Summary

The Hepeviridae includes enterically-transmitted, small, non-enveloped positive-sense RNA viruses (Table 1. Hepeviridae). Members of the family are assigned to two genera and five species. Members of the Piscihepevirus genus infect trout and members of the Orthohepevirus genus infect mammals and birds. The species Orthohepevirus A includes hepatitis E virus (HEV), which is usually responsible for self-limited acute hepatitis in humans and several mammalian species, but may become chronic in immunocompromised humans. Extrahepatic manifestations of Guillain-Barré syndrome, neuralgic amyotrophy, glomerulonephritis, and pancreatitis have been described in a proportion of HEV cases. The species Orthohepevirus B includes avian HEV that causes hepatitis-splenomegaly syndrome in chickens.

Table 1. Hepeviridae. Characteristics of members of the family Hepeviridae.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical member</td>
<td>hepatitis E virus Burma (M73218), species Orthohepevirus A, genus Orthohepevirus,</td>
</tr>
<tr>
<td>Virion</td>
<td>Non-enveloped, 27–34 nm diameter with a single capsid protein</td>
</tr>
<tr>
<td>Genome</td>
<td>6.4-7.2 kb capped positive-sense monopartite RNA containing 3 open reading frames</td>
</tr>
<tr>
<td>Replication</td>
<td>Occurs in association with the host endoplasmic reticulum.</td>
</tr>
<tr>
<td>Translation</td>
<td>From genomic (ORF1) and subgenomic (ORF2 and ORF3) capped RNA</td>
</tr>
<tr>
<td>Host range</td>
<td>Mammals (Orthohepevirus A, C and D), birds (Orthohepevirus B) and trout (Piscihepevirus)</td>
</tr>
<tr>
<td>Taxonomy</td>
<td>two genera</td>
</tr>
</tbody>
</table>

Virion

Morphology

Virions of HEV are icosahedral, non-enveloped, spherical particles with a diameter of approximately 27–34 nm (Figure 1. Hepeviridae). Spikes and indentations can be seen on electron micrographs (EM) of virions (Bradley 1990). The capsid is formed from capsomeres consisting of homodimers of a single capsid protein, forming the virus shell. Each capsid protein contains three linear domains forming distinct structural elements: S (the continuous capsid), P1 (three-fold protrusions), and P2 (two-fold spikes). Neutralizing epitopes have been found in the P2 domain. Each domain contains a putative polysaccharide-binding site that may interact with cellular receptors. Native T=3 capsids contain flat dimers, with less curvature than those of T=1 virus-like particles (Figure 2. Hepeviridae) (Mori and Matsuura 2011). Virion particles of avian HEV (Figure 3. Hepeviridae) revealed by negative staining EM of bile samples from chickens with hepatitis-splenomegaly syndrome are similar in size and morphology to HEV (Haqshenas et al., 2001).
Figure 1. **Hepeviridae.** Negative contrast electron micrograph of hepatitis E virus virions from a case stool collected in Nepal. (A) virion and (B) empty capsid. The bar represents 100nm (photo from M. Purdy).

Figure 2. **Hepeviridae.** Structure of the hepatitis E virus-like particle (VLP) (T=1). (A) Crystal structure of hepatitis E virus VLP. The three domains, S, P1 and P2 are colored blue, yellow and red, respectively. The VLP is positioned in a standard orientation with the 3 2-fold icosahedral symmetry axes aligned along the vertical, horizontal, and viewing directions, respectively. (B) Cryo-EM reconstruction at 14 Å resolution. The surface is coloured by radial depth cue from blue, yellow, to red. (C) Hepatitis E virus VLP with only the S domain. (D) VLP with S and P1 domains. (E) VLP with P1 and P2 domains (from (Guu et al., 2009) with permission).
Physicochemical and physical properties

