

GENERAL AND SYSTEMIC VIROLOGY (MICRO – 303)

Group III: ds RNA viruses:

REOVIRIDAE

BIRNAVIRIDAE

Group I: ds DNA viruses:

ADENOVIRIDAE

Delivered by:

Prof. Dr. Iftikhar Hussain
Chairman

Dept. of Veterinary Microbiology
Faculty of Veterinary Science
University of Agriculture
Faisalabad

Presented by:

Muhammad Sajjad Hussain

REOVIRIDAE

Classification

Genus	Viruses	Segments	Serotypes
Orthoreovirus	Avian orthoreoviruses	10	9
	Mammalian orthoreoviruses	10	3
Orbivirus	Blue tongue virus	10	24
	African horse sickness virus	10	9
	Epizootic hemorrhagic disease virus	10	2
Rotavirus	Human rotavirus	11	-
	Bovine rotavirus	11	-
	Avian rotavirus	11	-
	Rotaviruses of aquatic animals (Aquareovirus – of fish)	11	-
	Insects rotavirus (Polyhedrosis virus) e.g. Cypovirus	11	-
	Plants rotavirus Phytoreovirus, Fijivirus, Oryzavirus	11	-
Coltivirus	Colorado tick fever virus	12	

R E O stands for:
R → Respiratory
E → Enteric
O → Orphan

General Properties

Shape and size:

Naked, capsid presents icosahedral symmetry, measuring diameter 70-80 nm, double shelled. The outer shell is made up of hollow capsomers.

Genome:

10 -12 segmented double stranded RNA

Genome segments are divided into 3 size classes;

- L – *Large* – L1, L2, L3
- M – *Medium* – M1, M2, M3
- S – *Small* – S1, S2, S3, S4

Reassortment of gene segments creates hybrid viruses.

Resistant:

Proteolytic cleavage of the outer capsid (as occur in GI tract) activates the virus for infection and produces an intermediate “infectious subviral particle (ISVP).

Moderately resistant to heat, organic solvents and non – ionic detergents.

Both Orthoreo- and rota- viruses are stable at a wide range of pH but Orbiviruses lose infectivity at low pH.

1. ORTHOREOVIRUS

AVIAN ORTHOREOVIRUS

Associated disease:

Virus cause inapparent infection that may be characterized by viral arthritis /tenosynovitis (rupture of tendon Gastrocnemius muscle) in chickens and turkeys.

Synovial lesions resemble those of caused by *Mycoplasma synoviae* or *Staph aureus*

Virus Conformation:

Orthoreovirus involvement is conformed by virus isolation from specimens like; affected articular cartilage and tendon sheath. Synovial fluid is not reliable.

Transmission: Fecal oral route and *In ovo*

Isolation & Cultivation:

Suspension of tissue can be inoculated into yolk sac of embryonated egg or onto Monolayer of chicken embryo liver cells.

Diagnosis: Virus can be conformed by IF. Serological tests are established to conform immune of birds.

Vaccination: Both live and inactivated vaccines are used in parent flock. There is no heterologous cross protection.

Control: Total depopulation at the end of a production cycle followed by thorough cleansing and disinfection of premises.

MAMMALIAN ORTHOREOVIRUS

Associated disease:

The virus associated with mild enteric and respiratory disease in many species.

Severity of infection depends on secondary infections.

3 serotypes are recognized by SN or HI test.

Transmission: viruses are ubiquitous; found on sewage, river vats and transmission is via ingestion of contaminated food.

Susceptible Hosts:

Found in many mammal species; man, chimpanzee, monkey, pig, cattle, sheep, horses, dogs, cats, rabbits, mice, mink etc.

Pathogenicity: Natural infection occurs in mice (wild & lab colonies) affecting newborns and causing steatorrhoea, jaundice and stunted growth.

Cultivation: Primary or continuous cell line (HELA, VERO)

CPE: eosinophilic cytoplasmic inclusions which accumulate with perinuclear masses; contain numerous viral particles.

