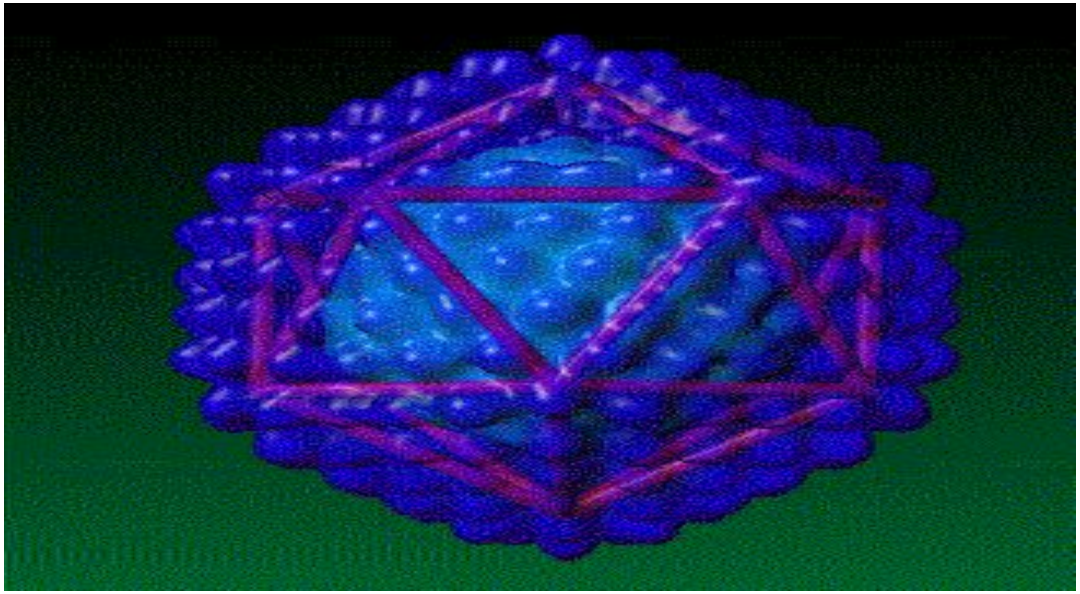
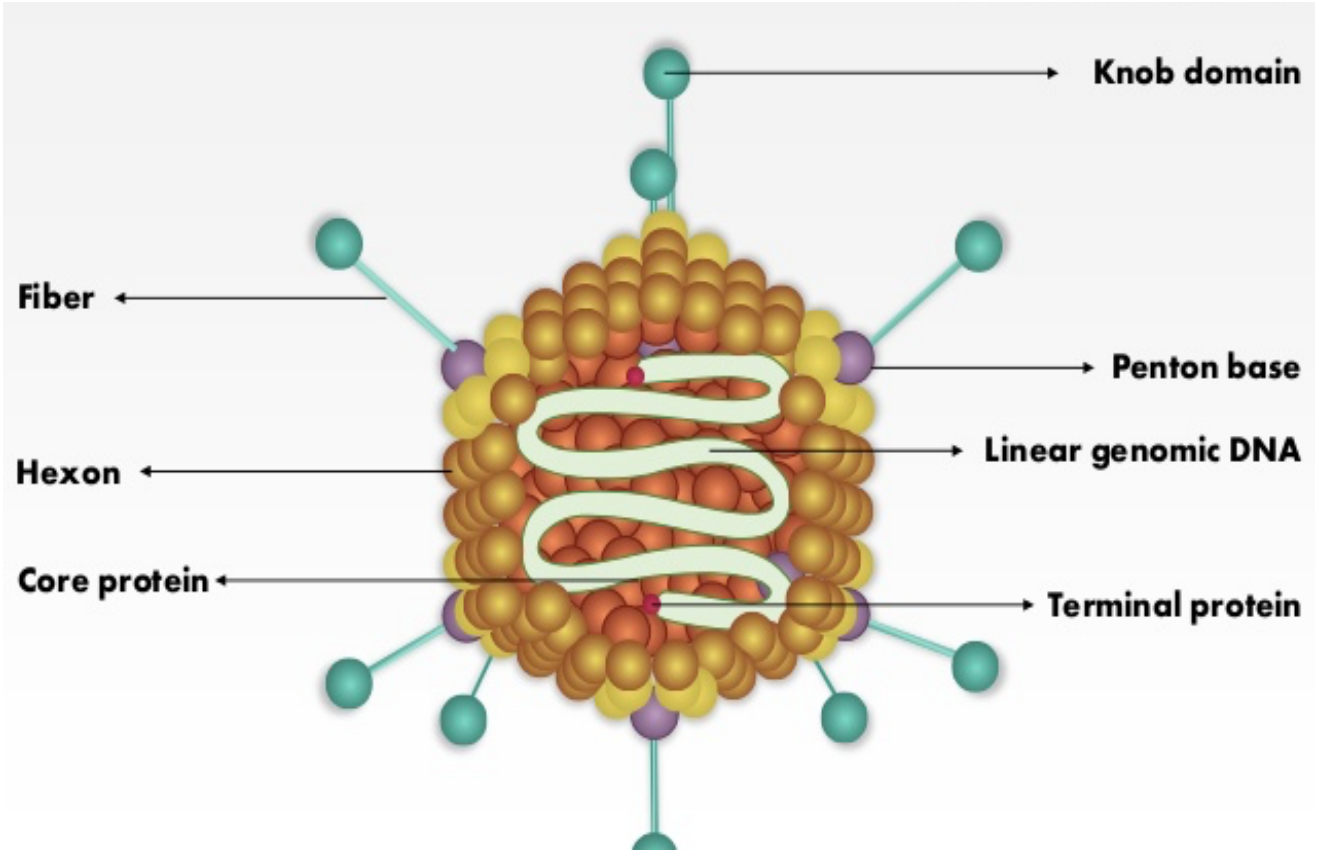
# ADENOVIRIDAE



