Volume 3 Datasheets – Micro-organisms

Part 1.4: Viruses

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*Norwalk* virus: see caliciviruses.

SARS has been covered with the influenza viruses.

**Notes**

Enteric viruses are a combined group of those that infect the human gastrointestinal tract and are predominantly transmitted by the faecal–oral route. Well-known members of this group include the enteroviruses, astroviruses, enteric adenoviruses, orthoreoviruses, rotaviruses, caliciviruses and hepatitis A and E viruses. The enteric viruses cover a wide spectrum of viruses, members of which are a major cause of morbidity and mortality worldwide.

Viruses cannot grow in water or any other environmental niche – they can only grow inside a suitable host cell. All viruses contain genetic material (DNA or RNA) surrounded by a protein coat (capsid). Some viruses also have an envelope (membrane-like sac) which encloses the capsid. The envelope is derived from the lipid membrane of the host cell in which the virus grows, and also contains proteins made by the virus. The first step of the infection process for any virus involves structures on the surface of the virus recognising and binding to the surface of a suitable host cell. The lipids on the surface of enveloped viruses make them more prone to damage by chemicals (detergents, solvents, oxidising agents) than the protein capsid of non-enveloped viruses. Damage to the envelope very quickly renders the virus unable to infect new cells. Enveloped viruses are generally regarded as more “fragile” than non-enveloped viruses when they are in the environment, and lose infectivity more rapidly. The viruses that have been associated with drinking water outbreaks due to absent or inadequate disinfection are all non-enveloped viruses (adenovirus, rotavirus, norovirus, enterovirus, polio virus, hepatitis A and E viruses).

# Adenoviruses

### Maximum Acceptable Value

No MAV has been set for the presence of adenoviruses in drinking-water. If adenoviruses are sought specifically, they should not be detected. If detected, advice should be sought from the relevant health authority.

### Sources in drinking-water

Adenoviruses, in the family Adenoviridae, are classified into two genera: *Aviadenovirus* (avian hosts), and *Mastadenovirus* (mammal hosts). Adenoviruses are widespread in nature, infecting birds, mammals and amphibians. To date 53 antigenic types of human adenoviruses (HAdVs) have been described, with several types still unclassified (<http://www.ncbi.nlm.nih.gov>; genus *Mastadenovirus*). Adenoviruses consist of a double-stranded DNA genome in a non-enveloped icosahedral capsid with a diameter of about 80 nm and unique fibres.

HAdVs have been classified into seven groups (A–G), with the subgroups A–E growing well in cell culture. Serotypes 40 and 41 however are more fastidious than other types and generally require the use of polymerase chain reaction (PCR) techniques as well as cell culture amplification for their identification. Although the subgroup F (mainly serotypes 40 and 41) is a major cause of gastroenteritis worldwide, especially in children, little is known about their prevalence in water sources, due largely to the fact that they are not easily identified and therefore less likely to be looked for.

Adenoviruses are excreted in large numbers in human faeces and are known to occur in sewage, raw water sources and treated drinking-water worldwide. In view of their prevalence as an enteric pathogen and detection in water, contaminated drinking-water represents a likely but unconfirmed source of HAd infections.

A study of viruses in the raw water at four WTPs in the UK (DWI 2013) found that 74 percent of raw water samples were positive for adenoviruses. AdV was present in raw waters throughout the year and, whilst the water treatment process reduced the level of AdV by between 2 and 4 orders of magnitude, the virus was apparently able to persist through to the pre-chlorination stages. Around 20 percent of all pre-chlorination (final stage) samples were AdV positive although none of the isolates proved to be infective when assessed by ICC-PCR.

### Health considerations

HAdVs cause a wide range of infections with a spectrum of clinical manifestations. Different serotypes are associated with specific illnesses; for example, types 40 and 41 are the main causes of enteric illness. Adenoviruses are an important source of childhood gastroenteritis. In general, infants and children are most susceptible to adenovirus infections, and many infections are asymptomatic. High attack rates in outbreaks imply that infecting doses are low.

Some species of adenovirus cause pharyngitis, conjunctivitis, or pharyngoconjunctival fever. Several large outbreaks of pharyngoconjunctival fever have been associated with swimming pools (Cabelli 1978; Foy et al 1968; Di Angelo et al 1979).

Some of these viruses are endemic and exist as latent infections of the tonsils and adenoids. Others are usually associated with epidemics of acute respiratory and ocular disease in closed communities such as boarding schools and military camps. Whereas the relevance of adenoviruses to disease was initially determined by the isolation of serovars in cell cultures, later investigations with electron microscopy discovered specific serovars of adenoviruses that could not be cultivated. These include the only types (40 and 41) that have been associated with gastroenteritis and are among the viral agents associated with acute non-bacterial infectious gastroenteritis. These fastidious adenoviruses have been found in many parts of the world and are probably the second most important cause of gastroenteritis (after rotavirus) in young children. They tend to be endemic rather than epidemic, although outbreaks have occurred. Cytopathogenic adenovirus can be detected easily from all kinds of water, therefore waterborne transmission has also been suspected for the more fastidious HAdV varieties (Bitton et al 1986).

### New Zealand significance

HAdVs have been shown to occur in substantial numbers in raw water sources and drinking-water supplies (Chapron et al 2000). The USEPA has included HAdV as a pathogen likely to be in drinking-water or drinking-water sources on the preliminary contaminant candidate list (PCCL) in the Drinking-Water Contaminant Candidate List 3 – Draft in the Federal Register: 21 February 2008 (Volume 73, Number 35; <http://www.epa.gov>), suggesting that HAdV is likely to be of significance to New Zealand drinking-water, and in view of their detection in New Zealand fresh waters (McBride et al 2002, Till et al 2008), contaminated drinking-water represents a likely but unconfirmed source of HAdV infections. Little is known about the prevalence of adenoviruses in New Zealand drinking-water, in some part due to the difficulty of detection.

Results of a nationwide survey of 10 pathogens and indicators undertaken at 25 freshwater sites in New Zealand over a 15-month period found that human enteroviruses and adenoviruses were each found in about one third of samples but seldom occurred together, and no seasonal patterns were seen (Till et al 2008).