Embryonated Eggs: Some strains grow on CAM

Lab animal: Newborn mice I/C with type III → develop fetal encephalitis. Pregnant mice with type I may lead to fetal resorption and intrauterine death.

Haemagglutination:

All 3 types haemagglutinate human O RBCs. Type III agglutinates bovine RBCs.

2. ORBIVIRUS

AFRICAN HORSE SICKNESS VIRUS

Associated disease:

Virus is associated with a seasonal non-contagious infection of equines affects horses, less mules and donkeys are rarely clinically affected.

The disease is characterized by edema of subcutaneous tissues and lungs, hemorrhages of the internal organs and accumulation of serous fluid in the body cavities. In severe cases death occurs from the pulmonary edema, hypothorax and hydropericardium.

Four different clinical forms of illness are as follows:

i) **Very mild form:** frequently overlooked. temperature is 41 C or higher.

ii) **Pulmonary form** (dunkop): Severe dyspnoea, coughing, frothy discharge from nostrils. Most affected horses usually die.

iii) **Cardiac form** (dikkop): Remarkable swelling of the head, neck and brisket. Many affected horses usually recover.

iv) **Mixed form:** Probably combination of cardiac and pulmonary forms and is often not diagnosed during life.

Mortality over 90% in exotic horses and 20% in indigenous horses.

Cultivation: Embryonated egg: Yolk sac

Cell culture: BHK – 21 and VERO cell lines. CPE: Rounding and shrinkage of cell. Acridin orange staining shows large perinuclear inclusions. Mosquitoes cell lines. Lab animals: Unweaned mice I/C → Death 5-15 days post infection. Ferrets I/V → febrile reaction 4-7 days pi.

- Vaccines:**
- Polyvalent vaccines are used.
 - Freeze dried live neurotropic mouse brain (non-pathogenic for horses) → horses show a slight rise in temperature, give protection upto 1 year.
 - Cell culture vaccine:

BLUE TONGUE VIRUS

(Sore mouth or ovine catarrhal fever disease virus)

Associated disease:

Virus is associated with a serious disease affecting sheep especially lambs with fever, erosions, crushing and cyanosis around the mouth, oedema of head, brisket and neck, sometimes pulmonary edema and lameness due to involvement of hooves (coronary band and lamellae) and muscle damage.

Pregnant ewe frequently abort. Cattle, goat and camel act as symptomless carriers. Transmission is carried out by *Collicoides* spp. of mosquitoes.

Mortality may be up to 90%. Spermatozoa from carrier bulls may show abnormalities and contain virus particles.

Cultivation: *Embryonated egg:* 6 days old – CAM at 33.5 C → Death and extensive hemorrhages of the developing embryo

Cell culture: BHK – 21 ; HELA cell lines. Calf kidney or testis cells. CPE: Dense acidophilic Intracytoplasmic inclusions may be seen.

Lab animals: Unweaned mice I/C → produces encephalitis followed by death in 3 – 7 days post infection.

Haemagglutination:

None

Immunity: Because of many serotypes, duration and degree of immunity can not be ascertained (determined).

Vaccines: Univalent & polyvalent *avirulent* (attenuated in chick embryo) cell culture vaccines.

Live vaccines should not be used in pregnant ewes because cerebral and other lesions may occur in lambs born from the vaccinated ewes. Vaccine may also interfere with estrus.

Sheep should be vaccinated 3 weeks before service and before rainy season. Maternal antibodies via colostrum protects lambs 3-6 weeks months and they should not be vaccinated before 3 months.

EPIZOOTIC HEMORRHAGIC DISEASE (EHD) VIRUS

Associated disease:

Severe epizootics in white tailed deer in North America but subclinical infection may occur in cattle... Animals are shocked and die in coma. Postmortem examination is similar to blue tongue; extensive hemorrhages, edema and serous effusion. Transmission via *Culicoides* spp. of mosquitoes. 8 serotypes have recognized.

Cultivation: Lab animals: new born mice I/C

Cell culture: Primary embryo cells. VERO cell lines. CPE: Cytoplasmic inclusion and plaques formation.