Dr. Rakhi Gangil

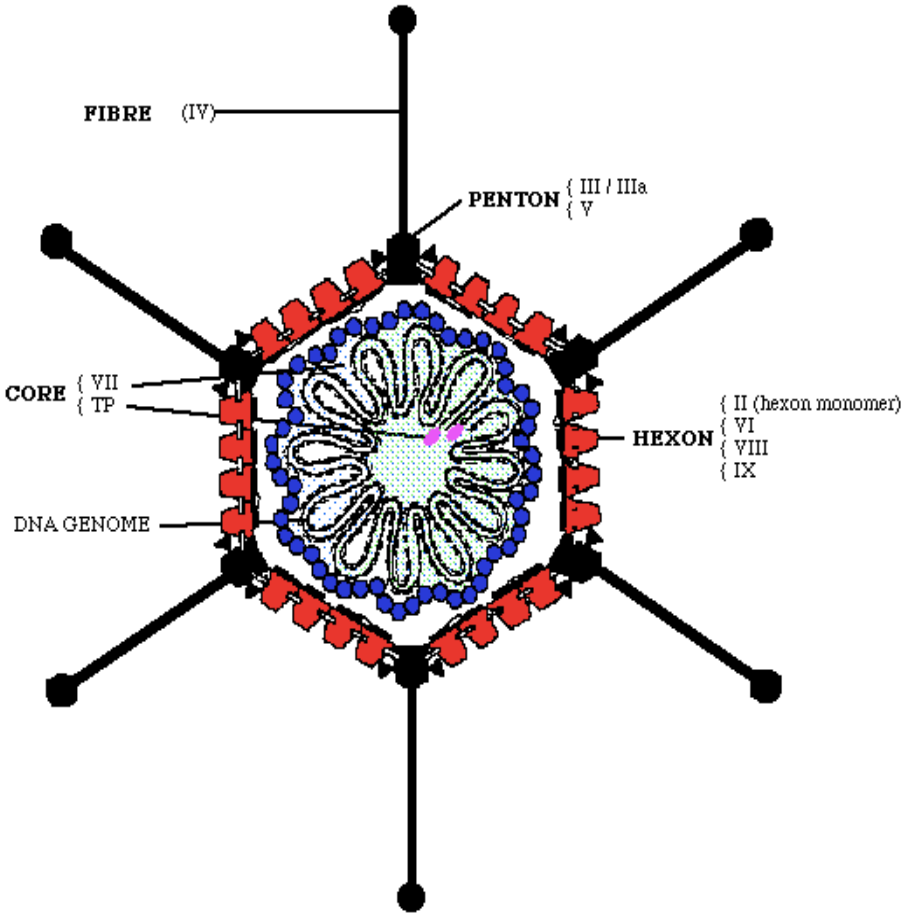
- Adenoviruses are DNA viruses first isolated from adenoidal tissue in 1953
- This family consists of double-stranded DNA viruses with an icosahedral nucleocapsid.
- They have been recovered from many mammalian and avian species.
- Many are found in the respiratory tract and infections are often persistent. Only a small number cause significant veterinary diseases.

# ADENOVIRUS


Knob domain
Fiber
Penton base
Hexon
Linear genomic DNA
Core protein
Terminal protein

# ADENOVIRUS - STRUCTURE

- Non-enveloped DNA virus
- 80-100 nm in size
- Single Linear ds DNA genome with core proteins



# ADENOVIRUS - ULTRA STRUCTURE

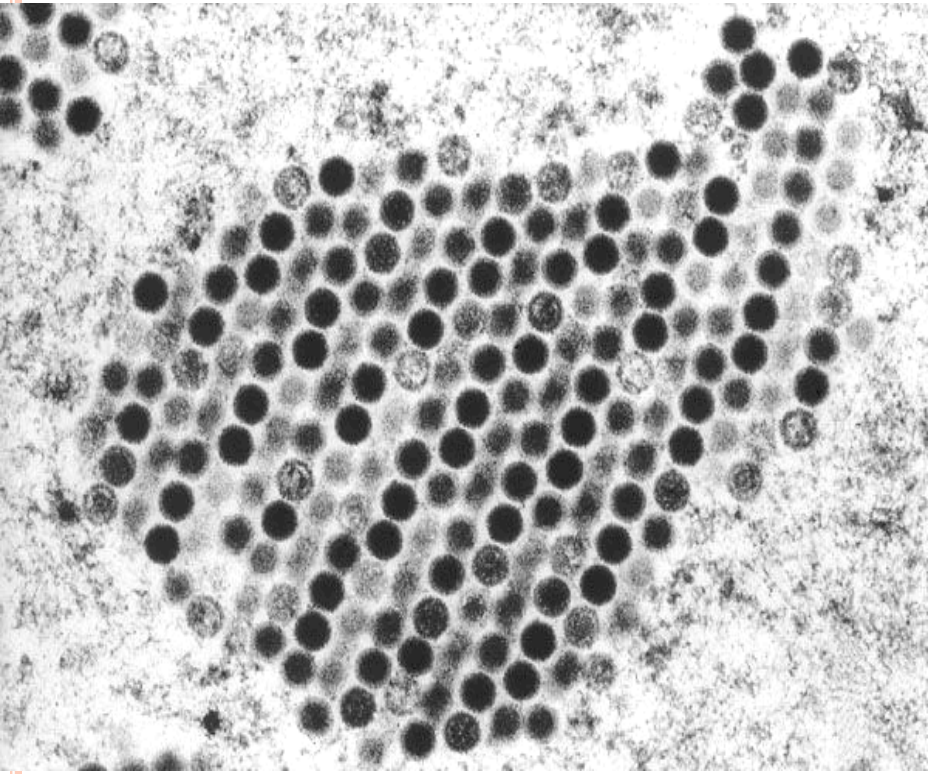
- Icosahedral capsid with 252 capsomere (12 pentons at vertices and 240 hexons)
- Capsid comprised of three surface coat proteins :- fibers, penton, hexons.
- These are the only viruses with a fiber (the fiber antigen) protruding from each of the 12 pentons .
- Fibre helps the virus in the attachment to the host cells it is also a type specific hemagglutinin.
- Adenovirus from avian origin have furgative penton fibre

