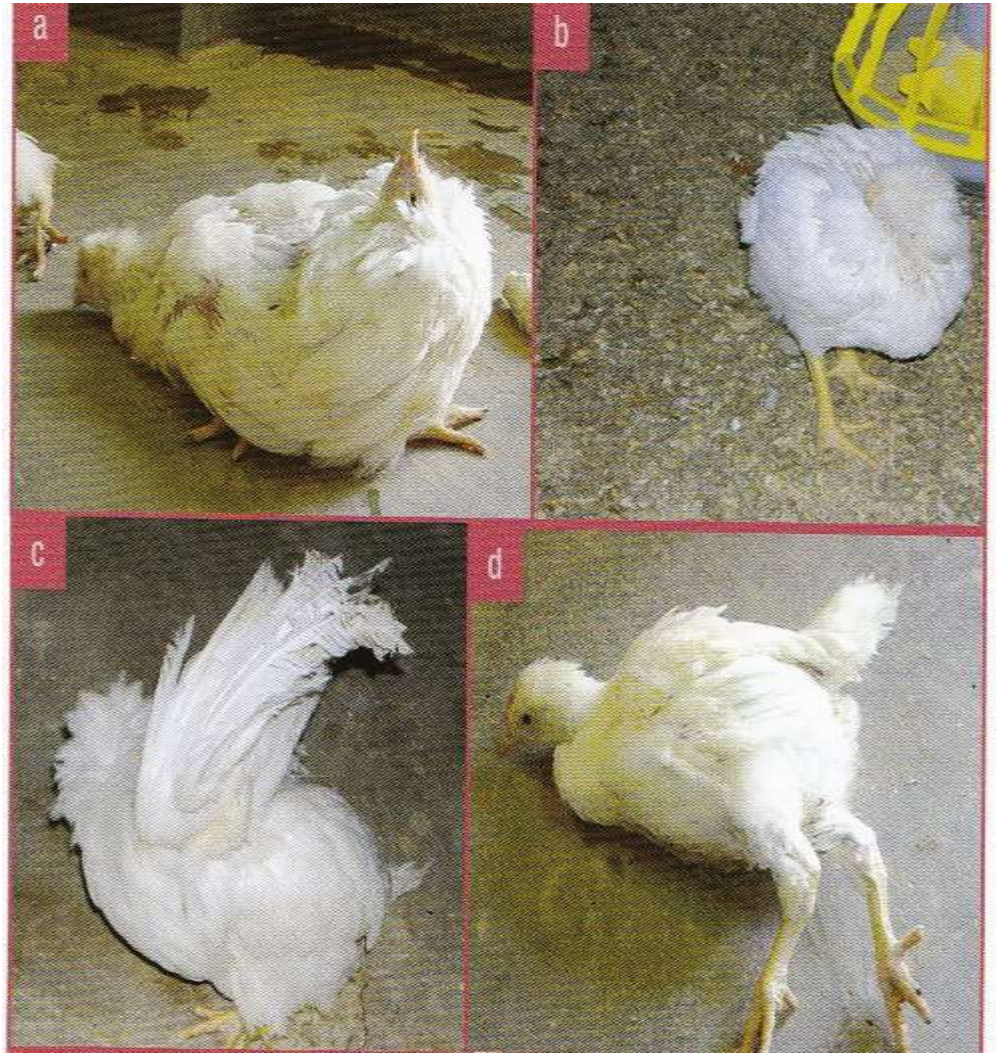


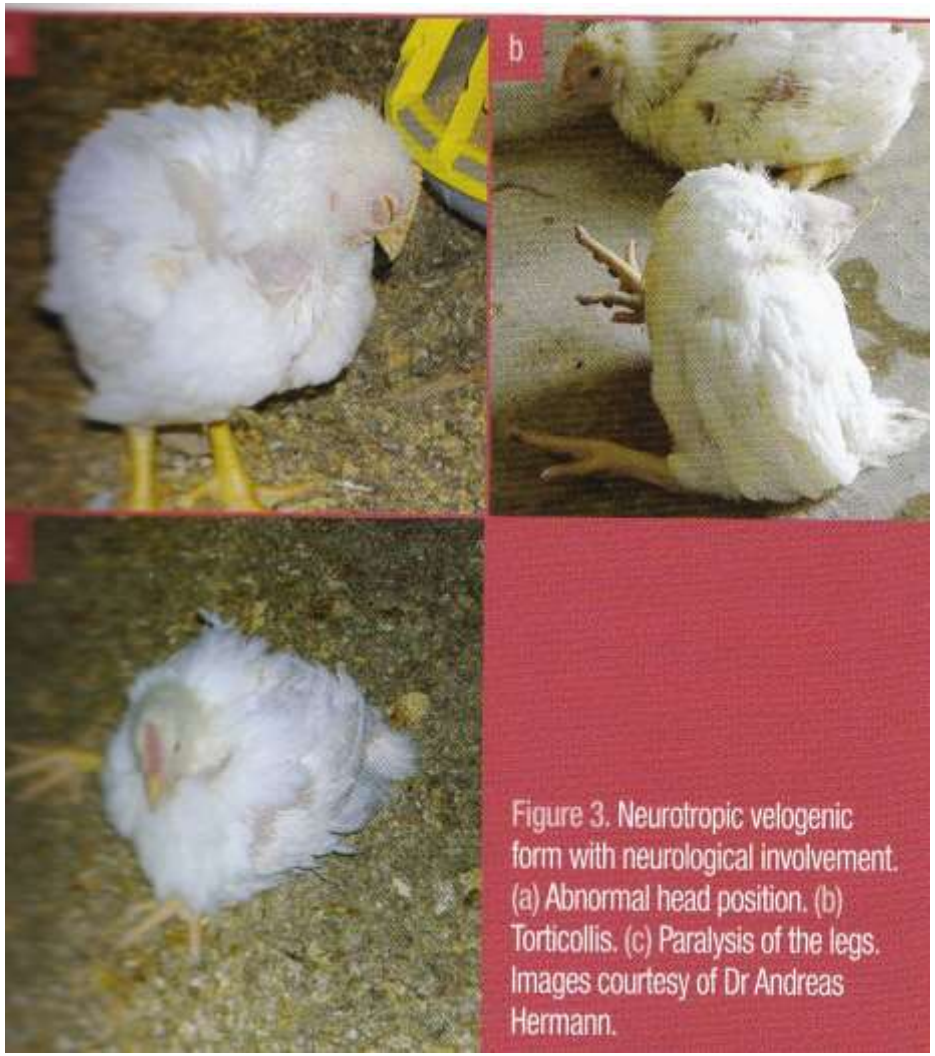
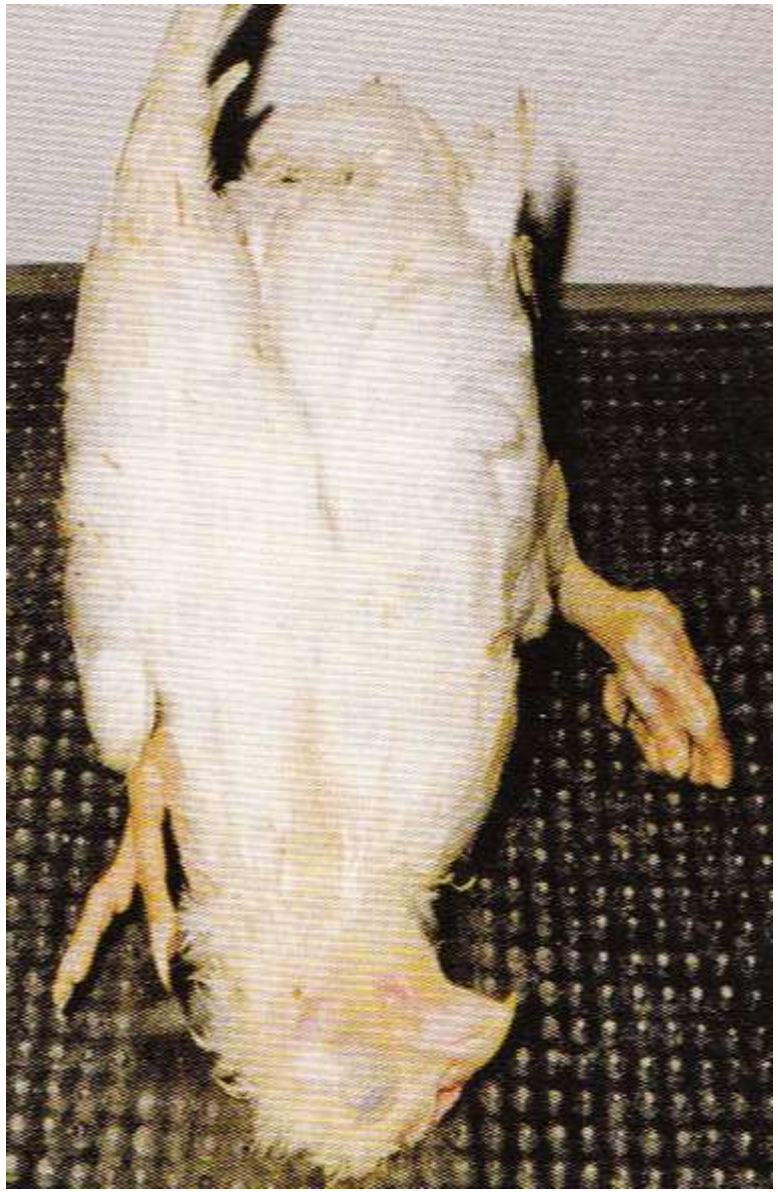




Figure 3. Neurotropic velogenic form with neurological involvement. (a) Abnormal head position. (b) Torticollis. (c) Paralysis of the legs. Images courtesy of Dr Andreas Hermann.



# Immunity & vaccines

4

# Immunity to N.D

Local Ab appear 3 days post vaccination by live vaccines

Circulating ab begin to develop 5-7 days ,reach peak after 3-4 weeks.

Antibody response to inactivated vaccines begin 10-12 days , peak 4-6 weeks.

# Immunity to N.D

Antibody develop 6-10 days post-infection, reach highest level 3-4 weeks.

Titers of MA ,excess 1/8 HI interfere with primary vaccination.

Secondary response to boost vaccination will be greater ,faster than the primary response.

# Vaccination against ND

In Egypt, both vaccinated and unvaccinated birds are affected by ND outbreaks

The most widely used ND vaccines belong to genotype I, II, while the most circulated genotypes are: G VII in the Middle East, Asia, and Africa

Conventional vaccines protect against mortality and morbidity but not against shedding or egg losses due to low similarity (87-89%) of the major protective antigen between current vaccines and prevalent strains

# Vaccination against ND

Inactivated or recombinant G.VII vaccines provide superior protection against the clinical disease, production losses, and viral shedding

# Types of Vaccines

Vector vaccine

Live vaccine

Inactivated vaccine

# Types of Vaccines

Live recombinant GVII provides better protection against field isolates in chicken rather than Lasota

Inactivated vaccines are more effective in chickens that have previously received a live vaccine

Lasota based inactivated vaccine produces lower HI titers against heterogenous virus than against homologous antigen


THANK
YOU!