IBARAKI VIRUS

Associated disease:

Similar to Epizootic hemorrhagic disease virus (EHDV). Acute febrile disease of cattle, similar to blue tongue. Probably arthropod-borne infection present in south east Asia.

EQUINE ENCEPHALOSIS VIRUS

Associated disease:

Infection is usually subclinical. Sporadic case of acute fetal disease occurs. Cerebral oedema, fatty liver and enteritis. 5 serotypes.

PALYAM VIRUS

Associated disease:

Arthropod-borne disease of cattle. Associated with abortion and teratogenic effects. Reported in south Africa, South east Asia and Australia.

3. ROTAVIRUS

General Properties

Double stranded RNA genome of the viruses contains 11 segments and each code for one protein. Out of the 11 proteins, six are structural and five are non-structural proteins. The VP4 and VP7 are the most important biologically. The VP4 protein also has the haemagglutination properties.

High titre of rotavirus excreted in feces (10^9 per gram of feces). Virus stable in the environment and buildings remain contaminated for longer period. Adults may become persistently carrier.

Transmission: Fecal oral

Pathogenesis: Upon ingestion → enters absorptive columnar epithelium of alimentary tract – lining apical half of intestinal villi → Thus, absorptive apical are destroyed → diarrhea

HUMAN ROTAVIRUS

Associated disease:

Virus is associated with gastro-enteritis in man. Disease affects mainly children during first 5 years.

Cultivation: Cell culture: Monkey cell culture

BOVINE ROTAVIRUS

Associated disease:

Disease affects young calves (3 week of age) and may cause diarrhea. Recovery in 3-4 days but there are fetal cases.

Passive lactogens protection of newborns.

ROTAVIRUS OF OTHER ANIMALS

In addition above, rotaviruses have been demonstrated in monkeys, sheep, goats, deer, gazelle, rabbits, dog and cats.

AVIAN ROTAVIRUS

Associated with enteritis in chicken and turkeys.

ROTAVIRUS OF AQUATIC ANIMALS

Viruses isolated from fish hosts chum, salmon, channel fish and oysters are classified in a new genus of the family.

4. COLTIVIRUS (12 segments of ds RNA genome)

COLORADO TICK FEVER VIRUS

Associated disease:

Human disease caused by the infected tick. Affected persons develop fever with chill, aches in head and limbs often vomiting. Encephalitis may occur especially in

children. Rodents act as reservoirs. Primarily disease of human, endemic in USA and Canada. (Zoonotic importance)

Cultivation: Embryonated egg: Yolk sac
Human cell lines

BIRNAVIRIDAE

Classification

Genus	Viruses	Serotypes
Avibirnavirus	Infectious bursal disease virus	2 serotypes
Aquabirnavirus	Infectious pancreatic necrosis virus	3 serotypes
Entomovirus	Infects insects /arthropods	---

General Properties

Shape and size:

Round slightly polygonal outlined cubical naked virion. 60-85 nm in diameter with single shelled capsid. Resistant to high temperature, acidic pH, chloroform and other disinfectants – remain in premises for 120 days after disease outbreak.

Nucleocapsid: Genome is bisegmented double stranded RNA (2 seg. ds RNA).

The small segment encodes a genome-linked protein (VPg) and the large segment encodes structural and non structural proteins.

Virion contains 4 structural polypeptides; VP1, VP2, VP3 and VP4. VP2 is the major capsid protein-derived from a precursor VPx.

1. AVIBIRNAVIRUS

INFECTIOUS BURSAL DISEASE (IBD) VIRUS

Associated disease:

Virus causes a disease mainly of chicken but natural infection has also been observed in turkeys, ducks and in pheasants. Chick (2-7 week of age) are most frequently affected. Bursa of Fabricius is enlarged. Mortality – 30 % and Morbidity – 100%. Concurrent infection with organism aggravated the disease.

