HEPATITIS A *VIRUS*

THE ORGANISM/TOXIN

Hepatitis A virus (HAV) causes hepatitis A, a severe food and waterborne disease that is primarily transmitted by the faecal/oral route. It is the only notifiable foodborne viral disease. It is a single-stranded RNA virus belonging to the Picornaviridae family and now classified in a distinct genus, Hepatovirus. HAV has a single serotype. Seven genotypes, four of which are found in humans, have been identified. These genotypes can be useful for tracing outbreak sources.

GROWTH AND ITS CONTROL

Growth: Hepatitis A virus is culturable in primate cell lines but wild type strains are difficult to culture and generally do not produce cell changes so are not easy to identify by culture alone. It will not grow in food or water.

Survival: HAV is very stable, shows high resistance to chemical and physical agents such as heat, acid and solvents and has been shown to survive in the environment for over 3 months.

Inactivation:

Temperature: HAV integrity and infectivity were retained after 60 min incubation at 60°C and the virus was only partially inactivated after 10-12 hours at 56°C. Infectivity was retained after 10 min at 80°C in 1M $MgCl_2$ but for only 5 min in phosphate buffered saline. HAV heat resistance is reported to be greater in foods and shellfish. HAV inoculated into oysters was not fully inactivated after heating in a can for 19 min at 60°C.

MAFF UK recommend a heat treatment of 1.5 min at 90°C to inactivate HAV in cockles

Under refrigeration and freezing conditions the virus remains intact and infectious for several years.

pH: Stable at acid pH. At pH 1.0 and 25°C, HAV retained high infectivity after 2 hours and was still infectious after 5 hours.

Drying: Infectious for >1 month at 25°C and 42% humidity. Greater resistance to inactivation at low humidity and temperatures.

Sanitisers/Disinfectants: (These products must be used as advised by the manufacturer).

Infectivity is decreased by exposure to 70% alcohol.

Resistant to chloroform, Freon, Arklone and 20% ether. Not inactivated by 300mg/l perchloroacetic acid or 1g/l chloramine at 20°C for 15min.

HAV is inactivated by:

Chlorine: 99.99% reduction in 6.5 min at pH6 and 49.6 min at pH10 (estimated Ct values under conditions described are 1.8 and 12.3 respectively.

Hypochlorite: 3-10mg/l at 20°C for 5 to15min.

Iodine: 3 mg/l for 5 min at 20°C

Formalin: 1:4000 for 72 hours at 37°C or 3% for 5 min at 25°C.

(N.B. The absence of a sanitiser/disinfectant from this section does not necessarily imply that it is ineffective).

Radiation: Inactivated by UV irradiation after 1 min exposure to 1.1W or 197μW/cm^2 for 4 min. Gamma irradiation has not been found to be effective for inactivation of HAV on fresh fruits and vegetables.

THE ILLNESS

Incubation: 2-6 weeks, average 28 days**.**

Symptons: Initially non-specific symptoms - fever, headache, fatigue, anorexia, nausea and vomiting, then viraemia, jaundice and hepatitis symptoms appear 1-2 weeks later. Virus is present in the blood at weeks 2- 4, and is shed in faeces (>10^6 particles/g) from the latter 2 weeks of the incubation period for up to 5 weeks. Jaundice is usually evident from weeks 4 to 7, and virus shedding generally continues throughout this period. Overall debility lasting several weeks is common and relapses may occur. Acute hepatitis is usually self-limiting but can occasionally cause fulminant disease that results in death. Estimated hospitalisation rate is 13%.

Condition: HAV infects hepatocytes, causes elevation of liver enzymes and inflammation of the liver. The cytotoxic T cell immune response destroys infected liver cells. Virus particles are released into the bile duct and excreted in faeces. The virus is believed to initially enter the liver via the bloodstream and it is not clear if intestinal replication occurs. The gastrointestinal tract is not a required route for infection.

Toxins: None produced in foods.

At Risk Groups: All age-groups. The disease is milder in young children under 6 years than older children and adults. Case-fatality risk increases with age so risks are higher for unexposed older people.

Long Term Effects: Overall case fatality rates in US are 0.3% (0.003 per 100,000) and range between 0.004% for 5-14 years to 2.7% in people > 49 years. HAV has not been associated with chronic liver disease.

Dose: not reported

NZ Incidence: 119 cases in 1999 and 107 cases in 2000; since 1986, generally < 350 cases per year. Most cases occur in Auckland and Northland. The NZ rate is low compared to Australia, USA and Canada but of comparable incidence to England and Wales. In 1999, 17.6% of NZ cases were hospitalised.