Members of the *Orthohepevirus* genus have virion buoyant densities of 1.35 to 1.40 g cm\(^{-3}\) in CsCl and 1.29 g cm\(^{-3}\) in glycerol and potassium tartrate gradients. The virion \(S_{20,w}\) is 183S. Virions are sensitive to low-temperature storage (between −70°C and +8°C) and iodinated disinfectants (Bradley 1990). The virion of HEV is more heat-labile than that of hepatitis A virus (HAV): HEV was about 50% inactivated at 56°C, but 1% infectivity remained after 60 min at this temperature; no infectivity was detected after heating at 66°C or 70°C for 1 h. (Emerson et al., 2005). Liver suspensions containing avian HEV remained infectious after treatment with chloroform and ether but lost infectivity after incubating at 56°C for 1 h or 37°C for 6 h. Viral infectivity in liver suspensions was reduced 1000-fold after treatment with 0.05% Tween-20, 0.1% NP40 and 0.05% formalin (Meng et al., 2006). Properties of members of the *Piscihepevirus* genus have not been investigated.

Nucleic acid

The genome of members of the *Hepeviridae* is a linear, positive-sense, ssRNA molecule of approximately 6.6 - 7.2 kb, with a 5′-m\(^{7}\)G cap structure and a 3′-poly(A) tail.

Proteins

Virions are constructed from a major capsid protein (CP) encoded by the second open reading frame (ORF2). The CP binds to surface heparin sulfate proteoglycans (HSPGs) on liver cells (Kalia et al., 2009) and may be proteolytically processed. A small immunoreactive protein (113-114 amino acids, 12.5 kDa) encoded by the third ORF (ORF3) has been identified and shown to exhibit multiple functions associated with virion morphogenesis, egress and viral pathogenesis. Recently, the ORF3 polypeptide has been shown to share several structural features with class I viroporins (Ding et al., 2017). Non-structural proteins encoded by the first major ORF (ORF1) have limited similarity with the “alpha-like supergroup” of viruses and contain domains consistent with a methyltransferase, papain-like cysteine protease, macro domain, RNA helicase and RNA-dependent RNA polymerase (Cao and Meng 2012). Some of these predicted enzymatic properties have been confirmed experimentally (Karpe and Lole 2010, Parvez 2015, Mahilkar et al., 2016).

A hypervariable region lies between the protease and helicase domains. The hypervariable region contains two subregions; the amino-terminal half of this region consists of a variable length polypeptide with host species-specific sequence conservation, which is unique to *Hepeviridae* (Kelly et al., 2016) and may be responsible for host specificity (Lara et al., 2014). The carboxyl-terminal half consists of an intrinsically-disordered polypeptide with a high frequency of proline residues (Kelly et al., 2016, Purdy 2012). The translational and post-translational processing of the non-structural polyprotein remain unresolved. In particular, it remains unclear whether the non-structural polyprotein functions as a single protein with multiple functional domains or whether it is proteolytically cleaved into smaller proteins with distinct enzymatic activities.

Lipids

Although HEV is shed in faeces as a non-enveloped virus there is evidence that, like HAV, HEV can hijack host membranes on assembly and exit. Possession of a host-derived envelope may allow the virus to circulate in a patient’s blood escaping detection by neutralizing antibodies (Yin et al., 2016).

Carbohydrates
Evidence for glycosylation of the major CP has been reported following its expression in mammalian cells (Jameel et al., 1996). The CP sequence contains three potential sites for N-linked glycosylation and a signal peptide sequence at its N terminus (Zafrullah et al., 1999). Mutations within Orthohepevirus A CP glycosylation sites prevent the formation of infectious virus particles, although the lethal effect is due to altered protein structure rather than elimination of glycosylation (Graff et al., 2008).