In postmortem examination: hemorrhages of muscles. Bursa is the primary target organ of virus. There are necrotic foci, echymosis in the mucosal surface lining bursa. Spleen enlarge with foci and hemorrhages at proventriculus and gizzard. There may be jaundice – liver bronze colored.

Transmission: Ingestion

Pathogenesis:

Virus has special affinity for Bursa of Fabricius → B-lymphocytes – lymphoid follicles (IgM bearing) → Cytolysis → Immune suppression

Humeral immune responses markedly depressed and may also cause suppression of cell mediated immunity (CMI).

Consequently: there is degeneration of bursa, infectious bursitis, Regressed bursa + hemorrhages in breast and thigh muscles.

Infection also has capability to lessen vaccination response of ND and HPS.

Cultivation: Embryonated egg: CAM → s/c edema, hemorrhages, dwarfing, liver necrosis and ultimately there may be death of embryo.

Cell culture: chick embryo kidney cell cultures. After 2-4 days CPE: perinuclear inclusions.

Lab animals: Newborn mice (Unweaned) → pruritis, tremor and locomotor ataxia.

- Immunity:** Both live and killed vaccines.
Inactivated oil emulsion vaccines
Passively acquired immunity can interfere an active immune response.
- Diagnosis:** ID (immuno-diffusion) IF (immuno-fluorescence), ELISA
Two serotypes can be distinguished by NT and electrophoresis of viral RNA and Proteins and may characterize as;
- i) Pathogenic subtype – infectious IBD in chickens
 - ii) Non-pathogenic subtype – prevalent in ducks and turkeys.

2. AQUABIRNAVIRUS

INFECTIOUS PANCREATIC NECROSIS (IPN) VIRUS

Associated disease:

Infectious pancreatic necrosis is a disease of salmonids. Other hosts include fresh wales, fish, marine fish, marine mollusks and crabs.
Most commonly affected spp. is rainbow trout but also occurs in brooks trout, cut throat trout, Amago, brown trout and Atlantic salmon.

The disease is limited to “fry” maintained under hatchery cultivation.

Main signs: darkened pigmentation, distended abdomen and spiraling motion. Large masses of mucus and sloughed cells accumulate in the stomach and intestines. The liver, spleen are often enlarged, petechial hemorrhages may be seen in the visceral mass. Necrosis of the pancreatic acinar cells. Mortality is very high up to 90%. Survivors become disease-free carrier probably for whole life.

Isolation: Virus can be isolated from the eggs and seminal fluid.

Cultivation: Homogenate of whole “fry” filtered.

Cell culture: RTG – 2 cells at 15 C, CPE: Plaque formation.

Haemagglutination:

Mouse RBCs

Diagnosis: NT, CFT, IF, EIA, HA.

Immunity: Live attenuated vaccines are used while the killed vaccines are under study.

ADENOVIRIDAE

(*Adenos*: gland); first isolated from explants culture of human adenoids.

Classification

The family “*adenoviridae*” comprises 4 genera:

Genus	Virus
<i>Mastadenovirus</i> (Infects mammals only share a common antigen and are serologically distinct from avian adenovirus)	Human adenovirus A F Canine adenovirus (e.g. Canine Infectious hepatitis virus) Equine adenovirus Bovine adenovirus Ovine adenovirus Caprine adenovirus
<i>Aviadenovirus</i> (group I Aviadenovirus)	Fowl adenovirus A, B, C, D, E Chicken Embryo lethal orphan (CELO) virus;(Fowl adenovirus I) Inclusion body hepatitis virus Quail bronchitis virus
<i>Atadenovirus</i> (group III Aviadenovirus)	Egg drop syndrome virus Snake adenovirus
<i>Siadenovirus</i> (group II Aviadenovirus)	Turkey hemorrhage enteritis virus Marble spleen disease virus of pheasants Hydro-pericardium syndrome (HPS) virus of chicken ?