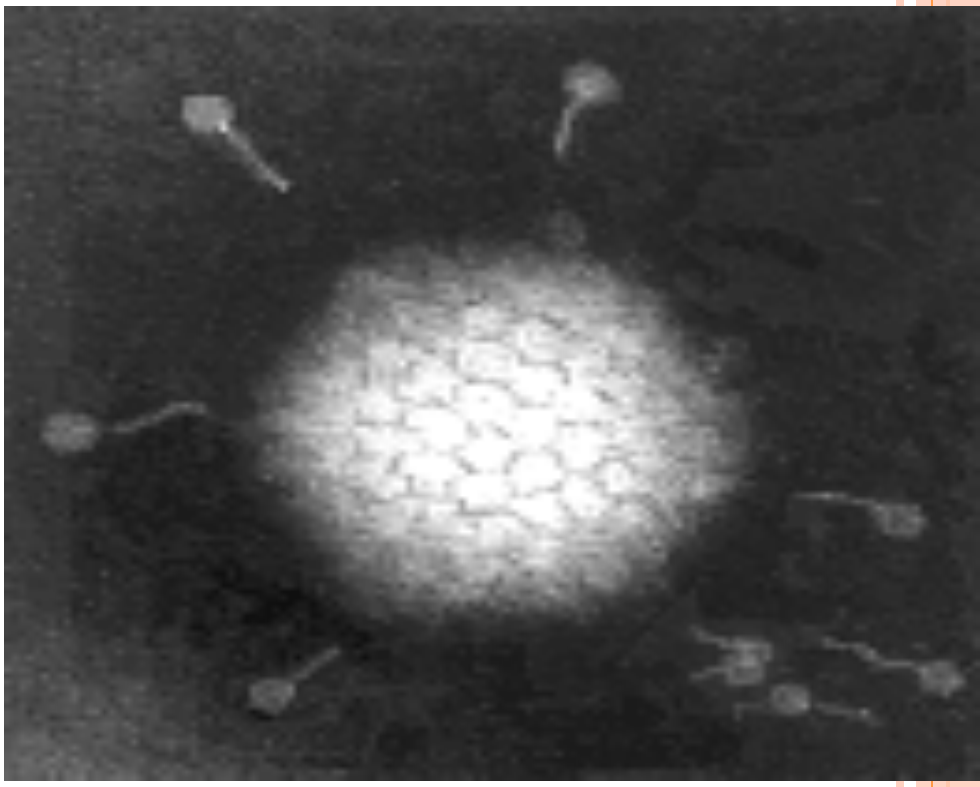
- The hexon of mammalian adenoviruses contains a cross-reacting group antigen.
- The fiber antigen attaches to a specific cell receptor and initiates replication.
- The dsDNA encodes approximately 30 proteins. Viral DNA replication, mRNA transcription and virion assembly occur in the nucleus, utilizing both host and virus-encoded factors. This results in the formation of basophilic and / or acidophilic intranuclear inclusions.

- Many adenoviruses agglutinate red cells of various animal species and some are capable of malignant transformation in tissue culture cell and oncogenesis when inoculated into laboratory animals.
- They are resistant to trypsin and lipid solvents, and moderately resistant on premises.

# ADENOVIRUS

## ELECTRON MICROSCOPIC APPEARANCE





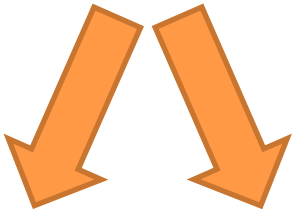
# Adenovirus- Properties

- Stable in the environment
- Relatively resistant to disinfection (Alcohol, chlorhexidine, detergents)
- Stable in GI tract- can withstand low pH, bile acids and proteolytic enzymes

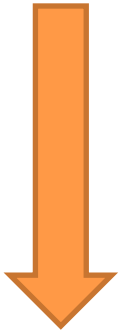
# ADENOVIRUS

**Family** ***Adenovirida***

**Two Genra**



**genus *Mastadenovirus*** **genus *Aviadenovirus***



***Cause disease in mammals*** ***Cause disease in birds***

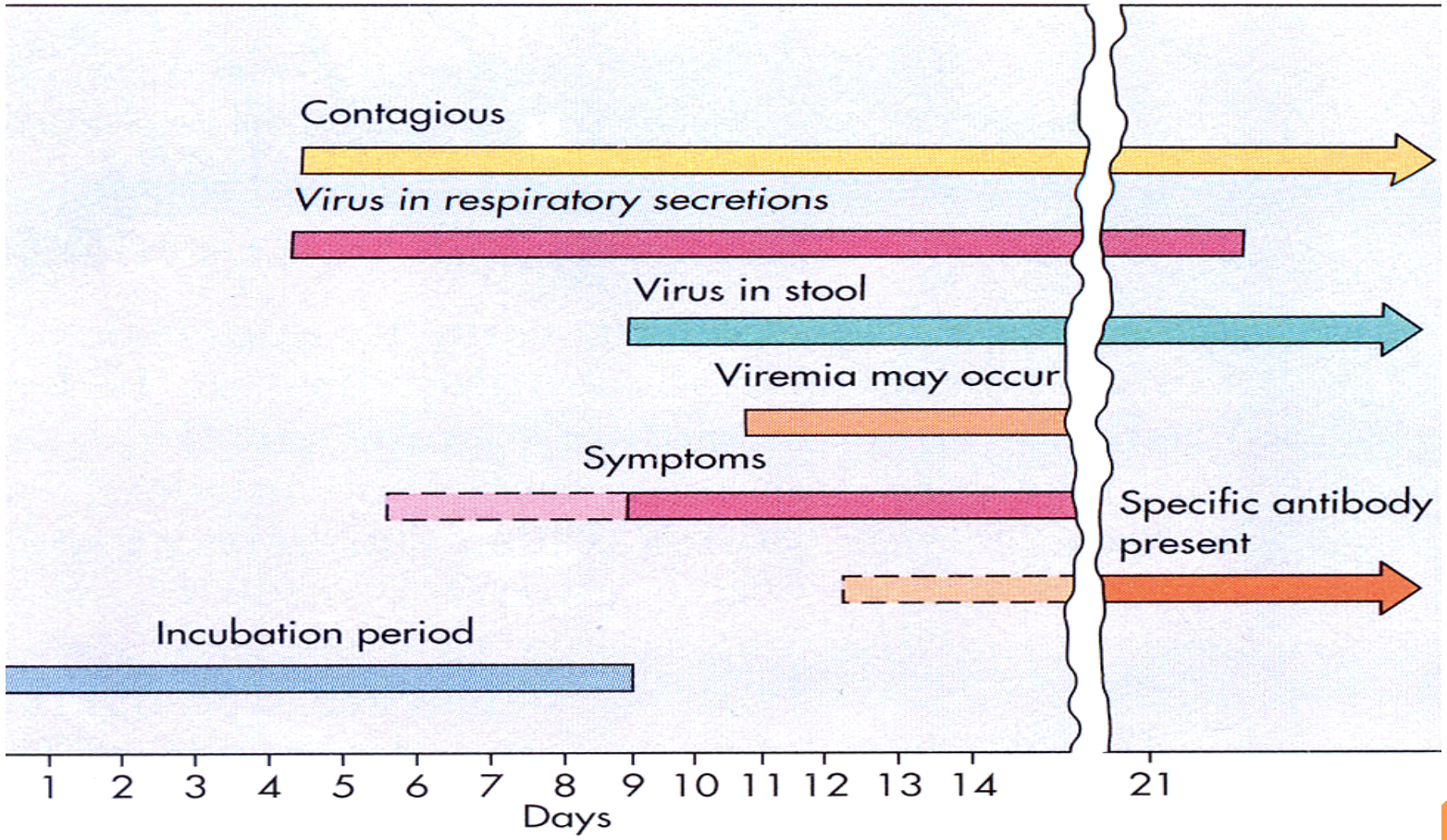
# TRANSMISSION

- Droplets
- Fecal-oral route
- Direct and through poorly chlorinated water
- Fomites