Genome organization and replication

The RNA genome of members of the Hepeviridae is organized into three ORFs, with the non-structural proteins encoded toward the 5′ end of the genome and the structural protein(s) toward the 3′ end. Capped genomic RNA of an Orthohepevirus B isolate has been shown to be infectious for chickens (Huang et al., 2005a) and that of Orthohepevirus A for pigs, rhesus monkeys and chimpanzees (Panda et al., 2000, Emerson et al., 2001, Huang et al., 2005b). The 5′-untranslated region (UTR) is up to 34 nt in length among members of Orthohepevirus A, but is 100 nt in cutthroat trout virus (Piscihepevirus A). A 5′-methylguanine cap structure has been identified at the 5′ end of the HEV genome and plays a role in the initiation of virus replication (Ahmad et al., 2011). The 3′-UTR of HEV contains a cis-reactive element (Emerson et al., 2001) as does a central region of ORF2 (Emerson et al., 2001). The 3′ end of the genome is also polyadenylated.

In all members of the Hepeviridae, ORF1 encodes a non-structural polyprotein, followed by ORF2 that encodes the CP, and an overlapping reading frame, ORF3 which encodes a small phosphoprotein of 113–114 aa with a multifunctional C-terminal region (Figure 4. Hepeviridae). A bicistronic subgenomic mRNA encoding both ORF2 and ORF3 proteins has been identified (Graff et al., 2006). Isolates have been described from chronically infected individuals that contain an insertion of host-derived sequences such as human ribosomal protein S17 within the ORF1 hypervariable region that confers a growth advantage in cultured hepatoma cells (Shukla et al., 2011, Nguyen et al., 2012).
Figure 4. Hepeviridae. Genome organization of members of Hepeviridae. Schematic diagrams are shown for cutthroat trout virus (Piscihepevirus A), human hepatitis E virus (Orthohepevirus A), avian hepatitis E virus (Orthohepevirus B), rat hepatitis E virus (Orthohepevirus C), bat hepatitis E virus (Orthohepevirus D) and unassigned isolates from moose, kestrel and little egret. The diagram shows a short 5′ non-coding region (NCR), a 3′ NCR, and three ORFs. ORF1 encodes non-structural proteins including putative functional domains MT, methyltransferase; P, a putative papain-like cysteine protease; HUD, Hepeviridae unique domain also called the Z domain (Kelly et al., 2016); PP, a hypervariable polyproline region that is dispensable for virus infectivity; Hel, helicase; RdRp, RNA-dependent RNA polymerase (Kelly et al., 2016, Koonin et al., 1992). ORF2 (green) encodes capsid protein and ORF3 (brown) encodes a small phosphoprotein with a multi-functional C-terminal region. ORF2 and ORF3 overlap each other but neither overlaps ORF1.

Replication of HEV is not well understood. The viral RdRp associates with the host endoplasmic reticulum (ER) through residues 4449-5109 encoding a predicted transmembrane domain to begin replicating the viral genome. It appears that replication involves temporal separation and alternating cycles of positive- and negative-sense RNAs to produce capsid, ORF3 protein, ORF1 polypeptide, and new genomes, resulting in the generation of progeny virions (Varma et al., 2011).

Biology

Hepevirus infection occurs by the faecal-oral route for Orthohepevirus A and Orthohepevirus B, and is usually associated with a self-resolving mild, acute infection of the liver. More recently, chronic hepatitis E has become a significant clinical problem in immunosuppressed individuals especially in solid organ transplant recipients. Transmission can occur through contaminated water, consumption of raw/undercooked meats or faeces from infected animals, and rarely through blood transfusions. Little is known about the mode of transmission or pathology for other species in the family.

Derivation of names

Hepe: from hepatitis E virus.

Genus demarcation criteria

Members of different genera differ in host range with piscihepeviruses only known from fish, while orthohepeviruses have been isolated from a wide range of mammals and birds. Members of the two genera also differ in phylogenetic relationships observed for comparisons between three conserved regions within ORF1. These relationships are mirrored in sequence distance between representatives of each genus with inter-genus amino p-distances greater than 0.6 compared to intra-genus p-distances of less than 0.5 (Smith et al., 2014). Members of the same genus all share similar genome organisation: in orthohepeviruses ORF3 overlaps the 5′-end of ORF2 while in piscihepeviruses the overlap is more central (Batts et al., 2011).