General Properties

Shape and size:

Virion is non-enveloped (naked) icosahedron with a diameter of 70-90 nm. The capsid contains 252 capsomers which consists of 240 hexons and 12 pentons. The 12 pentons are located at each of the vertices, have a penton base and a fiber. The fiber contains a viral attachment proteins and act as a hemagglutinin. The pentons and fibers also carry type specific antigens. Capsid contains viral DNA and at least 2 major proteins. There are at least 11 structural proteins, 9 of which have an identified structural function.

Nucleic acid: The adenovirus genome is a linear, dsDNA with a terminated protein (55kDa) attached at each 5' end.

Adenoviruses cause lytic, persistent and latent infection in humans, animals and birds. Some strains can immortalize certain animal cells.

Replication: Transcription occurs in the nucleus of the infected cell and DNA dependant RNA polymerase is involved in transcription mRNA → Cytoplasm.

Capsid proteins are produced in the cytoplasm and then transported to the nucleus for viral assembly. Viral DNA replication occurs in the nucleus. Empty procapsids first assemble and then the viral DNA and core proteins enter the capsid through an opening at one of the vertices.

Resistance: Adenoviruses remain viable for about 1 week at 37C. They are inactivated by heating at 56C for more than 10 min. They resist ether and bile salts, also withstand freezing, mild acids and lipid solvents.

Cultivation: Primary cells (Embryonic kidney cells, Liver cells), Continuous cell lines (HELA, human epidermal carcinoma cells), HEP – 2 cells and KB cells.

CPE: after 1-4 weeks → rounding and aggregation of cells in grape like clusters. Infected cells swell and became ballooned and show characteristic basophilic intranuclear inclusions.

Transmission: Virus is spread by aerosol, close contact or fecal –oral means, and inadequately chlorinated swimming pools/ponds.

Pathogenesis: Adenoviruses infect mucoepithelial cells in the respiratory, gastrointestinal tract and conjunctiva causing cell damage directly. The virus initially multiply in the conjunctiva, pharynx or small intestine and spread to pre-auricular, cervical and mesenteric lymph nodes. Most of the enteric and some respiratory are subclinical.

The toxic activity of the penton base proteins can result in inhibition of cellular mRNA transport and protein synthesis, cell damage and tissue damage.

Adenovirus has a tendency to become latent in lymphoid and other tissues such as adenoids, tonsils, and Peyer's patches, and can be reactivated in patients who are immuno-compromised or have been infected with other agents. Some human adenovirus (A & B) can transform and are oncogenic in rodent cells. Transformation of human cells has not been observed.

Immunity: Antibody is important for resolving lytic adenovirus infection and protects the person from reinfection with the same serotypes but not other serotypes. Cell mediated immunity (CMI) is important in limiting virus outgrowth. Adenovirus encodes several early proteins that help the virus to avoid immune defenses.

Diagnosis: From throat swabs, nasal secretions etc. Fecal samples
ImmunoAssays – FAT, ELISA

Molecular techniques – PCR, DNA probes, Immuno electron microscopy

Control: Vaccine; Live oral, genetically engineered

1. MASTADENDOVIRUS

INFECTIOUS CANINE HEPATITIS VIRUS

(Canine adenovirus type I)

Synonym: Disease caused by ICH virus may be termed as; Canine infectious hepatitis; Rubarth's disease; hepatitis contagiosa canis; fox encephalitis.

Associated disease:

Virus is associated with a febrile, distemper-like disease which affects mainly foxes and young dogs and is associated with "centrilobular necrosis of liver".

Susceptible Hosts:

Dogs and foxes are the natural hosts but guinea pigs, coyotes, raccoons and wolves are susceptible to experimental infectious. Grey foxes and ferrets are said to be resistant. The disease is widespread in UK, and North America.

Resistance: The virus resists inactivation by heat and acid, and as it contains lipid, its infectivity is not affected by organic solvents or detergents.

Haemagglutination:

Both canine adenoviruses (infectious hepatitis and infectious laryngotracheitis) agglutinate human group O RBCs, but only ICH virus will agglutinate guinea pig RBCs.

Serotypes: Infectious canine hepatitis virus has only one serotype, but strains may vary in virulence.