# Transmission

- Disease have a prolonged infective period
- Intermittent and prolonged rectal shedding
- Stable in the environment
- Incubation period- 2-14 days
- Infective period continues for weeks
- Secondary attack rate within families up to 50%

# Timecourse - Respiratory infection



# Pathogenesis and Replication

**Infects mucoepithelial cells of respiratory, GI and GU tracts**

**Enter via epithelium, replicate and spread to lymphoid tissue**

**Viremia occurs**

**Secondary involvement of viscera**

# Pathogenesis and Replication (contd.)

Fiber protein determines target cell specificity and attachment

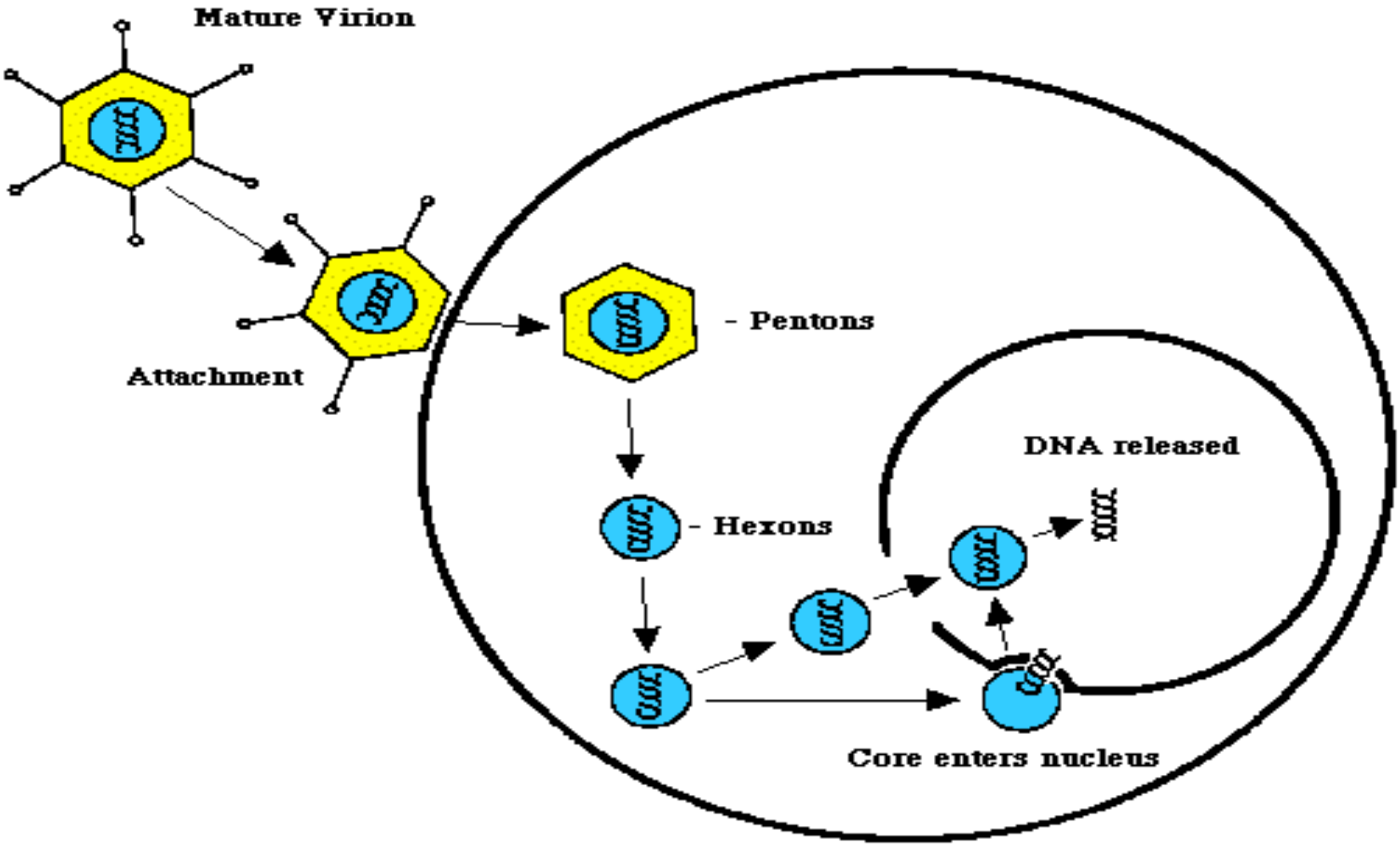
↓

Viral DNA enters host cell nucleus

↓

then formation of basophilic and / or acidophilic intranuclear inclusions occur.

# ADENOVIRUS- REPLICATION



# REPLICATION (CONTD.)

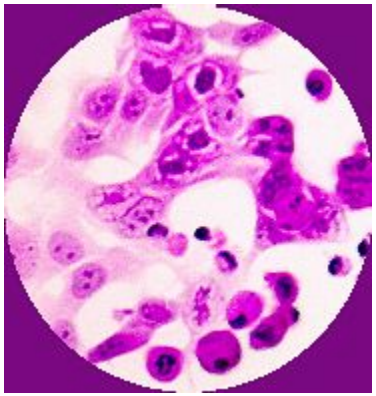
**Adenovirus have a:-**

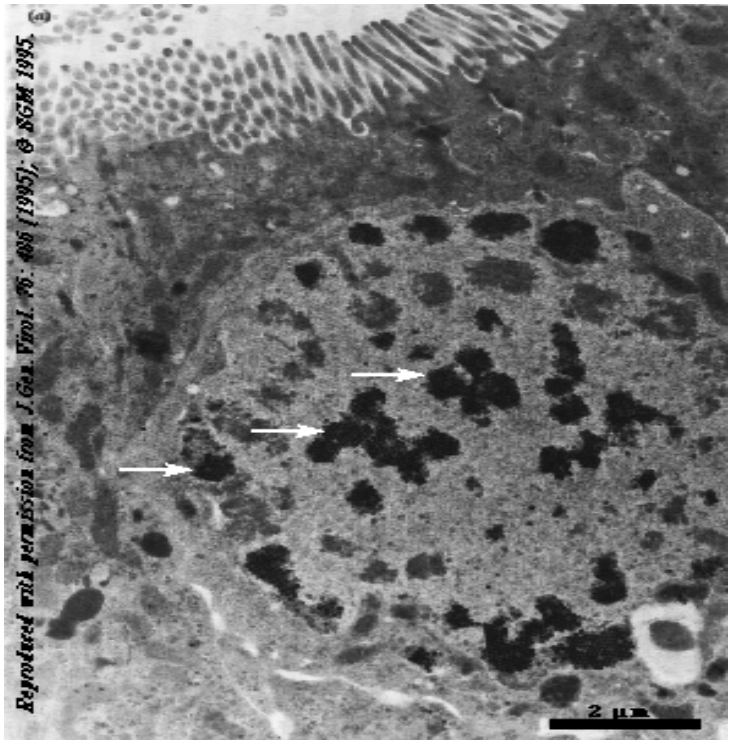
**Early and late phases of replication**