Relationships within the family

The family includes two genera whose members are phylogenetically distinct; the genus Orthohepevirus includes four species that are phylogenetically distinct and have different host ranges (Figure 5. Hepeviridae). Only one species has been described in the genus Piscihepevirus.
Figure 5A. **Hepeviridae.** Phylogenetic trees for members of the *Orthohepevirus* genus. Maximum likelihood trees for amino acid distances were created using MEGA 6 ([Tamura et al., 2013](https://www.ncbi.nlm.nih.gov/pubmed/23553287)) with the Le and Gascuel substitution model with frequencies ([Le and Gascuel 2008](https://www.ncbi.nlm.nih.gov/pubmed/28911967)) and 4 gamma rate categories. Partial amino acid deletion was allowed using a 90% coverage cutoff. 1000 bootstrap replicates were used. Branches supported by > 80% of bootstrap replicates are indicated. (Figure 5A. **Hepeviridae** - above) ORF1 sequences from assigned and unassigned *Orthohepevirus* species were used with regions between 485-776 aa and 923-931 aa (numbered with reference to the ORF1 polyprotein of M73218) removed from the analysis due to indels within these regions. (Figure 5B. **Hepeviridae** - below) Complete ORF2 sequences. Sequences are identified by their accession ID and host. *Orthohepevirus A* sequences are additionally identified by genotype. *Orthohepevirus* species are denoted by their species names. Unclassified viruses are denoted by the host from which they were first isolated. These phylogenetic trees and corresponding sequence alignments are available to download from the Resources page. 

www.ictv.global/report/hepeviridae
Relationships with other taxa

HEV is similar to members of the family Caliciviridae based on the superficial structural morphology as revealed by EM, and its genome organization (Bradley 1990). However, members of the two families show little detectable sequence homology and the cap structure at the 5′ end of the HEV genome is absent in caliciviruses. HEV shows highest, but limited, amino acid sequence similarity in its replicative enzymes with Rubella virus and alphaviruses of the family Togaviridae and with plant furoviruses. The capping enzyme, helicase and replicase of HEV have properties very similar to those of viruses within the “alphavirus-like supergroup” (Kelly et al., 2016).

Related, unclassified viruses

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<td>1926998</td>
</tr>
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</table>

Virus names and virus abbreviations are not official ICTV designations.
**Genus: Orthohepevirus**

**Distinguishing features**

Members of the *Orthohepevirus* genus infect mammals and birds. They share a common genome organisation with a short 5′-untranslated region, a long ORF1 followed by the overlapping and shorter ORF2 and ORF3, with a poly-A tail at the end of ORF2.

**Virion**

See discussion under family description.

**Genome organization and replication**

See discussion under family description.