There have been no reported adenovirus outbreaks associated with Australian drinking water supplies (NHMRC, NRMMC 2011).

### Method of identification and detection

The concentration of the virus in treated drinking-water is likely to be low. The enteric adenoviruses, types 40 and 41, are difficult to grow in cell culture, whereas most other non-faecal types are culturable.

Detection requires concentration of the viruses from large volumes of water, often exceeding 1,000 litres. Identification in environmental samples is generally based on PCR techniques with or without initial cell culture amplification. The presence of the virus may also be detected by electron microscopy of faeces or of other samples. DWI (2013) discusses analytical techniques.

### Treatment of drinking-water

Conventional water treatment should result in water that is essentially virus-free, except where the intake water has a high virus load. This would occur where the intake water receives partially treated or untreated sewage. HAdVs are exceptionally resistant to some water treatment and disinfection processes, notably UV light irradiation; if using UV for control of protozoa, chlorination is recommended as well, see Chapter 7: Virus Compliance, Section 7.6. Under such circumstances *E. coli* may not be a suitable indicator of treatment processes (Grabow et al 2001). HAds have been detected in drinking-water supplies that met accepted specifications for treatment, disinfection and conventional indicator organisms. Because of the high resistance of the viruses to disinfection, *E. coli* (or, alternatively, thermotolerant coliforms) is not a reliable index of the presence/absence of HAdVs in drinking-water supplies.

### Derivation of Maximum Acceptable Value

The infectious dose for many viruses may be as low as one particle. Many tentative guidelines give figures of one particle per 1000 litres of water, but testing for viruses is difficult and results can be variable. Although no MAV has been established, *E. coli* is generally used as an indicator but may not be reliable depending on the integrity of source water.

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# Astroviruses

### Maximum Acceptable Value

No MAV has been set for the presence of astroviruses in drinking-water. If sought specifically, astroviruses should not be detected. If detected, advice should be sought from the relevant health authority.

### Sources to drinking-water

Human and animal strains of astroviruses are classified in the family Astroviridae (genus *Mamastrovirus*; <http://www.ncbi.nlm.nih.gov>). Astroviruses consist of a single-stranded RNA genome in a non-enveloped icosahedral capsid with a diameter of about 28 nm. In a proportion of the particles, a distinct surface star-shaped structure can be seen by electron microscopy. Eight different serotypes (1–8) of human astroviruses have been described.

Infected individuals generally excrete large numbers of astroviruses in faeces; hence the virus will be present in sewage. Human astroviruses (HAstV) have been detected in water sources and in drinking-water supplies (Pinto et al 2001).

### Health considerations

Human astroviruses have a low pathogenicity. They cause gastroenteritis, predominantly diarrhoea, mainly in children under five years of age or younger, although it has also been reported in adults. The illness is self-limiting, of short duration and has a peak incidence in the winter. The infectious dose may be <100 virus particles. Human astroviruses cause only a small proportion of reported gastroenteritis infections; however, since the illness is usually mild, many cases may go unreported. Seroprevalence studies showed that more than 80 percent of children between 5 and 10 years of age have antibodies against HAstVs.

HAstVs are transmitted by the faecal–oral route. Person-to-person spread is considered the most common route of transmission, and clusters of cases are seen in nurseries, child care centres, schools, paediatric wards, families, homes for the elderly, and military establishments. Ingestion of contaminated food or water could also be important.

### New Zealand significance

There has been no reported astrovirus identification in New Zealand environmental or drinking-water; however, there are few New Zealand data available suggesting there has been little investigation undertaken. The USEPA has included HAstV as a pathogen likely to be in drinking-water or drinking-water sources on the preliminary contaminant candidate list (PCCL) in the Drinking-Water Contaminant Candidate List 3 – Draft in the Federal Register: 21 February 2008 (Volume 73, Number 35; <http://www.epa.gov>). HAstV are transmitted via the faecal–oral route, suggesting that HAstV is likely to be of significance to New Zealand drinking-water too.

### Methods of identification and detection

The most commonly identified HAstV is serotype 1. Human astroviruses can be detected in environmental samples using PCR techniques with or without initial cell culture amplification.

### Treatment of drinking-water

Since the viruses are transmitted by the faecal-oral route, transmission by drinking-water seems likely, but has not been confirmed. Human astroviruses have been detected in drinking-water supplies that met accepted specifications for treatment, disinfection and conventional indicator organisms (Grabow et al 2001). Conventional water treatment should result in water that is essentially virus-free except where the source water has a heavy virus load. This would occur where the intake water receives partially treated or untreated sewage and thus control measures should focus on prevention of source water contamination by human waste. The effectiveness of treatment processes to remove astroviruses still requires validation. Owing to the higher resistance of the viruses to disinfection, *E. coli* (or, alternatively, thermotolerant coliforms) is not a reliable index of the presence/absence of HAstVs in drinking-water supplies.

Astroviruses survive heating for 30 min at 50ºC.

### Derivation of Maximum Acceptable Value

The infectious dose for many viruses may be as low as one particle. Many tentative guidelines give figures of one virus particle per 1,000 litres of water, but testing for viruses can be difficult and the results often variable. *E. coli* has generally been used as an indicator of water quality but owing to the higher resistance of viruses to disinfection it may not be a reliable indicator. The use of enteroviruses and F-RNA coliphages has been proposed as indicators but are not widely used.

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# Caliciviruses (Noroviruses/Norwalk)

### Maximum Acceptable Value

No MAV has been set for the presence of noroviruses in drinking-water. If sought specifically, noroviruses should not be detected. If detected, advice should be sought from the relevant health authority.

### Sources to drinking-water

The virus family Caliciviridae consists of four genera of single-stranded RNA viruses with a non-enveloped capsid (diameter 35–40 nm), which generally displays a typical surface morphology resembling cup-like structures. Human caliciviruses (HuCVs) include the genera *Norovirus* (Norwalk-type viruses) and *Sapovirus* (Sapparo-type viruses). *Sapovirus* spp. demonstrate the typical calicivirus morphology and are called classical caliciviruses. Noroviruses (NoVs) generally fail to reveal the typical morphology and were in the past referred to as small round-structured viruses. The remaining two genera of the family contain viruses that infect non-human vertebrates.

