Picornaviridae

VMC 321

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ETYMOLOGY

• Picornaviridae
• ‘Pico’ - very small
• ‘rna’ - RNA
• viridae - suffix used for family of virus

• The Picornaviridae family includes small, icosahedral viruses with single-stranded, highly diverse positive-sense RNA genomes.
### Taxonomy

**Group IV : (+) sense, ss RNA viruses**

**Order: Picornavirales**

<table>
<thead>
<tr>
<th>Family</th>
<th>Genus</th>
<th>Species</th>
<th>Host</th>
</tr>
</thead>
<tbody>
<tr>
<td>Picornaviridae</td>
<td>Aphthovirus</td>
<td>Foot-and-mouth disease virus</td>
<td>cloven footed animal</td>
</tr>
<tr>
<td></td>
<td>Avihepatovirus</td>
<td>Duck hepatitis A virus</td>
<td>Duck</td>
</tr>
<tr>
<td></td>
<td>Erbovirus</td>
<td>Equine rhinitis B virus</td>
<td>Equine</td>
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<tr>
<td></td>
<td>Hepatovirus</td>
<td>Hepatitis A virus</td>
<td>Vertebrates</td>
</tr>
<tr>
<td></td>
<td>Sapelovirus</td>
<td>Porcine sapelovirus</td>
<td>Pig</td>
</tr>
<tr>
<td></td>
<td>Teschovirus</td>
<td>Porcine teschovirus</td>
<td>Pig</td>
</tr>
</tbody>
</table>
## Characteristics of members of the family *Picornaviridae*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Description</th>
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<tbody>
<tr>
<td><strong>Prototype</strong></td>
<td>poliovirus 1, species Enterovirus C, genus Enterovirus</td>
</tr>
<tr>
<td><strong>Virion</strong></td>
<td>Non-enveloped, 30–32 nm virions comprising 60 protomers</td>
</tr>
<tr>
<td><strong>Genome</strong></td>
<td>6.7–10.1 kb of positive-sense, non-segmented RNA with a poly(A) tail</td>
</tr>
<tr>
<td><strong>Replication</strong></td>
<td>RNA synthesis occurs in reorganized cytoplasmic replication organelles containing non-structural proteins derived from the 2BC-P3 region of the encoded polyprotein; RNA structures at the 5′ and 3′ ends of the genome direct initiation of RNA synthesis and uridylated 3B serves as primer for synthesis of both RNA strands</td>
</tr>
<tr>
<td><strong>Translation</strong></td>
<td>Directly from genomic RNA containing an internal ribosomal entry site (IRES)</td>
</tr>
<tr>
<td><strong>Host range</strong></td>
<td>Vertebrates (at least five of the seven classes)</td>
</tr>
</tbody>
</table>
Virion Morphology

- Virions consist of a capsid, with no envelope, surrounding a core of ssRNA.
- Virus particles size: 30–32 nm in diameter
- Shape— Isometric; non-enveloped
- Symmetry — Icosahedral
- Genome— linear; 7.2- 8.4 kb; single segment; positive sense; single stranded RNA
- Molecular weight— 2.5 × 10^6 k Da
- Virus has four major protein (VP₁, VP₂, VP₃, & VP₄) & one minor protein(VP₀)
Replication

i. virion attaches to receptor, penetrates the host cell

ii. uncoating occurs & VP₀ is removed from RNA segment by cellular enzyme

iii. FMDV use entire RNA as mRNA, whole genome is translated & cleaves into different functional protein.
The Disease

• Foot and Mouth Disease (FMD)
Susceptible host:

- Cattle
- Buffalo
- Sheep
- Goat
- Deer
- Pig
- Camel
- Mithun
- Yak
Reservoir/Carrier:

- Wild ruminants
- Cattle - indicator host
- Sheep - maintenance host
- Pig - amplifying host
Transmission:

• spread at rapid rate through:
  • contact with infected animal
  • contaminated feed, Utensil
  • aerosol
  • ingestion
Pathogenicity:

- RNA itself is infectious
- Virus invades epithelium of upper respiratory tract or alimentary tract by via inhalation or ingestion and multiplies at the site of infection
- Formation of vesicle within 1-4 days
- Primary vesicle initiates viraemia and associated with fever
- Dissemination through viraemia and lodgment in distant epithelia & formation of secondary vesicle
- Stratified squamous epithelium are the predilection site of FMDV and vesicles are found in these tissues or mucous membrane.
Clinical sign:

• Appearance of vesicles on mucous membrane of tongue, lips, gums, cheeks, dental pad & skin of interdigital space, teats and udder

• Profuse salivation, drooling of foamy & ropy saliva and protrusion of tongue

• Pregnant animals abort

• Suckling calves die as a result of myocardial degeneration (tigeroid heart)
Diagnosis:

1. Clinical signs and symptoms

2. Isolation and identification of virus
   - Specimen for virus isolation: vesicular fluid, epithelial tissues from ruptured vesicle & transported in glycerol buffer

3. Demonstration of viral antigen in clinical specimen
   - Virus neutralization test (VNT)
   - Immunofluorescence test (IFT)
   - Complement fixation test (CFT)
   - Enzyme linked immunosorbent assay (ELISA)
4. Demonstration of antibody in convalescent sera
   - Liquid phase block - Enzyme linked immunosorbent assay (LPB-ELISA)
   - Complement fixation test (CFT)

5. Detection of viral nucleic acid
   - *In situ* hybridization (with labeled gene probes)
   - Reverse transcriptase – polymerase chain reaction (RT-PCR)
## Precaution:

<table>
<thead>
<tr>
<th>Vaccination</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>4 months &amp; above</td>
</tr>
<tr>
<td>Booster</td>
<td>1 month of first vaccination</td>
</tr>
<tr>
<td>Revaccination</td>
<td>Every six months</td>
</tr>
</tbody>
</table>

1. **Vaccination**
   
   Trivalent FMD vaccine containing O, A, , Asia-1
   
   Dose- 2ml (Cattle & buffalo) ; 1 ml (Sheep & Goat)

2. **Disease awareness, early detection, proper disposal of affected carcass, timely notification,**

3. **Strict biosecurity, isolation, zoning, quarantine, control of animal movement are prerequisites for effective control programmes aimed at eradication**
Duck hepatitis virus (DHV)

- Three types of viruses are recognized:
  - Type 1
  - Type 2
  - Type 3
- All the above three types causes hepatitis in duck, however, type 1 is prevalent in India
The Disease

- DHV causes duck virus depotitis

- **Duck virus hepatitis** :- Duck hepatitis is a highly fatal, rapidly spreading viral infection of young ducklings characterized primarily by hepatitis.

- **Susceptible host:** Ducklings

- **Reservoir:** Wild birds, recovered ducks and rats

- **Transmission:** Contact with infected bird

- **Incubation period:** 18-24hrs.
Pathogenicity:

I. Duckling of 3-4 weeks old are infected; disease takes rapid course

II. Virus invades epithelium of upper respiratory tract by inhalation; multiplies at the site of infection

• Mortality is as high as 100%; morbidity may be up to 95%
Clinical sign:

I. The duckling reluctance to move & squat down

II. Birds fall down on their sides; starts kicking their legs spasmodically and death ensues with head bent backward

III. Death occurs within an hour
Diagnosis:

1. Clinical signs and symptoms

2. Post – mortem lesions :-
   ✓ Hepatomegaly with punctuate & ecchymotic haemorrhages ;
   ✓ Mottling & discolouration of liver ;
   ✓ Spleenomegaly & its mottling ;
   ✓ In few cases swelling of kidney & congestion of blood vessel is observed.
• 2. Isolation and identification of virus

• Specimen for virus isolation: Liver of infected bird
  
  ➢ Suspected sample is inoculated into SPF/antibody free embryonated duck egg (10-14 day old) or chicken egg (9-11 day old) via allantoic sac; infected duck embryo dies within 24-72 hrs.; Chicken embryo dies within 5-8 days. The gross lesion of embryo shows stunting & sub cutaneous haemorrhages of body, oedema of abdomen & hind limb. Swollen, reddened & yellowish discoloration of liver.
  
  ➢ Primary cell culture of duck embryo liver cells.
    • CPE – includes rounding & necrosis of cells,
• 3. Demonstration of viral antigen in clinical specimen
  ➢ Virus neutralization test (VNT)
  ➢ Immunofluorescence test (IFT) – rapid & accurate diagnostic method
  ➢ Enzyme linked immunosorbent assay (ELISA)

• 4. Demonstration of antibody in convalescent sera
  ➢ Virus neutralization test (VNT)
  ➢ Enzyme linked immunosorbent assay (ELISA)
Thanks