**Biology**

HEV is associated in humans with outbreaks and sporadic cases of acute hepatitis. The virus is considered endemic in tropical and subtropical countries of Asia, and Africa, as well as Mexico, but antibody prevalence studies suggest a global distribution for this virus. Large outbreaks that may involve thousands of cases of acute hepatitis occur in endemic regions. Sporadic cases are more common in industrialized countries. Human-to-human transmission seems rare in hepatitis E epidemics although infections can be transmitted by blood transfusion from acutely infected donors (Huzly et al., 2014, Hewitt et al., 2014). Members of *Orthohepevirus A* have been assigned to 7 different genotypes, HEV-1 to HEV-7. Recently a new HEV genotype (HEV-8) was proposed for viral variants isolated from Bactrian camels (Woo et al., 2016). Of these genotypes; HEV-1, HEV-2, HEV-3 and HEV-4 are most commonly associated with HEV infection in humans. Genotypes 1 and 2 are restricted to humans, whereas genotypes 3 and 4 have a broader host range and are zoonotic. Interspecies transmission of genotypes 3 and 4 hepatitis E virus between swine and non-human primates has been demonstrated experimentally. Pig handlers in both developing and industrialized countries are at increased risk of HEV infection. Human isolates from southern Asia typically belong to HEV-1 and are epidemiologically transmitted faecal-orally. Isolates of HEV-1 in Western Europe are usually associated with recent travel to southern Asia or Africa. Very few isolates of HEV-2 have been described, but these comprise a range of locations (Mexico and Africa) and are associated with epidemic faecal-oral transmission. Genotypes HEV-3 and HEV-4 have been isolated from humans, pigs and deer in Western Europe, South America, Northern America and Eastern Asia, with human infection presumed to result from the consumption of raw or undercooked pig meat or products although a direct link has seldom been proven. Exceptions are the consumption of figatellu sausage in France (Colson et al., 2010) and raw deer liver in Japan (Tei et al., 2003, Takahashi et al., 2004). Recent studies have expanded the host range of HEV-3 to include goats (Di Martino et al., 2016) and bottlenose dolphins (Montalvo Villaíba et al., 2017), and of HEV-4 to include cattle (Hu and Ma 2010, Huan et al., 2016) and sheep (Wu et al., 2015). A variant of HEV-3 found in rabbits has distinctive insertions in ORF1 (Zha et al., 2009) and has also been isolated from a human (Izopet et al., 2012). HEV-5 and HEV-6 have only been isolated from wild boar in Japan (Takahashi et al., 2014). HEV-7 has been isolated from dromedary camels (Woo et al., 2014) with one report of human infection (Lee et al., 2016). HEV-8 has only been isolated from Bactrian camels in China (Woo et al., 2016).

Human infection with HEV is typically self-limiting and frequently asymptomatic; studies of blood donors in several European countries report that there are typically no or minor symptoms with infrequent mild elevation of liver enzymes (Vollmer et al., 2012, Juhl et al., 2014, Tedder et al., 2016). The incubation period for hepatitis E ranges from 15 to 40 days. Symptoms include diarrhoea, epigastric pain, nausea, hepatomegaly, splenomegaly and vomiting. The icteric phase of illness begins with jaundice, dark urine and clay-coloured stools. Extrahepatic manifestations are associated with the brain, central nervous system, muscle tissue, kidney, pancreas and placenta (Bose et al., 2014, Dalton et al., 2011, Kamar et al., 2013). Mortality ranges from 0.4% to 2% among immunocompetent individuals, although it may be as high as 20 to 30% among women in the second or third trimester of pregnancy.

Exposure to HEV infection is widespread with serological evidence of infection in a majority of individuals by the age of 20-40 (Izopet et al., 2015). Following acute infection with HEV any symptoms usually resolve and the virus is cleared in a little over 6-7 weeks (Tedder et al., 2016). However, chronic infection has been described in immunosuppressed individuals (Kamar et al., 2008, Haagsma et al., 2008). In recent years it has been recognized that a proportion of infected individuals suffer from neurological symptoms (Dalton et al., 2016).

In pigs, infections with HEV are asymptomatic. Viremia lasts for 1-2 weeks, with faecal viral shedding occurring 1-2 weeks after infection and persisting for up to 8 weeks. Hepatic changes are minimal; sites of extrahepatic replication have been identified in the small intestine, colon, and hepatic and mesenteric lymph nodes (Williams et al., 2001).