**Error-prone process**

**Inclusion bodies in nucleus**

# ADENOVIRAL INCLUSION BODIES





# Types of infection

- ***Lytic***
  - **Results in cell death; seen in mucoepithelical cells**
- ***Latent/occult***
  - **Virus remains in host cell; seen in lymphoid tissue.**
- ***Oncogenic Transformation***
  - **Uncontrolled cell growth and replication occur; seen with Group A viruses in hamsters**

**Table 62.1.** Diseases of Domestic Animals Caused by Adenoviruses

| Virus | Type of Disease |
|---|---|
| *Mastadenovirus* | |
| Bovine adenoviruses Types 1–10 | Conjunctivitis, pneumonia, diarrhea, polyarthritis |
| Canine adenovirus Type 1 (CAV-1; infectious canine hepatitis) | Hemorrhagic and hepatic |
| Canine adenovirus Type 2 | Respiratory |
| Equine adenovirus Types 1–2 | Pneumonia |
| Ovine adenoviruses Types 1–6 | Respiratory and enteric |
| Porcine adenoviruses Types 1–4 | Diarrhea or meningoencephalitis, or both |
| *Aviadenovirus* | |
| Chicken adenoviruses Types 1–12 | Respiratory disease, enteric disease, egg-drop syndrome, aplastic anemia, atrophy of the bursa of Fabricius |
| Turkey adenoviruses Types 1–4 | Respiratory disease, enteritis, marble spleen disease |

# Infectious Canine Hepatitis(BLUE EYE)

S.host:-Dogs,foxes,wolves,younger animals are more susceptible due to less maturation of immune system

Cause :-by two serotype

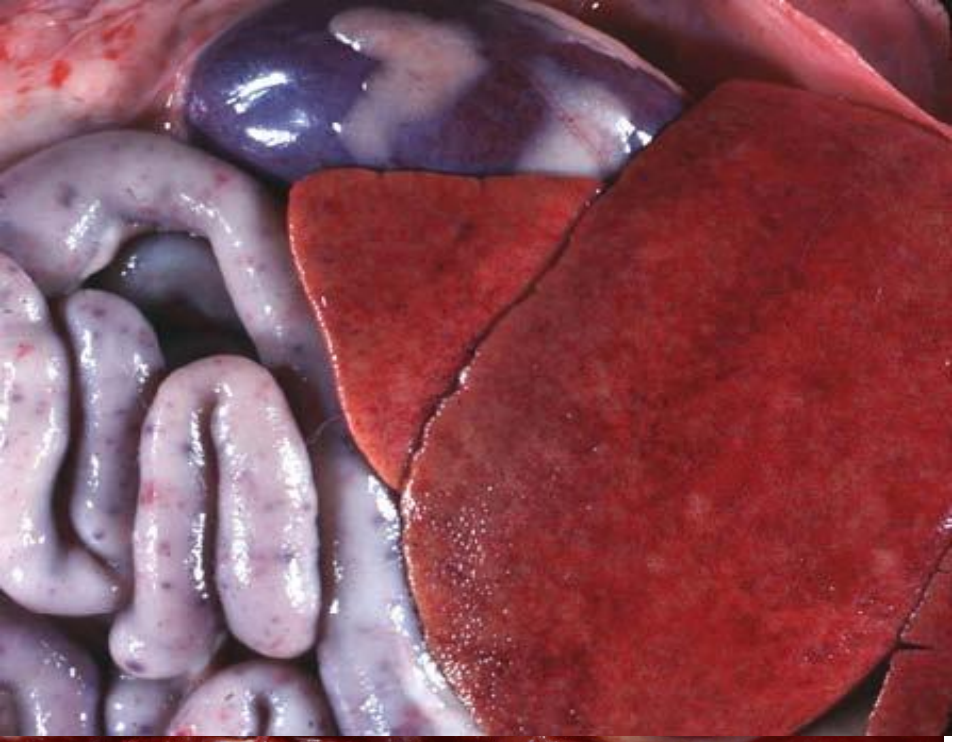
| Canine adenovirus-1 | Canine adenovirus-2 |
| --- | --- |
| causing acute hepatitis, respiratory or ocular disease, encephalopathy, chronic hepatitis, and interstitial nephritis | causes respiratory disease: tonsillitis, pharyngitis, tracheitis, bronchitis, and bronchopneumonia |

## Transmission

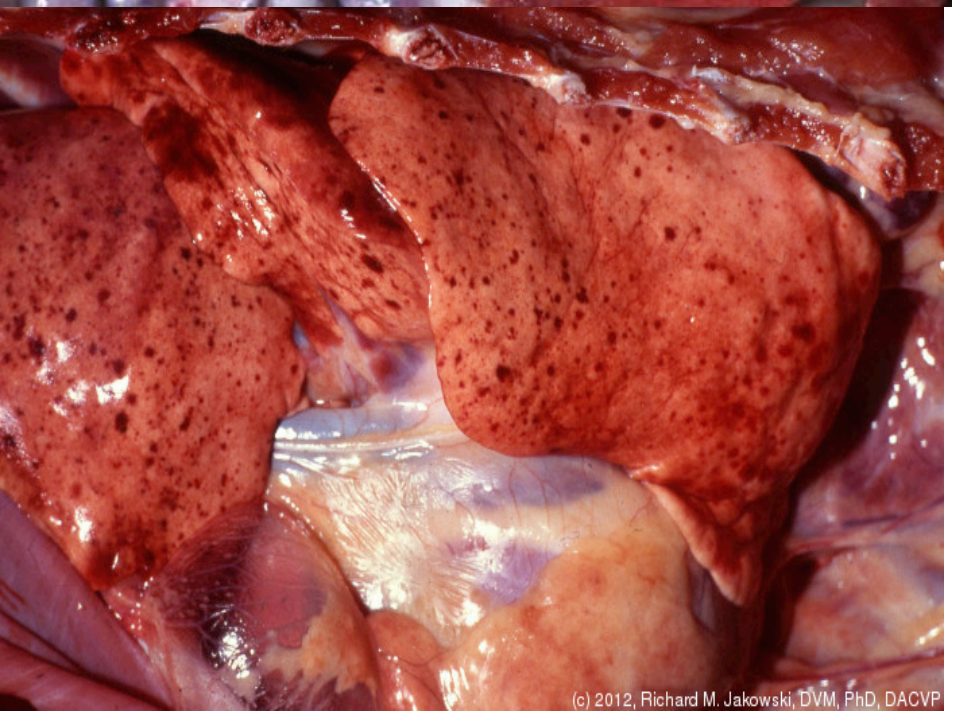
- Infection is by inhalation and ingestion. Spread is by direct and indirect contact.

