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Global Water Pathogen Project part Three. Specific Excreted Pathogens: Environmental And epidemiology Aspects Hepatitis E

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Summary

Hepatitis E virus (HEV) is a small, non-enveloped, single-stranded, positive-sense RNA virus. The genome size is approximately 7.2 Kb. In 2004, Hepatitis E virus (HEV) was designated as the sole member of the genus Hepevirusin the family hepeviridae. Recently, consensus proposals were made to divide the family of Hepeviridae into the genera, Orthohepevirus and Piscihepevirus, of which the genus Orthohepevirus contains all mammalian and avian HEVs. The HEV genome has three open reading frames (ORFs): ORF1 encoding the non-structural polyprotein (nsp), ORF2 encoding the viral capsid protein and ORF3encoding a small regulatory phosphoprotein. Hepatocytes are assumed to be the primary target cells in which the virus replicates in the cytoplasm. Due to the lack of an efficient cell culture system, the mechanism by which HEV enters the cells and how the virions are released from the cells is not fully understood yet. In humans HEV infections often run an asymptomatic course but after an incubation period of 4 to 5 weeks HEV disease can present like anacute icteric viral hepatitis. Frequently observed symptoms include anorexia, nausea, jaundice, fever and abdominal pain. Mortality rates are generally under 0.5% but mayreach up to 25% in pregnant women for at least genotype 1.HEV is a main cause of epidemic hepatitis in developing countries and single cases of hepatitis in higher income countries. According to the World Health Organisation http://www.who.int/mediacentre/factsheets/fs280/en/)worldwide there are 20 million hepatitis E infections, over three million acute cases of hepatitis E and 57,000 hepatitis E-related deaths every year. Orthohepevirus variants most closely related to those infecting humans can be divided into at least four genotypes. Within genotypes 1 to 4 subdivisions into subtypes have been suggested by different authors, based on whole genome sequences or (partial) sequences derived from different open reading frames of the virus genome. An increasing

number of HEV sequences are reported and based on findings of divergent lineages in an increasing number of animal species deeper taxonomic groupings and genera have been proposed. Reported lineages of HEV strains within genotypes 1 and 2 are less divergent and seem to be more conserved compared to HEV strains from genotype 3 and 4. Genotypes 1 and 2 only seem to affect humans. Genotype 1 viruses are predominantly isolated from outbreaks and sporadic cases in Asia and Africa, whereas genotype 2 strains mainly have been observed in outbreaks in Mexico and Africa. Genotypes 3 and 4 are zoonotic and are observed in different animal species and sporadic human cases, worldwide for HEV genotype 3 and mainly in Asia for HEV genotype 4.HEV in infected individuals is shed enterically and ingestion of HEV particles is assumed to be the most important infection route. The virus may replicate in the intestinal tract but this has not been demonstrated to date. Via the portal vein, HEV can reach the liver, which is believed to be the main HEV target organ. Hepatocytes most likely are the main replication cell type. HEV attaches to host cells via specific high affinity receptors and enters the cytoplasm by clathrin-mediated endocytosis. Virtually nothing is known about the process by which HEV RNA enters target cells. It has been estimated that the infectivity titre of HEV for macaques is 1000-fold higher when inoculated intravenously compared to when it is ingested; however there is no information on the infectious dose for humans. Before the onset of disease symptoms up to 1088HEV genome copies per milligram faeces can be excreted for several days. In swine HEV RNA has also been detected in urine and it has been suggested that this may play a role in HEV transmission within this animal reservoir. Aerosol transmission of hepatitis E virus has not been reported. However if infectious HEV is excreted via urine, this route of transmission might be possible too. The concentration of viable virus in an environmental or food matter in oculum may be an important factor in the outcome of clinical hepatitis E infection. In developing countries HEV outbreaks of mainly genotypes1 or 2 are generally associated with unhygienic drinking water conditions. In tropical countries HEV epidemics from contaminated drinking water sources are common and affect mainly older children and young adults. Besides the direct infection from a water source horizontal transmission and vertical HEV transmission may play role in outbreaks. Large waterborne HEV outbreaks have recently been reported from Sudan, Chad, India and Pakistan. Improvement of hygienic conditions and decontamination of drinking water will be the most important preventive measurements in these regions. Food borne transmission of HEV was first demonstrated in clusters of Japanese patients after eating raw or undercooked meat from swine, wild boar or Sika deer. The genomic sequences of HEVs identified from the infected patients were identical to those recovered from the frozen leftover meat. Through either detection of HEV sequences and/or epidemiological study, more hepatitis E cases have been linked to the consumption of food products contaminated with the virus. This includes infection via locally produced meat products but also from game meat, processed pork, mussels, shellfish and other bivalves. Eating raw or undercooked meat products has been identified as a higher risk factor. Bivalves are known transmitters of enteric viruses and especially oysters are eaten worldwide as raw seafood. More recently HEV sequences have been detected on

soft fruits and vegetables, with irrigation water as the suspected contamination origin. For hepatitis E virus acquired locally in developed countries, it is generally very difficult to definitely identify the source of infection. Due to the long incubation period of up to 60 days, potentially implicated foods or environmental samples for analyses often will not be available for analysis.HEV can remain infectious at temperatures used in some cooking regimes, although inactivation by heating at 71degrees Celsius for 20 min has been demonstrated. HEV infectivity can also be reduced using chlorine (hypochlorite)as a disinfectant. Cell culture propagation of HEV has been difficult. Different cell types and culture methods have been used for the development of an efficient cell culture system which is needed to better assess HEV stability and inactivation and to elucidate the HEV transmission routes in susceptible populations

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