**Orthohepevirus B** includes isolates of avian hepatitis E virus detected in chickens (Haqshenas et al., 2001, Haqshenas et al., 2002, Payne et al., 1999, Huang et al., 2002). Infection in chickens is widespread with approximately 71% of chicken flocks and 30% of chickens in the United States positive for IgG antibodies to the virus (Huang et al., 2002). Infection has also been reported in wild birds (Zhang et al., 2016) and can be experimentally transmitted to turkeys but not monkeys, mice or pigs. The clinical disease in infected chickens has been referred to as big liver and spleen (BLS) disease, and hepatitis-splenomegaly (HS) syndrome. Disease in infected chickens is associated with increased mortality and decreased egg production. Virus replication occurs in the liver as well as extrahepatic tissues, including the gastrointestinal tract. **Orthohepevirus B** isolates have been divided into 4 genotypes that differ from each other by 18% in complete nucleotide sequence (Blic et al., 2009, Banyai et al., 2012). These genotypes have different geographical distributions; genotype 1 is restricted to Australia, genotypes 2 and 3 have been isolated in the USA and Europe, and genotype 4 in Hungary.

Serological reactivity to **Orthohepevirus C** has been detected in 25% of brown rats (Rattus norvegicus) from Germany, with virus detected in 10% of animals (Johns et al., 2010, Johns et al., 2012); a higher frequency of seroreactivity was reported in rats from the USA (Purcell et al., 2011). Genetically similar viruses have been detected in a variety of other host species; genotype C1 in rodents (Rattus sp, Bandicota indica).
and eulipotyphlids (musk shrew) while genotype C2 has been detected in mustelids (ferret and mink). Isolates of this species appear similar to those of Orthohepevirus A in EM structure and hepatotropism, although following experimental transmission liver enzymes levels were unaltered (Purcell et al., 2011, John et al., 2010a); other biological features are unknown.

Orthohepevirus D has a global distribution in bats with no evidence of transmission to humans (Drexler et al., 2012).

Antigenicity

Infected individuals typically develop antibodies directed against the capsid protein. Cross-reactivity has been demonstrated between the capsid proteins of isolates of Orthohepevirus A and Orthohepevirus B (Hagshenas et al., 2002), and antibodies raised against the capsid from dromedary camels, expressed in baculovirus, exhibited antigenic cross-reactivity against several Orthohepevirus A genotypes as well as rat and ferret HEVs (Zhou et al., 2015). Given the diversity of hosts from which hepeviruses have now been described, reports of serological reactivity to hepatitis E virus antigens in novel host species are difficult to interpret without corresponding virus sequence information. Vaccines capable of protecting against human infection with HEV have been produced using portions of the capsid protein (Purdy et al., 1993, Tsarev et al., 1997) and tested in Nepal (Shrestha et al., 2007), and China (Zhu et al., 2010). Xiamen Biotech has a vaccine that is licensed in China, but that is not currently licensed for use in other countries. A vaccine comprising part of the Orthohepevirus B capsid protein has been shown to protect chickens against infection (Guo et al., 2007).

Derivation of names

Orthohepevirus A: genus name followed by letter in order of discovery.

Species demarcation criteria

Four species have been demarcated on the basis of phylogenetic analysis of complete coding region nucleotide sequence and of partial amino acid sequence from the methyltransferase, helicase and RNA polymerase domains (Smith et al., 2014). The four species also have distinct host ranges; members of Orthohepevirus A have been isolated from mammals including humans (human hepatitis E virus), pigs (swine hepatitis E virus), deer, rabbits, camels, cattle, sheep, goats, mongooses, and bottlenose dolphins. Isolates of Orthohepevirus B are restricted to birds, primarily chickens, and have been allocated to several genotypes, although these are more closely related to each other than are those within Orthohepevirus A. Orthohepevirus C includes isolates from rodents, eulipotyphlids and mustelids. Bats remain the only source from which isolates of Orthohepevirus D have been reported. Related but distinct viruses have been reported from moose, fox, kestrel and little egret, and these are likely to represent additional species.

Member species

<table>
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<th>Isolate</th>
<th>Accession number/RefSeq number</th>
<th>Available sequence</th>
<th>Virus Abbrev.</th>
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Virus names, the choice of exemplar isolates, and virus abbreviations, are not official ICTV designations.