Noroviruses have been classified into five genogroups (G-I to G-V) of which GI, GII and GIV are known to infect humans (NZFSA 2010), with a many types still unclassified (<http://www.ncbi.nlm.nih.gov>; genus Norovirus). Human NoVs (HNoV) are found primarily in G-I and G-II. Noroviruses are excreted in the faeces of infected individuals and will therefore be present in domestic wastewater as well as faecally contaminated food and water, including drinking-water.

A prospective epidemiological study among city dwellers receiving a bacteriologically satisfactory drinking-water showed that the group receiving conventionally treated water had 25 percent more gastrointestinal symptoms than those receiving water treated by reverse osmosis (Payment et al 1991). The observed symptoms were compatible with infection caused by the Norwalk-type viruses, which were probably incompletely removed from the sewage-contaminated river water used as the source.

From 23 August to 7 September 2008, 1,699 cases of acute gastroenteritis were reported in Podgorica, Montenegro, population 136,000. The total size of the outbreak was estimated to be 10,000 to 15,000 corresponding to an attack rate of about 10 percent. Analyses of faecal samples identified six norovirus genotypes and occasionally other viruses. Multiple defects in the water distribution system were noted. These results suggest that the outbreak was caused by faecally contaminated municipal water, despite the water supply being chlorinated (Werber et al 2009).

A study of viruses in the raw water at four WTPs in the UK (DWI 2013) found NV was generally not detected in raw waters except during the winter months when 94 percent of the raw water samples were positive. Numbers were often so low it was not possible to measure removal rates.

Noroviruses are thought to have physicochemical stability and are relatively resistant to environmental challenge. They may retain their infectivity in cold water for up to a year and are able to tolerate temperatures up to 65°C for 30 minutes, pH ranges from 2–9, and free chlorine concentrations of 1 mg/L for 30 minutes. They can resist drying; infectious NoV were detected on environmental surfaces, including carpets, for up to 12 days after NoV outbreaks (NZFS 2010).

In a human volunteer study where human norovirus was spiked into groundwater and stored in the dark at room temperature, the virus was shown to be infective for at least 61 days and remained detectable for over three years (from DWI 2013).

### Health considerations

Human noroviruses (NoV) are now the most common cause of outbreaks of epidemic non-bacterial gastroenteritis worldwide (NZFSA 2010).

Electron microscopy has shown faecal specimens from people with non-bacterial gastroenteritis to contain many “small round viruses” ranging in size from 20 to 40 nm. The first of these described was the Norwalk agent, detected in volunteers fed filtered faecal suspension from an outbreak of winter vomiting disease. Morphologically similar viruses known as Hawaii, Wollan, Ditching, Parramatta, Snow Mountain, and Montgomery County agents were subsequently found. Definitive classification was delayed by failure to culture these viruses.

Norwalk virus infects the villi of the jejunum. Viruses are shed in stools during the first 72 hours after the onset of illness. The viruses are transmitted by the faecal-to-oral route. Water has been responsible for about 40 percent of all Norwalk-related outbreaks; this has included drinking-water supplies, recreational bathing water, and water used for growing shellfish (Goodman et al 1982). WHO (2004) states that the epidemiology of the disease indicates that person-to-person contact and the inhalation of contaminated aerosols and dust particles, as well as airborne particles of vomitus, are the most common routes of transmission. Drinking-water and a wide variety of foods contaminated with human faeces have been confirmed as major sources of exposure. Numerous outbreaks have been associated with contaminated drinking-water, ice, water on cruise ships and recreational waters. Shellfish harvested from sewage-contaminated waters have also been identified as a source of outbreaks.

Human caliciviruses are a major cause of acute viral gastroenteritis in all age groups. The Norwalk virus usually causes rapid, self-limiting epidemics of gastroenteritis that last 24–48 hours, and rarely more than three days (Baron et al 1982, Kaplan et al 1982). The symptoms are usually relatively mild. High attack rates in outbreaks indicate that the infecting dose is low (maybe as low as 10–100 virus particles). Infected individuals produce norovirus particles in high numbers, and concentrations in stools may reach 1010 particles per mL. The epidemics tend to be community-wide and involve school-age children, family contacts and adults. Roughly one third of such outbreaks can be attributed to the Norwalk virus. Infections result in delayed gastric emptying, nausea, vomiting, and abdominal cramps. About half of infected persons have associated diarrhoea, some have fever and chills. Some cases present with vomiting and no diarrhoea, a condition known as “winter vomiting disease”. A transient lymphopaenia also has been observed.

### New Zealand significance

There were 69 cases of waterborne gastroenteritis at the Mt Hutt Skifield in 1996 caused by Southampton virus, a Genogroup 1 norovirus (Greening et al 2001). Studies indicate that New Zealand has the same incidence of norovirus (from all sources) as the UK, where only one in every 1,500 cases is considered reported. In the first nine months of 2004, 164 New Zealand norovirus outbreaks have been reported to ESR, with the majority occurring in rest homes and hospitals. The virus has been found in sewage, environmental water and shellfish in New Zealand and represents a significant problem impacting upon food, recreation and drinking-water provision.

A norovirus outbreak at Cardrona was reported by Jack et al (2013). On 27 August 2012, Public Health South were notified that 11 out of a group of 15 diners at a hotel had become ill with gastroenteritis between 24 and 48 hours after dining and consuming tap water on 24 August. There were 53 cases that met the case definition out of 66 people contacted. This hotel was located near a ski resort which had separate water supply and sewage systems. Based on the findings of the investigation it is believed that this was a waterborne outbreak caused by contamination of the bore which provided drinking water to the hotel. Inspection showed the bore to be poorly situated in relation to several possible contamination sources. It was downstream of the effluent disposal field for the hotel septic system, in the drainage path of visible surface runoff from the disposal field from the neighbouring resort and close to the septic tank of a private residence. Operation of the chlorination pump for the drinking water supply was also faulty. As a consequence of this investigation at the hotel, shortcomings in water management were also found at the resort.