Treatment: Rest and an appropriate low-fat diet.
Post-exposure prophylaxis may be recommended for certain groups such as day-care centre staff, military and food industry staff. Prevention is possible via HAV vaccination for at-risk groups including overseas travellers and for foodhandlers when situations warrant it.

SOURCES

Human: Human faeces are the major reservoir.
Animal: HAV infects primates including chimps and some species of monkeys. Non-primates are also infected but disease is milder. Transmission via these hosts is unlikely.
Food: Contaminated bivalve shellfish, salads, fresh fruits and vegetables, water, and any manually prepared food products. HAV survived in crème-filled cookies for >1 day at 21ºC. Poor hygiene practices and poor sanitation are major risk factors. Presymptomatic foodhandlers excreting HAV pose a risk. Food is rarely available for analysis because of the long incubation period.
Environment: Survival of HAV in the environment (fresh and seawater, wastewater, marine sediments, soils and shellfish), is prolonged (>12 weeks) at 25°C. Excreta from infected humans may contaminate soil or water. Human faecal pollution from sewage discharges, septic tank leachates and boat discharges has caused contamination of shellfish beds, recreational water, irrigation water and drinking water.
Transmission Routes: The faecal/oral route is the established route of transmission and infection occurs following ingestion of faecally-contaminated food and water. Viral contamination of fresh fruits and salad vegetables through the global marketplace is becoming a significant route of exposure, especially in countries with low endemicity of hepatitis A. Person to person transmission is also important especially among young children in overcrowded living conditions, day-care centres or institutions. Parenteral transmission occurs in the drug-using population and via contaminated blood products.

OUTBREAKS AND INCIDENTS

Outbreaks: Most cases of disease are outbreak-related. Large outbreaks have been reported in developed countries where there is little immunity. Secondary transmission may account for 20% of cases in an outbreak.
New Zealand Outbreaks: Outbreaks of Hepatitis A traced to a common source are rare in NZ. Five outbreaks occurred in New Zealand in 1999 and 3 in 2000. In 1999, 54.7% of HAV cases were linked to consumption of contaminated food or water.
Foodhandling: 36 cases associated with contaminated food in a delicatessen prepared by infected owner/operator. Control point failure: infected foodhandler.
Overseas Outbreaks:
Strawberries/raspberries: US multistate outbreak of 189 cases associated with frozen strawberries harvested by an infected fruit picker. Two similar outbreaks of 4 and 24 cases in UK associated with frozen raspberries**.** Control point failure: fruit contaminated at harvest by infected pickers.
Bakery: 50 cases contracted hepatitis A from bread. Symptomatic baker continued to work. Control point failure: bread contaminated by foodhandler
Salads: 2 outbreaks (12 and 18 cases) in Finland associated with contaminated imported salad ingredients widely distributed from a central kitchen. Control point failure: imported contaminated food.
Shellfish: The largest recorded outbreak in China involved 290,000 cases associated with consumption of raw clams. US multistate outbreaks caused by faecal pollution of oyster beds by oyster harvesting boats (1993-1996). NSW outbreak of 440 cases in 1997 due to consumption of contaminated oysters. Control point failure: Faecal contamination of shellfish growing waters.
Water: Multistate outbreak of 5000 cases in US due to contaminated well water used in food processing. Control point failure: contaminated water used in food processing or for consumption.
Epidemiology: Worldwide HAV infection is very common. High incidence is linked to poor hygiene and sanitation standards. In developing countries 90% of children may be infected by 6 years of age, and most infections will be asymptomatic. In developed countries, HAV infection is more common in older age-groups, is more likely to be food or waterborne and result in large outbreaks. In New Zealand, few outbreaks are reported annually. Risk factors associated with these are generally contaminated foods, water, exposure to sewage or via person-to-person transmission.

ADEQUATE PROCESSING GUIDELINES

N.B. These guidelines have been derived from published information. Industry is advised to ensure that processing steps they are using are adequate to meet their particular food safety objectives.

	Internal temperature reached	Time
Shellfish	90^{o}C	1.5 min
Thoroughly wash all fruit and vegetables with potable water		
Ensure shellfish are harvested from approved shellfish gathering waters		
Avoid direct handling of food by infected food handlers		
Ensure all foodhandlers are trained in effective handwashing techniques (asymptomatic foodhandlers can cause infection)		
Boil, filter or chemically treat non-potable water for drinking		

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