Cultivation: Cell culture: Canine kidney and testis cell cultures. CPE: rounding and swelling of infected cells and formation of intranuclear inclusions.

Pathogenicity:

The usual incubation period is 2-5 days and may be up to 14 days depending on the virulence of the virus strains.

In most severe form: an apparently healthy dog suddenly collapses with acute abdominal pain, vomiting, diarrhea and dies within 12-24 hours.

In less acute cases; there is high fever accompanied by leucopenia, enlargement of tonsils and submaxillary lymph nodes and sometimes edema of the cornea giving rise to transient corneal opacity.

At autopsy: subcutaneous edema and an hemorrhagic exudate in the peritoneal cavity and intestinal tract. The liver is enlarged, pale and often mottled. The spleen is enlarged and hemorrhagic and wall of the gall bladder is edematous and thickened.

Diagnosis: Serologically: Electron microscopy of infected cells reveals crystalline arrays of virus particles in the nucleus. Other tests are; FAT, VN, CFT or IHA test.

Vaccines: Both formalin inactivated virus and live attenuated virus vaccines are available.

2. AVIADENODVIRUS

CHICKEN EMBRYO LETHAL ORPHAN (CELO) VIRUS

General Properties

- ▶ Generally endogenous contaminant from embryonating chicken eggs.
- ▶ No apparent disease – in chickens.
- ▶ May activate other infections like Mycoplasma, infectious bronchitis.
- ▶ May interfere with propagation of other viruses in eggs.
- ▶ Since CELO virus is contaminant in kidney cell culture, thus may contaminate vaccine (If kidney cells are used vaccine production).

QUAIL BRONCHITIS VIRUS

General Properties:

- ▶ The virus causes an acute & fetal respiratory disease in young quails are most susceptible.
- ▶ Coughing, sneezing, conjunctivitis, lacrimation.
- ▶ Mortality may be up to 50%.
- ▶ The virus may develop malignant tumors in hamsters.
- ▶ Immunity may be up to 6 months.
- ▶ Virus neutralizing antibodies have been demonstrated in recovered quails.

3. ATADENOVIRUS

EGG DROP SYNDROM (EDS) VIRUS

Associated disease:

Virus causes thin shelled or shell less eggs produced by otherwise healthy birds. The virus affects only avian spp. and therefore, has no public health significance.

About Virus: EDS virus is hemagglutinating adenovirus, which is transmitted vertically but not horizontally. The virus remain latent until birds approach peak egg production. It has been isolated from normal ducks and regarded as duck adenovirus. It has been isolated from the chicken in many countries.

Replication: EDS virus replicates in the nucleus. Intranuclear inclusions in infected cell culture, in epithelial cell of the infundibulum, tubular shell gland, pouch shell gland, isthmus, nasal mucosa and spleen. In ultra thin sections, type I-IV inclusions are obvious in the nucleus.

Resistance: EDS virus is stable to treatment with chloroform and variation in pH (3-10). It is inactivated by heating for 30 mn at 60C. It survives for 3 hours at 56C and is stable in monovalent but not divalent cations. Infectivity is lost after treatment with 0.5% formaldehyde or 0.5% gluteraldehyde.

Serotypes: Only one serotype has been recognized. With restriction endonuclease analysis, the virus has been divided into 3 genotypes.

Cultivation: Cell culture: duck kidney, duck embryo liver and duck embryo fibroblasts cells. Chick embryo liver cells. It grows less well in chicken kidney cells and poorly in chicken embryo fibroblast cultures. There is a poor growth in turkey cells and no replication in a range of mammalian cells. The virus grows to high titre in goose cell cultures,

Embryonated Egg: No growth in embryonated chicken eggs, but high titre when inoculated into Allantoic sac of embryonated duck and goose eggs.

Susceptible Hosts: Natural hosts: ducks and geese but disease may be seen in laying hens. A wide range of breeds of chicken are susceptible. Broiler breeder and heavy breeders producing brown eggs are more affected than with egg producers.