The USEPA has included caliciviruses as pathogens likely to be in drinking-water or drinking-water sources on the preliminary contaminant candidate list (PCCL) in the Drinking-Water Contaminant Candidate List 3 – Draft in the Federal Register: 21 February 2008 (Volume 73, Number 35; <http://www.epa.gov>), suggesting that HNoV is likely to be of significance to New Zealand drinking-water.

The infectious agent responsible for rabbit haemorrhagic disease (RHD) is rabbit calicivirus (RCV), genus [*Lagovirus*](http://en.wikipedia.org/wiki/Lagovirus) of the family [Caliciviridae](http://en.wikipedia.org/wiki/Caliciviridae). The virus infects only rabbits, and has been used in some countries to control rabbit populations, including New Zealand.

### Method of identification and detection

The concentration of the virus in treated drinking-water is likely to be low. Detection requires concentration of the viruses from large volumes of water, often exceeding 1,000 litres. In addition to electron microscopy the development of rapid, sensitive, specific reverse transcriptase polymerase chain reaction (PCR)-based molecular methods for detection means that these viruses can be identified but the expertise is not available routinely and is expensive. There is currently no routine culture of noroviruses. DWI (2013) discusses analytical techniques.

### Treatment of drinking-water

Conventional water treatment should result in water that is essentially virus-free except where the source water has a high virus load. This would occur where the intake water receives partially treated or untreated sewage and thus control measures should focus on prevention of source water contamination by human waste. The effectiveness of treatment processes to remove noroviruses still require validation. Owing to the higher resistance of the viruses to disinfection, *E. coli* (or, alternatively, thermotolerant coliforms) is not a reliable index of the presence/absence of HuCVs in drinking-water supplies.

### Derivation of Maximum Acceptable Value

The infectious dose for many viruses may be as low as one particle. Many tentative guidelines give figures of one particle per 1,000 litres of water, but testing for viruses is difficult and the result can be variable. *E. coli* has generally been used as an indicator of water quality but owing to the higher resistance of the viruses to disinfection it may not be a reliable indicator. The use of F-RNA coliphages has been proposed as indicators but are not widely used.

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# Coliphages

### Maximum Acceptable Value

No MAV has been set for the presence of phage indicators in drinking-water. If sought specifically, phages are indicative of faecal contamination and should not be detected.

DWI (2002) states: Bacteriophage have been suggested as a surrogate for human enteric viruses, due to similarities in size and behaviour through the water treatment process. Methods of analysis are relatively cheap and easy. It is probable that there may be some pressure from Europe to introduce monitoring for bacteriophage as many continental supplies are not chlorinated. For this reason they are considered to be of medium priority.

### Background

Bacteriophage (known typically as ‘phages’) are viruses that only infect bacteria as hosts for replication. Coliphage use *E. coli* and closely related species as hosts and are released by these hosts into the faeces of human and other warm blooded animals and are thus indicative of faecal contamination. Phages share many properties with human viruses, notably composition, morphology, structure and mode of replication. Consequently, coliphage are useful models or surrogates to assess the behaviour of enteric viruses in water environments and the sensitivity to treatment and disinfection processes and as an indicator of the possible presence of human enteric viruses. There is however no direct correlation between numbers of coliphage and numbers of enteric viruses. Phages that have been used as indicators of possible presence of enteric viruses include, particularly, somatic phages and F-specific phages. Both are listed by Payment et al (2003) as ‘suitable alternative’ microbial targets for various types of monitoring associated with water supplies, including assessment of disinfection and investigation of outbreaks.

Somatic phages infect host bacteria via cell wall (somatic) receptors and are common in both human and animal faeces. They replicate more frequently in the gastrointestinal tract of warm-blooded animals but can also replicate in water environments. Somatic coliphage consist of a wide range of phages (members of the phage families Myoviridae, Siphoviridae, Podoviridae and Microviridae) with a spectrum of morphological types. They are thought to be potentially useful as indicators of faecal pollution, but more research is required on their ecology to support wider such use (Payment et al 2003).

F-RNA coliphage initiate infection by attaching to fertility (F-, sex) fimbriae on *E. coli* hosts. These F-fimbriae are produced only by bacteria carrying the fertility (F-) plasmid. Since F-fimbriae are produced only in the logarithmic growth phase at temperatures above 30°C, F-RNA phages are not likely to replicate in environments other than the gastrointestinal tract of warm-blooded animals. F-RNA coliphages comprise a restricted group of closely related phages, which belong to the family Leviviridae, and consist of a single-stranded RNA genome and an icosahedral capsid that is morphologically similar to that of picornaviruses. F-RNA coliphage have been divided into serological types I–IV, which can be identified as genotypes by molecular techniques such as gene probe hybridisation. Members of groups I and IV have to date been found exclusively in animal faeces, and group III in human faeces. Group II phages have been detected in human faeces and no animal faeces other than about 28 percent of porcine faeces. This specificity, which is not fully understood, offers a potential tool to distinguish between faecal pollution of human and animal origin under certain conditions and limitations. For reasons that are not yet clear they have been reported to appear to multiply during sewage holding and treatment. Despite that, F-RNA coliphages provide a more specific index of faecal pollution than somatic phages.

Coliphage used in water quality assessment are divided into the major groups of somatic coliphage and F-RNA coliphage. Differences between the two groups include the route of infection. The DNA-containing tailed coliphage (T type) and RNA‑containing phages that infect via the F-pili (sex factor) (F-RNA coliphages) have been the most used. The F-RNA phages are similar in size, shape and composition to many enteric viruses, and have been proposed as an adequate model for enteric viruses (Havelaar et al 1993).

### Sources to drinking-water

Phages may be present in raw water due to its faecal contamination by humans or animals.

Coliphage are excreted by humans and animals in relatively low numbers. As a result of their respective modes of replication and host specificity, somatic coliphage are generally excreted by most humans and animals, whereas F-RNA coliphage are excreted by a variable and generally lower percentage of humans and animals. Available data indicate that in some communities, F-RNA phages are detectable in 10 percent of human, 45 percent of bovine, 60 percent of porcine and 70 percent of poultry faecal specimens. Somatic coliphage have been found to generally outnumber F-RNA phages in water environments by a factor of about 5 and cytopathogenic human viruses by a factor of about 500, although these ratios vary considerably. Sewage contains somatic coliphage in numbers of the order of 106 to 108 per litre; in one study, slaughterhouse wastewater was found to contain somatic coliphage in numbers up to 1010 per litre. There are indications that they may multiply in sewage, and somatic coliphage may multiply in natural water environments using saprophytic hosts. Somatic phages and F-RNA phages have been detected in numbers up to 105 per litre in lake and river water.