## Pathogenesis

- The virus replicates initially in tonsils and Peyer's patches producing a viremia with secondary localization and replication in the liver and kidney.

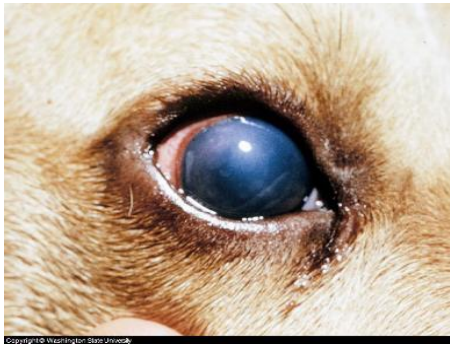


infectious canine hepatitis - pale areas of necrosis in liver, hemorrhages on other tissues

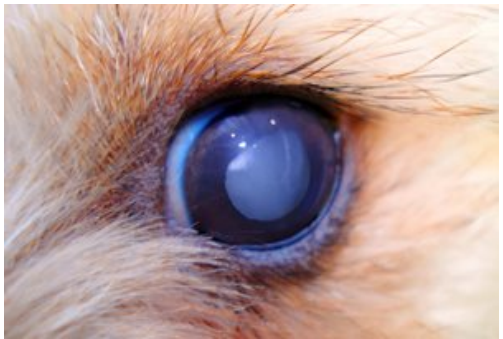



Canine lungs multifocal hemorrhage (Infectious Canine Hepatitis)

- Circulating immune complexes in the glomeruli may result in glomerulonephritis.
- Recovered dogs may develop a transient corneal opacity ("blue eye") as a result of local immune complex deposition
- Recovery from infectious canine hepatitis (ICH) results in lasting immunity.



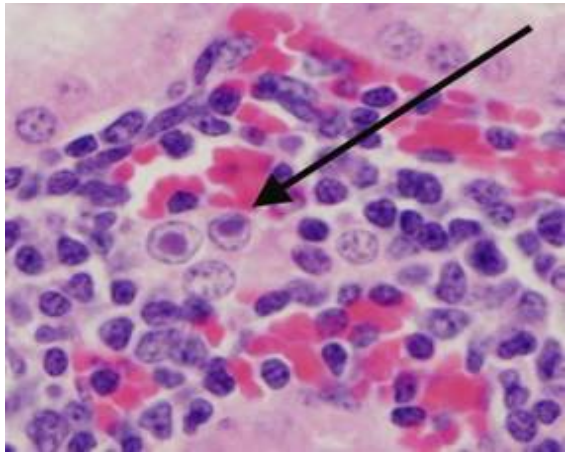
BLUE EYE



# DIAGNOSIS

- Sample collection :-
  - Urine, faces, saliva, ocular and nasal discharge in case of live animals
  - Spleen,lungs,lymph node,kidney in case of dead animals
- Virus Cultivation:-

Sample cultivated in cell cultures of canine origin(MDCK), result into the presence of basophilic intranuclear inclusion bodies in hepatocytes, endothelial cells, and Kupffer cells



Presence of intranuclear inclusion bodies

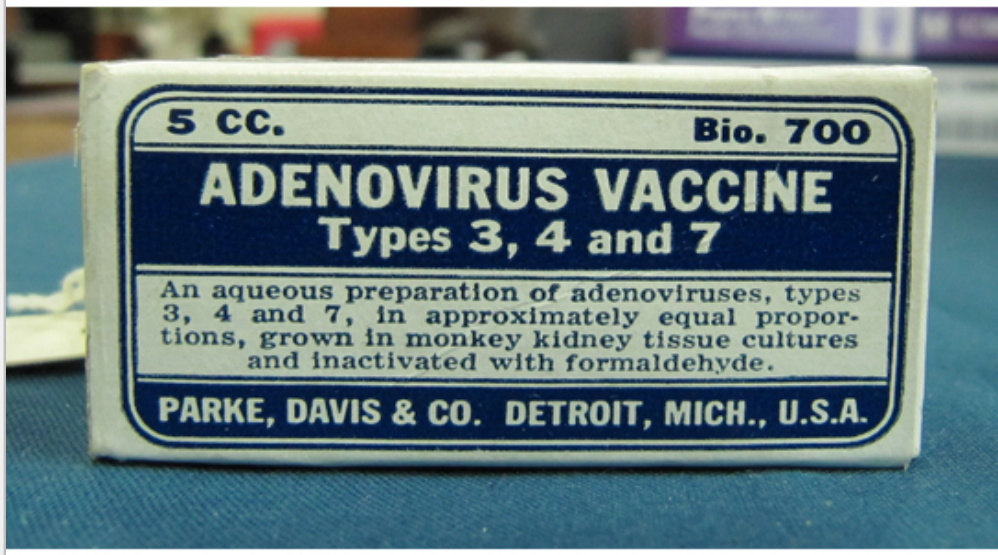
- Serology:- ELISA test
  VNT test
  HI test
- Molecular technique:- PCR-

Prevention :-

1)Live vaccine :->6weeks of animals

2)Inactivated vaccine;-can be safely given at any age .it should be repeated annually