All ages of birds are susceptible to infection of EDS virus.

Transmission: Contamination of ducking water by droppings. The laying hens spread EDS virus by contaminating the environment by feces. Needles or blades used for vaccination or bleeding of viremic birds can transmit the virus. The main method of spread of EDS virus is vertical.

Immune Response:

Antibody to EDS virus can be detected in 5-7 days after infection. They tend reach a peak in about 4-5 weeks.

Birds still excrete virus even in the presence of high titre antibody.

Antibody is transferred via yolk sac. The chicks have antibody of high titre with a half life of 3 days. Active antibody production is stimulated at 4-5 weeks of age when maternal Ab is nearly undetectable.

If flock as a whole develops antibody to EDS virus before coming into lay egg production, is not affected.

Isolation: Abnormal eggs, pouch shell eggs and infected eggs are source of virus.

Diagnosis: Serologically: HI, ELISA, SN, FAT, DID - for antibody to EDS virus.

Many flocks containing birds infected with EDS virus in ovo don't show antibody during the growing period, and it is only apparent following clinical signs.

Vaccines: An oil adjuvanted-inactivated vaccine is widely used which gives good protection against clinical disease. Vaccine is given between 14-16 weeks of age. Infected birds give better response to vaccine than previously uninfected birds. Immunity lasts for 1 year.

Where vertically or laterally transmitted infection is thought to be a possibility, the flock in danger can be protected by vaccination in growing period.

4. **SIADENOVIRUS**

The viruses included in this group are;

- i. Hemorrhagic enteritis virus (turkeys)
- ii. Marble spleen disease virus (pheasants)
- iii. Avian adenovirus group II spleenomegaly virus (chickens)

Characteristics:

Members of group "siadenovirus" are distinguishable from each other by restriction endonuclease fingerprinting. These viruses are indistinguishable in *agar gel double diffusion trts* and are unrelated to *chicken embryo lethal orphan virus*.

Replication: The viruses replicate intranuclearly in cells of the reticuloendothelial system, primarily in the spleen. Sensitive techniques such as ELISA and Immunofluorescence have been detected viral antigen in small amounts in many tissues.

Resistance: Infectivity of HEV can be destroyed by heating at 70C for 1 hour, drying at 37C for 1 week or by treatment with 0.0086% sodium hypochloride, 0.4% phenoxide or 1% lysol. But infectivity can not be destroyed at 65C for 1 hour, storage for 6 months at 4C, 4 year at -40C, 4 week at 37C, at pH3 or by treatment with 50% chloroform or 50% ethyl ether.

Natural Host: Turkey (6-11 wk), pheasants (3-8 wk) and chickens (mature or market age) are the only natural hosts of HEV, MSDV and AASV respectively. Cause up to 60% mortality.

Cultivation: Turkey cell line of lymphoblastoid B cell derived from a Marek's disease tumor.
Turkey lymphocytes (normal)

Transmission: i) Oral, cloacal infection by contaminated feces, litter.
ii) No egg transmission
iii) Carriers and vectors have no role in transmission.

Pathogenesis:

The viruses first multiply in reticuloendothelial cells of spleen and then viraemic leads to sloughing of tips of villi and followed by hemorrhage into the lumen from broken capillaries.

Immunity: Both active and passive immunity have been observed. The HEV is immunosuppressive as lymphocytes and RE cells are the target cells of HEV.

Haemagglutination: None

Diagnosis: Serologically, AGPT, ELISA, PCR.

Vaccines: Passively injection of convalescent sera can be used to treat the sick birds.
A live avirulent, water-administered vaccine may be used to control infection.

HYDRO-PERICARDIUM SYNDROM (HPS) VIRUS

General Features:

- ▶ Virus belongs to genus *siadenovirus* of family *adenoviridae*.
- ▶ Non-hemagglutinating virus
- ▶ Predilaction site: Liver (hepatocytes enlarged)
- ▶ Transmission via fecal-oral route
- ▶ No egg transmission
- ▶ Immunosuppressive.