### Health considerations

Phage indicators, in common with viruses generally, are very host-specific. Phages are not pathogenic to people, ie, are not a health risk to humans, but have been used to indicate the presence of faecal contamination of waters. Coliphage cannot be absolutely relied upon as an index for enteric viruses. This has been confirmed by the isolation of enteric viruses from treated and disinfected drinking-water supplies that yielded negative results in conventional tests for coliphage. Due to the limitations of coliphage, they are best used in laboratory investigations, pilot trials and possibly validation testing. They are not suitable for operational or verification (including surveillance) monitoring.

### New Zealand significance

There is little information of studies of phage concentrations in New Zealand drinking water supplies. However phages are potentially valuable indicators of treatment efficiency for removal of viruses. Both somatic and F-RNA phages have been used in New Zealand for studies of disinfection of wastewaters and faecally-contaminated waters (eg, Sinton et al 2002).

Results of a nationwide survey of 10 pathogens and indicators undertaken at 25 freshwater sites in New Zealand over a 15-month period found somatic coliphage being detected in 89 percent of all samples, tending to have the highest levels in summer/autumn (Till et al 2008). F-RNA phages were found in 52 percent of samples, levels tended to be elevated in winter.

### Method of identification and detection

Phages are usually determined by direct quantitative plaque assays, following Adams (1959). Soft agar is mixed with the water to be tested and a culture of a specific host bacterium. This mixture is poured on to the top of agar in a petri dish, and the plaques caused by lysing of host cells near the sites of phage virions, are counted the following day. Specific methods, including references and host cell specifications, are given in Koster et al (2003).

Somatic coliphages are detectable by relatively simple and inexpensive plaque assays, which yield results within 24 hours. Plaque assays for F-RNA coliphage are not quite as simple, because the culture of host bacteria has to be in the logarithmic growth phase at a temperature above 30°C to ensure that F-fimbriae are present. Plaque assays using large petri dishes have been designed for the quantitative enumeration of plaques in 100 mL samples, and P/A tests have been developed for volumes of water of 500 mL or more.

### Treatment of drinking-water

Conventional water treatment should result in water that is essentially virus-free, and therefore free of phage indicators, except where the intake water has suffered recent faecal contamination. The presence of coliphage in drinking-water indicates shortcomings in treatment and disinfection processes designed to remove enteric viruses. The absence of coliphage from treated drinking-water supplies does not confirm the absence of pathogens such as enteric viruses and protozoal parasites.

### Derivation of Maximum Acceptable Value

Not applicable.

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# Enteroviruses

### Maximum Acceptable Value

No MAV value has been set for the presence of enteroviruses in drinking-water. If enteroviruses are sought specifically, they should not be detected. If detected, advice should be sought from the relevant health authority.

### Sources to drinking-water

Enteroviruses (EnV) – sometimes called picornaviruses, classified in the family Picornaviridae, are widespread in nature, infecting most vertebrates, with humans types including enterovirus (types A–D) with variant names such as poliovirus types 1–3, coxsackievirus types B1–B6, and echovirus types 1–33, and the numbered enterovirus types EV68–EV73 (<http://www.ncbi.nlm.nih.gov>). Members of the genus are collectively referred to as enteroviruses. Other species of the genus infect animals other than humans, eg, the bovine group of enteroviruses. Enteroviruses are among the smallest known viruses and consist of a single-stranded RNA genome in a non-enveloped icosahedral capsid with a diameter of 20–30 nm.

The viruses are shed in the faeces of infected individuals. They occur in water either through faecal contamination or by discharge of sewage effluents (Dahling 1989). Enteroviruses are ubiquitous and as such are a good indicator of human virus contamination of water, their presence in water is seasonal but their presence in sewage is stable and all the year round. Among the types of viruses detectable by conventional cell culture isolation, enteroviruses are the most numerous in sewage, water resources and treated drinking-water supplies.

They are spread by the faecal-oral route and, while waterborne transmission is probable, it has not been proven. The part played by low-level transmission has also been suspected but not proven. There is a suggestion that small numbers of viruses present intermittently or continuously in drinking-water cause symptomless infections, and these spread by person-to-person contact to cause outbreaks of disease that have no apparent connection with water.

The virus can also be spread on unwashed foods, particularly in areas where raw or treated sewage is used as fertiliser or irrigation water, or it may be transmitted on the feet of vectors such as houseflies. Infants, with their faeces contained in diapers, are also a major route of dissemination, particularly in day-care centres.

### Health considerations

Enteroviruses have a world-wide distribution. Most major epidemics within temperate climates occur during the late summer months, whereas in the tropics, disease can occur throughout the year. Enteroviruses are one of the most common causes of human infections. They have been estimated to cause about 30 million infections in the USA each year.

The enteroviruses cause a wide range of diseases including sore throat, rashes, aseptic meningitis, gastrointestinal symptoms, paralysis, cardiac symptoms, and conjunctivitis (Bitton 1986; Rao 1986). WHO (2004) states that the spectrum of diseases caused by enteroviruses is broad and ranges from a mild febrile illness to myocarditis, meningoencephalitis, poliomyelitis, herpangina, hand-foot-and mouth disease and neonatal multi-organ failure. They are generally thought not to cause gastroenteritis despite being transmitted by the faecal-oral route and excreted in faeces. The persistence of the viruses in chronic conditions such as polymyositis, dilated cardiomyopathy and chronic fatigue syndrome has been described.

Most infections, however, are mild or even symptomless. Serious or clinical disease occurs in between 1 in 100 and 1 in 1,000 infections.