Vaccine against ICH



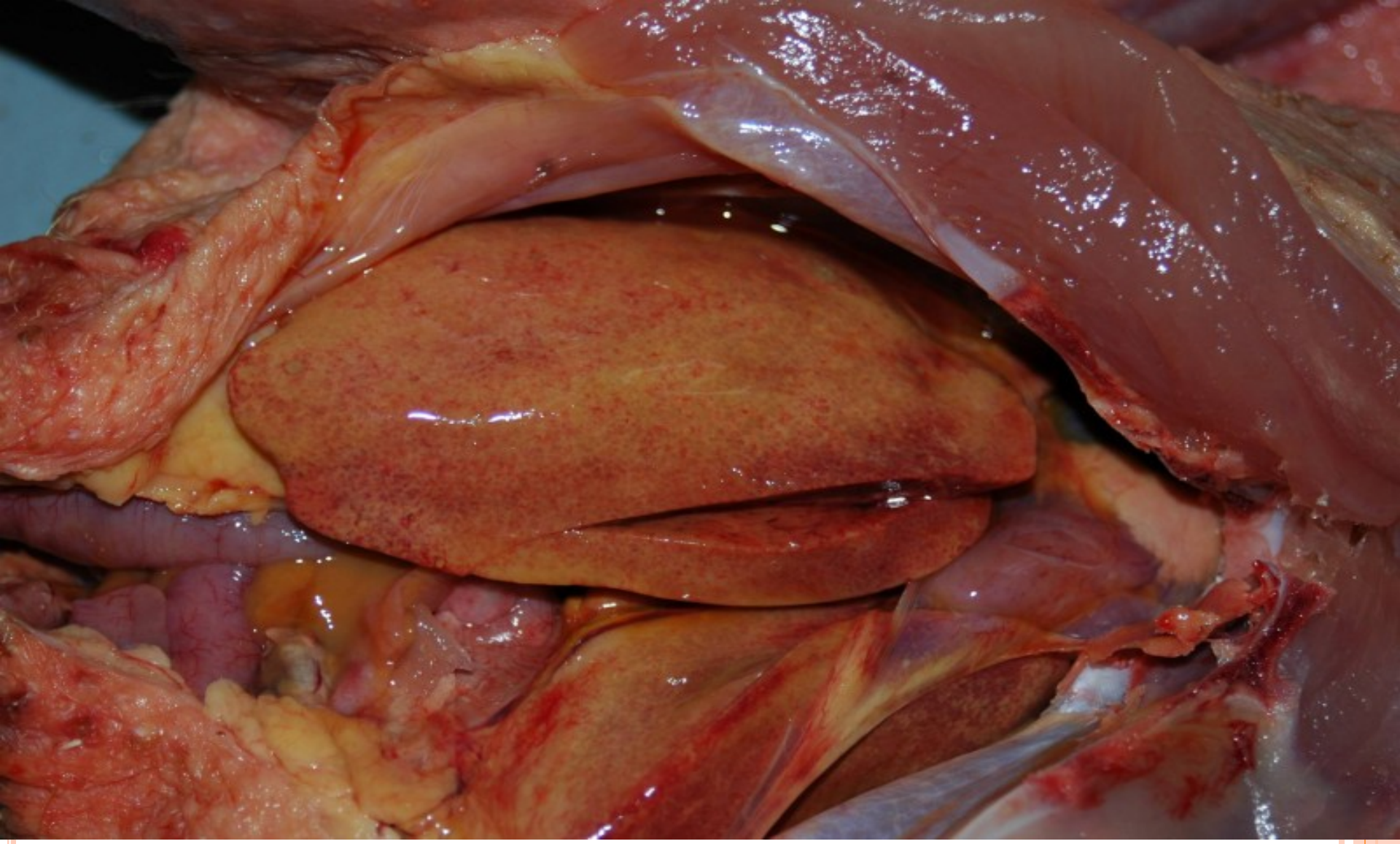
# *AVIADENOVIRUS*

## Inclusion Body Hepatitis

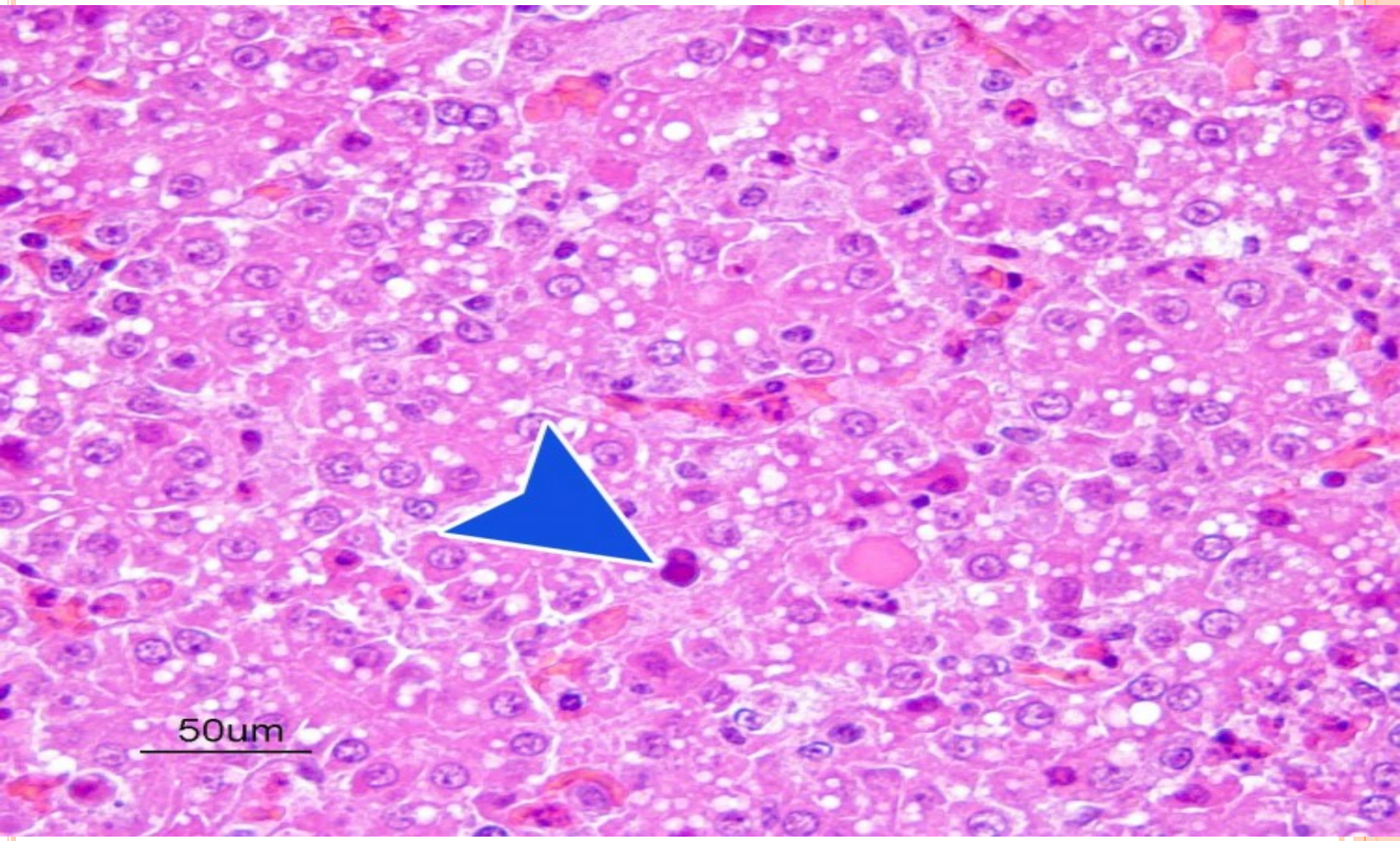
- Cause:-A number of serotypes of aviadenoviruses have been implicated.
- Occurrence:-The disease, which most often affects young chickens, occurs infrequently worldwide.
- Transmission:-Aviadenoviruses are transmitted horizontally and vertically.