Related, unclassified viruses

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<td>little egret hepatitis E virus</td>
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Virus names and virus abbreviations are not official ICTV designations.

www.ictv.global/report/hepeviridae
**Genus: Piscihepevirus**

**Distinguishing features**

Piscihepeviruses have only been isolated from salmonid fish in North America. Genome organisation is similar to that of orthohepeviruses but piscihepeviruses are phylogenetically distinct (Smith et al., 2014, Batts et al., 2011).

**Virion**

**Morphology**

Virions have a diameter of 31 nm (Batts et al., 2011). Other properties of the virion have not been described.

**Genome organization and replication**

The 5′ untranslated region of cutthroat trout virus is longer (100 nt) than has been observed for orthohepeviruses (< 35nt). Three open reading frames are present, arranged as for other members of the Hepeviridae (Figure 4) except that ORF3 of cutthroat trout virus overlaps the central region of ORF2 rather than the 5′ terminus as is typical of orthohepeviruses. The protein encoded by ORF3 has a predicted isoelectric point of 11.8, similar to that encoded by hepatitis E virus (12.5), although this reading frame is not conserved in another partial genome sequence that covers this region (AF030878, haemorrhagic kidney syndrome virus). The capsid protein encoded by ORF2 has a predicted isoelectric point of 5.7 with acidic residues clustered at the COOH-terminus; this contrasts to the basic capsid protein encoded by ORF2 of orthohepeviruses (Batts et al., 2011).

**Biology**

Cutthroat trout virus has been detected in the ovarian fluids of several trout species and is widely distributed in the western United States (Hedrick et al., 1991). A similar virus has been detected in salmon in New Brunswick, Canada (Kibenge et al., 2000). Although not associated with disease, cutthroat trout virus produces a slow, focal type of cytopathic effect in the Chinook salmon embryo cell line CHSE-214 without destroying the monolayer. Virus can sometimes be re-isolated from infected animals after 4-6 weeks. A single complete genome sequence is available for cutthroat trout virus as well as one partial genome sequence (haemorrhagic kidney syndrome virus) and >30 partial helicase sequences; all of these virus sequences are closely related to that of cutthroat trout virus with nucleotide p-distances of < 0.08 and amino acid distances of < 0.06.

**Derivation of names**

**Pisci:** fish

**Species demarcation criteria**

Not applicable

**Member species**

<table>
<thead>
<tr>
<th>★ Exemplar isolate of the species</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Species</strong></td>
</tr>
<tr>
<td>Piscihepevirus</td>
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</tbody>
</table>

Virus names, the choice of exemplar isolates, and virus abbreviations, are not official ICTV designations.
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Disclaimer

The contributions to this report by Dr. Purdy do not necessarily represent the official position of the Centers for Disease Control and Prevention. It has not been formally disseminated by the Centers for Disease Control and Prevention/Agency for Toxic Substances and Disease Registry. It does not represent and should not be construed to represent any agency determination or policy.
**Resources: Hepeviridae**

**Websites:**

ICTV *Hepeviridae* Study Group Wiki: [https://talk.ictvonline.org/ictv_wikis/hepeviridae/w/sg_hepe](https://talk.ictvonline.org/ictv_wikis/hepeviridae/w/sg_hepe)

**Sequence alignments and tree files:**

**Figure 5.** *Hepeviridae*:

- Figure 5A. *Hepeviridae* - ORF1 - Alignment file (FASTA format)
- Figure 5A. *Hepeviridae* - ORF1 - Tree file (newick format)
- Figure 5B. *Hepeviridae* - ORF2 - Alignment file (FASTA format)
- Figure 5B. *Hepeviridae* - ORF2 - Tree file (newick format)
References: Hepeviridae


International Committee on Taxonomy of Viruses (ICTV) - www.ictv.global


Citation: Hepeviridae

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