Person-to-person contact and inhalation of airborne viruses or viruses in respiratory droplets are considered to be the predominant routes of transmission of enteroviruses in communities. Transmission from drinking-water could also be important, but this has not yet been confirmed. Waterborne transmission of enteroviruses (coxsackievirus A16 and B5) has been epidemiologically confirmed for only two outbreaks, and these were associated with children bathing in lake water in the 1970s.

### New Zealand significance

Enteroviruses have not been reported as detected in New Zealand drinking-water. It is probable, however, that water has been the source of many outbreaks in New Zealand, but environmental investigations are lacking to inform us of the epidemiology of enterovirus outbreaks recorded. They have been detected in raw water sources and drinking-water in many other countries, both developed and developing. In view of their prevalence, drinking-water represents a likely, although unconfirmed, source of enterovirus infection. The limited knowledge on the role of waterborne transmission could be related to a number of factors, including the wide range of clinical manifestations, frequent asymptomatic infection, and the diversity of serotypes and the frequency of person-to-person spread. Overseas, enteroviruses have been detected in drinking-water supplies that met accepted specifications for treatment, disinfection and conventional indicator organisms.

The USEPA has included enteroviruses as pathogens likely to be in drinking-water or drinking-water sources on the preliminary contaminant candidate list (PCCL) in the Drinking-Water Contaminant Candidate List 3 – Draft in the Federal Register: 21 February 2008 (Volume 73, Number 35; <http://www.epa.gov>), suggesting that human EnV is likely to be of significance to New Zealand drinking-water, and in view of their detection in New Zealand fresh waters (McBride et al 2002; Till et al 2008), contaminated drinking-water represents a likely but unconfirmed source of HEnV infections.

Results of a nationwide survey of 10 pathogens and indicators undertaken at 25 freshwater sites in New Zealand over a 15-month period found that human enteroviruses and adenoviruses were each found in about one third of samples but seldom occurred together, and no seasonal patterns were seen (Till et al 2008).

### Method of identification and detection

The concentration of the virus in treated drinking-water is likely to be low. Detection requires concentration of the virus from large volumes of water, often exceeding 1,000 litres. The final volume of the concentrate should be as small as possible. This can then be inoculated into cell cultures. The infected cells form plaques, and from the number of these the concentration of virus and types, using immunological procedures, can be calculated in the original sample.

The viruses may also be detected using electron microscopy of faeces, or other samples.

### Treatment of drinking-water

Conventional water treatment should result in water that is essentially virus-free except where the source water has a virus load. This would occur where the intake water receives partially treated or untreated sewage and thus control measures should focus on prevention of source water contamination by human waste. The effectiveness of treatment processes to remove enteroviruses still requires validation. Enteroviruses have been detected in drinking-water supplies that met accepted specifications for treatment, disinfection and conventional indicator organisms. Owing to the higher resistance of the viruses to disinfection, *E. coli* (or, alternatively, thermotolerant coliforms) is not a reliable index of the presence/absence of enteroviruses in drinking-water supplies.

### Derivation of Maximum Acceptable Value

The infectious dose for many viruses may be as low as one particle. Many tentative guidelines give figures of one particle per 1,000 litres of water, but testing for viruses is difficult and the results can be variable. *E. coli* has generally been used as an indicator of water quality but owing to the higher resistance of the viruses to disinfection it may not be a reliable indicator. The use of enteroviruses and F-RNA coliphages has been proposed and used as indicators, but are not widely used.

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# Hepatitis viruses

### Maximum Acceptable Value

No MAV has been set for the presence of hepatitis viruses in drinking-water. If hepatitis viruses are sought specifically, they should not be detected. If detected, advice should be sought from the relevant health authority.

### Sources to drinking-water

Hepatitis, a broad term for inflammation of the liver, has a number of infectious and non-infectious causes. Two of the viruses that cause hepatitis (hepatitis A and E) are excreted in faecal material of infected people and can be transmitted via water and food.

Hepatitis A virus (HAV) is the only species of the genus *Hepatovirus* in the family Picornaviridae. The virus shares basic structural and morphological features with other members of the family, as described for enteroviruses. HAV consists of only one clearly defined serotype. Human and simian HAVs are genotypically distinguishable. There is stronger epidemiological evidence for waterborne transmission of HAV than for any other virus. It will not grow in food or water. 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 three months.

Hepatitis E virus (HEV) is the only member of the Hepevirus family. HEV consists of a single-stranded RNA genome in a non-enveloped icosahedral capsid with a diameter of 27 to 34 nm. HEV shares properties with a number of viruses, and classification is a challenge. At one stage, HEV was classified as a member of the family Caliciviridae, but most recently it has been placed in a separate family called hepatitis E-like viruses (Hepevirus). There are indications of antigenic variation, and possibly even differences in serotypes of the virus. HEV is excreted in faeces of infected people, and the virus has been detected in raw and treated sewage. Contaminated water has been associated with very large outbreaks. HEV is distinctive, in that it is the only enteric virus with a meaningful animal reservoir, including domestic animals, particularly pigs, as well as cattle, goats and even rodents. The lower level of person-to-person spread suggests that faecally polluted water could play a much more important role in the spread of HEV than of HAV. Waterborne outbreaks involving thousands of cases are on record. These include one outbreak in 1954 with approximately 40,000 cases in Delhi, India; one with more than 100,000 cases in 1986–1988 in the Xinjiang Uighar region of China; and one in 1991 with some 79,000 cases in Kanpur, India. WHO (2017) states that every year, there are an estimated 20 million HEV infections worldwide, leading to an estimated 3.3 million symptomatic cases of hepatitis E, with approximately 44 000 deaths in 2015 (accounting for 3.3 percent of the mortality due to viral hepatitis).

HAV and HEV are shed in the faeces of infected individuals, and occur in water either by faecal contamination or by discharge of sewage effluent. Although there are some unexplained features about hepatitis E, the viruses are spread by the faecal-oral route, and waterborne transmission has been proven, person-to-person transmission is uncommon (Gerba et al 1985; Belabbes et al 1985) (WHO 2004, 2017).

Epidemics can usually be traced to contaminated water or food (hepatitis A). Hepatitis A has been detected in polluted rivers and in drinking-water (Nedachin et al 1989). Several very large outbreaks of drinking-water transmitted hepatitis have been recognised in India, China, Morocco, the Commonwealth of Independent States (formerly the Soviet Union), and Algeria.