**Clinical and Pathological feature**

- Infected chickens may appear anemic and weak but other characteristic clinical signs are absent.
- The liver of affected chickens is enlarged and pale and frequently with hemorrhages throughout.
- Microscopically there is evidence of diffuse hepatitis with intranuclear eosinophilic inclusion bodies in hepatocytes



Pale and enlarged liver with hemorrhages




Hepatocyte with eosinophilic intranuclear inclusion body characteristic of this disease.

# Diagnosis

- Clinical specimens: Sick, live birds are preferred.
- This is usually based on macroscopic liver lesions and microscopic examination of characteristic intranuclear inclusion bodies in hepatocytes.
- The virus can be propagated on the chorioallantoic membrane (CAM) of chicken embryos or in avian kidney cell cultures and identified by the virus neutralization test.

# *ATADENOVIRUS*
## EGG DROP SYNDROME

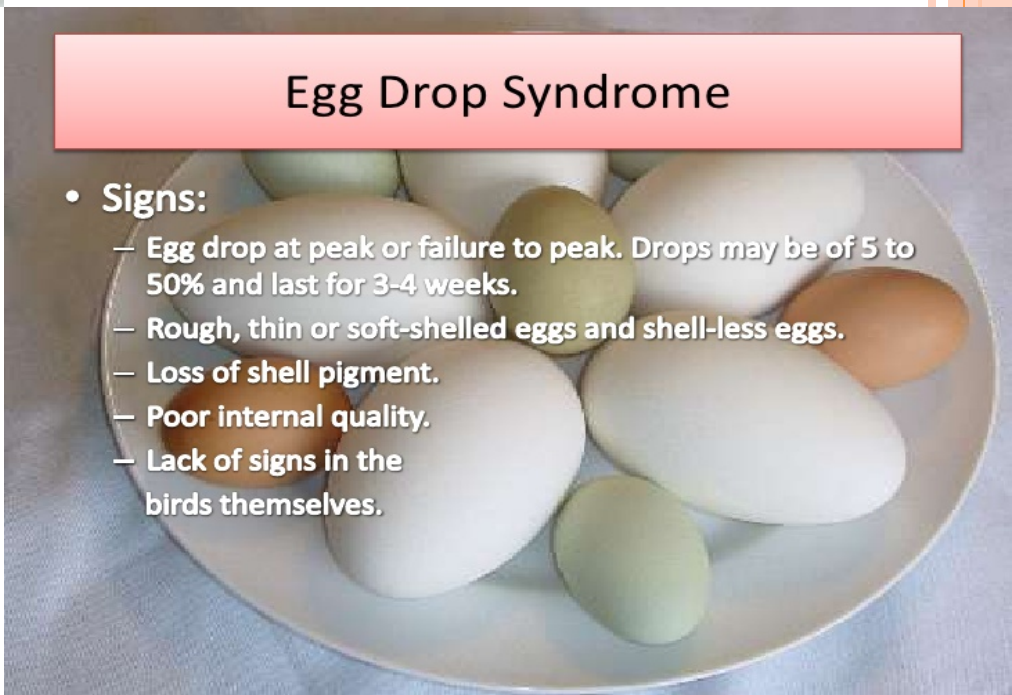
- Cause :-Duck adenovirus 1.
- Occurrence:-Egg drop syndrome (EDS) is common and worldwide in distribution. It occurs most frequently in broiler chicken breeder flocks 5 - 6 weeks of age.
- Transmission:-It is mainly transmitted vertically through the egg. The virus is shed in the feces and spread can be by contaminated water and fomites.

## Clinical & Pathological Features:

- Infected birds appear healthy. Egg production is variably depressed and abnormal eggs are produced. Shells of the latter may be absent, thin, underpigmented and rough surfaced. Outbreaks last 4 to 10 weeks.
- Among the effects noted are inactive ovaries, atrophied oviducts, edema of the uterus, exudates in the cell gland and intranuclear inclusions in tissue of the cell gland.

# Thin shell egg

## Abnormal eggs



# DIAGNOSIS

- Loss of egg production with abnormal eggshells suggest EDS.
- Clinical specimens: eggs and reproductive tissues including the shell gland.
- Cultivation of Virus : Duck and goose embryonated eggs (preferred) and also in duck kidney or fibroblast cell lines.
- The presence of virus is indicated by hemagglutination of avian red cells (HA test).
- Diagnosis is made with the hemagglutination inhibition test (HI test). It is used to screen flocks but negative tests do not indicate that birds are necessarily free of infection.

# PREVENTION

- Replacement birds should be from uninfected flocks.
- An oil adjuvant inactivated vaccine provides immunity for a year.
- Good hygiene to prevent lateral spread particularly from infected egg contamination

# Hydropericardium Syndrome

- Also known as “Hydropericardium–hepatitis syndrome” “leechi disease” or “angara disease”
- The disease occurs usually in 3-5 week-old healthy broilers .
- Characterized by high morbidity and mortality, excess accumulation of fluid under pericardium and many areas of necrotic foci in the liver

# Etiology

- Fowl adenovirus -4 ( mostly serotype 4 & 8)
- These viruses are capable of producing the disease without the immunosuppressive effects of associated viruses such as IBD or other immunosuppressive agents.

# Transmission

- It is transmitted both vertically and horizontally
- Adenovirus may remain latent in breeding stock until the onset of maturity and then are shed following immunosuppression or stress
- Horizontal spread of virus by carriers occurs.
- Fecal contamination of clothes, footwear and equipment including transport crates and vehicles may spread infection.

# Pathogenesis

- Virus enters into the body
- Initial multiplication of the virus occurs in small and large intestines
- Viraemia occurs with spreading of virus to many organs like fever, Kidney, respiratory tract, Bone marrow and bursa
- Virus can be readily isolated from faeces, occular and nasal mucosa, bursa
- Chicken once affected with adenovirus becomes lifelong carrier

## Sign and Symptoms

- Sudden increase in mortality (20-80%): Mortality starts at about 3 weeks and reaches it's peak in 4 to 5 weeks.
- —flocks of 3-5 weeks old broilers with HP may not show specific signs i.e. bird may remain active just until before death, but abrupt onset of mortality with **lethargy, ruffled feather & yellow mucoid dropping** may be seen

# Diagnosis

- Necropsy: gross and microscopic lesion
- Histological investigations and detection of intra-nuclear inclusion bodies in hepatocytes
- Detection of the antigen or virus particles using immunofluorescence test or electron microscopy
- PCR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