Hepatitis B, C and D are spread by contact with body fluids of infected individuals. There have been no reported cases of waterborne transmission.

### Health considerations

Hepatitis A virus (HAV) and enterically transmitted hepatitis E (also known as HEV) cause infections of the liver with a typical illness consisting of lassitude, anorexia, weakness, nausea, vomiting, headache, abdominal discomfort, fever, dark urine, and jaundice. Hepatitis, if mild, may require only rest and restricted activities for a week or two, but severe cases can be much more debilitating. In the case of hepatitis A, fatal cases are exceptional and chronic liver disease has not been shown to occur.

HAV is highly infectious, and the infecting dose is considered to be low. The virus causes the disease hepatitis A, commonly known as “infectious hepatitis.” Like other members of the group enteric viruses, HAV enters the gastrointestinal tract by ingestion, where it infects epithelial cells. From here, the virus enters the bloodstream and reaches the liver, where it may cause severe damage to liver cells. In as many as 90 percent of cases, particularly in children, there is little, if any, liver damage, and the infection passes without clinical symptoms and elicits lifelong immunity. In general, the severity of illness increases with age. After a relatively long incubation period of
28–30 days on average (range 15 to 50 days), there is a characteristic sudden onset of illness, including symptoms such as fever, malaise, nausea, anorexia, abdominal discomfort and eventually jaundice. Although mortality is generally less than 1 percent, repair of the liver damage is a slow process that may keep patients incapacitated for six weeks or longer. This has substantial burden of disease implications. Mortality is higher in those over 50 years of age.

HEV causes hepatitis that is in many respects similar to that caused by HAV. However, the incubation period tends to be longer (2–10 weeks, average 40 days), and infections typically have a mortality rate of up to 25 percent in pregnant women. In endemic regions, first infections are typically seen in young adults rather than young children. Despite evidence of antigenic variation, a single infection appears to provide lifelong immunity to HEV. Hepatitis E (HEV) was not recognised as a distinct human disease until 1980. HEV infection (fulminate hepatitis) induces a mortality rate of 20 percent among pregnant women in the third trimester (WHO 2017).

### New Zealand significance

Hepatitis viruses have not been reported as detected in New Zealand drinking-water, and there is little information on the occurrence of Hepatitis A or E in Australian drinking water supplies either. This may be because of the difficulties associated with detection and the limited number of studies carried out in this country. Internationally, the transmission of hepatitis A, and hepatitis E, by drinking-water supplies is well-established.

Infections related to hepatitis A, B, C, D and E are notifiable to the Medical Officer of Health.

The USEPA has included both HAV and HEV as pathogens likely to be in drinking-water or drinking-water sources on the preliminary contaminant candidate list (PCCL) in the Drinking-Water Contaminant Candidate List 3 – Draft in the Federal Register: 21 February 2008 (Volume 73, Number 35; <http://www.epa.gov>), suggesting that HAV and HEV are likely to be of significance to New Zealand drinking-water.

### Method of identification and detection

The concentration of the virus in treated water is likely to be low. Detection would require concentration of the virus from large volumes of water, often exceeding 1,000 litres. The final volume of the concentrate would have to be as small as possible. Detection by PCR techniques is possible but still only available in specialised laboratories. The implication that water supplies may be involved in outbreaks of hepatitis A or hepatitis E infection depends on epidemiological evidence as they cannot be readily detected or cultivated in conventional cell culture systems, and identification in environmental samples depends on PCR techniques.

Electron microscopy of faeces, or other samples, may be used to detect the presence of viruses.

### Treatment of drinking-water

Conventional water treatment should result in water that is essentially virus-free except where the intake water has a virus load. This would occur where the intake water receives partially treated or untreated sewage and thus control measures should focus on prevention of source water contamination by human effluent. The effectiveness of treatment processes to remove HAV and HEV still require validation. Owing to the higher resistance of the viruses to disinfection, *E. coli* (or, alternatively, thermotolerant coliforms) is not a reliable index of the presence/absence of HAV or HEV in drinking-water supplies. HAV is inactivated by chlorine: 99.99 percent reduction in 6.5 minutes at pH6 and 49.6 minutes at pH10 (estimated Ct values under conditions described are 1.8 and 12.3 respectively (ESR 2001).

### Derivation of the guidelines

The infectious dose for many viruses may be as low as one particle. Many tentative guidelines give figures of one particle per 1,000 litres of water, but testing for viruses is difficult and results can be variable. *E. coli* has generally used as an indicator of water quality but owing to the higher resistance of the viruses to disinfection it may not be a reliable indicator. The use of enteroviruses and F-RNA coliphages has been proposed as indicators but are not widely used.

Microbial agents (which includes enteric hepatitis viruses) are included in the [plan of work of the rolling revision](http://www.who.int/entity/water_sanitation_health/gdwqrevision/en/index.html) of the WHO *Guidelines for Drinking-water Quality*.

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# Influenza viruses

### Maximum Acceptable Value

No MAV has been set for the presence of influenza viruses in drinking-water. If influenza viruses are sought specifically, they should not be detected. If detected, advice should be sought from the relevant health authority.

### Sources to drinking-water

Wild waterfowl are considered the natural reservoir of all influenza A viruses. Most infected birds exhibit no symptoms.

Infected waterfowl carry avian influenza viruses in their gastrointestinal tract, where the viruses replicate. Birds infected with avian influenza virus shed large quantities of virus in their faeces as well as in their saliva and nasal secretions, without necessarily exhibiting any symptoms. Shedding occurs in the first two weeks of infection. The period of avian influenza infectivity in faeces and secretions depends on pH and temperature conditions, but generally four weeks after infection, avian influenza virus can no longer be detected. Ducks infected with the H5N1 virus have been found to shed the virus at high titres from the trachea as well as from the cloacae, with peak levels of virus shedding after three days.

Besides direct deposition of faeces into lake water by migratory waterfowl, it has been suggested that faecal waste from duck and chicken farms may spread to bodies of water via wind, surface runoff or possibly enter groundwater through disposal and composting of waste on poultry or duck farms.

The isolation of the H5N1 virus from the faeces of a child presenting with diarrhoea followed by seizures, coma and death suggests that the virus may be excreted by infected humans and can enter sewage in this manner. Swine influenza A infection (the H1N1 virus) also is frequently accompanied by gastrointestinal symptoms which means that H1N1 might enter the host via the gastrointestinal tract. Although called swine flu, there is no evidence to suggest that H1N1 Influenza 09 (Human Swine Influenza) can be transmitted to people through eating pork products. Swine Influenza has never been reported or detected in pigs in Australia.

Avian influenza virus has been isolated from unconcentrated water from six lakes in Canada where ducks gathered and deposited large amounts of faeces. Avian influenza viruses can persist for extended periods of time in water, although quantitative information on the subtype H5N1 is lacking. Related studies have shown the avian influenza subtype H3N6 resuspended in Mississippi River (USA) water was detected for up to 32 days at 4°C and was undetectable after four days at 22°C. The data showed a decrease of about 4 logarithmic units (LU) in 32 days at 4°C (T90, the time taken to eliminate 90 percent of the virus in the sample, was estimated to be eight days) and of more than 8 LU at 22°C (estimated T90 = 0.5 days). In a second study, which used five low-pathogenicity avian influenza viruses (H3N8, H4N6, H6N2, H12N5, and H10N7), infectivity of virus in distilled water (initial concentration 106 TCID50, or median tissue culture infective dose, per mL) was retained for up to 207 days at 17°C and 102 days at 28°C. The T90 ranged between 21 and 32 days at 17°C and between 5 and 17 days at 28°C, depending on the strain. In a study that showed a high level of positive water samples (23 percent) for a strain of influenza A virus in a lake where ducks were nesting, the proportion of positive samples remained high (14 percent) in the autumn after the ducks had left for migration, indicating that the virus is able to persist in water.

H5N1 typically persists in colder temperatures and produces outbreaks during the colder months of the year. However, recent (unpublished) studies mentioned above have shown that current H5N1 strains survive longer in faeces at warmer temperatures than previously circulating viruses, which may explain how the virus has resurfaced in summer months in Asia. In general, influenza A virus viability in natural water (fresh, brackish, seawater) decreases with increasing salinity and pH. One modelled system predicts that infectivity is potentially greatest in cooler, freshwater habitats ranging 7.4 to 7.8 pH.

Avian influenza viruses are known to persist for extended periods of time in water, depending on temperature, pH and salinity. However, information on the persistence of highly pathogenic avian influenza viruses, including H5N1 avian influenza virus, in water is lacking. In general, the avian influenza virus viability in natural water (fresh, brackish and seawater) decreases with increasing salinity and increasing pH above neutral.

Swine influenza A viruses can persist for extended periods of time in water depending on temperature, pH and salinity but information on environmental persistence of H1N1 in water is lacking.

### Health considerations

Animal influenza viruses are distinct from human seasonal influenza viruses and do not easily transmit between humans. However, zoonotic influenza viruses – animal influenza viruses that may occasionally infect humans through direct or indirect contact – can cause disease in humans ranging from a mild illness to death. Most swine influenza viruses do not cause disease in humans, but some countries have reported cases of human infection from certain swine influenza viruses. Close proximity to infected pigs or visiting locations where pigs are exhibited has been reported for most human cases, but some limited human-to-human transmission has occurred. Just like birds and pigs, other animals such as horses and dogs, can be infected with their own influenza viruses (canine influenza viruses, equine influenza viruses, etc).

The epizootic of the Avian influenza A/H5N1 virus that started affecting domestic and wild birds and humans in South-East Asia in mid-2003, and has since spread to the rest of Asia, Africa and Europe, is the largest and most severe outbreak on record. Previously, outbreaks of highly pathogenic avian influenza in poultry and wild birds were rare. Since December 2003, more than 50 countries in Africa, Asia, Europe and the Middle East have reported outbreaks of H5N1 avian influenza in poultry and/or wild birds. More than 10 countries have also reported human H5N1 influenza cases.

There are three types of influenza viruses: A, B and C.

Influenza A viruses exist in at least 16 H subtypes and 9 N subtypes. Only viruses of the H5 and H7 subtypes are known to cause the highly pathogenic form of the disease. However, not all viruses of the H5 and H7 subtypes are highly pathogenic and not all will cause severe disease in poultry.

Influenza type A viruses can infect humans, birds, pigs, horses, and other animals, but wild birds are the natural hosts for these viruses. Only influenza type A viruses can cause pandemics. On 11 June 2009, the [World Health Organization](http://www.who.int/csr/disease/swineflu/en/index.html) (WHO) signalled that a global pandemic of novel influenza A (H1N1) was underway by raising the worldwide pandemic alert level to [Phase 6](http://www.who.int/csr/disease/avian_influenza/phase/en/). At the time, more than 70 countries had reported cases of novel influenza A (H1N1) infection, and it has spread since.

Influenza B viruses (sometimes called seasonal flu) are usually found only in humans and generally are associated with less severe epidemics than influenza A viruses.

Influenza type C viruses cause mild illness in humans and are not a significant concern for human health.

Within influenza A viruses, there are many different subtypes, some of which have low pathogenicity and others that have high pathogenicity. Everyone is familiar with human influenza virus, the seasonal affliction that causes symptoms such as fever, cough, sore throat and headaches. Human influenza is caused by human influenza viruses that can be transmitted between humans.

The influenza virus has eight genes, the two most important being H or haemagglutinin, and N or neuraminidase, where H and N refer to certain proteins on the surface of the viruses that carry the viral receptor sites. H1N1 virus, for example, has an HA 1 protein and an NA 1 protein. Three influenza A subtypes: H1N1, H1N2 and H3N2 have caused major outbreaks in humans. Human influenza virus replicates primarily in the respiratory tract of humans.

Avian influenza or bird flu is less well known. Avian influenza is an infection caused by avian influenza viruses, with transmission normally occurring between birds. In contrast to human influenza, the virus replicates in the gastrointestinal tract. Less commonly, avian influenza has infected pigs, and on rare occasions, humans. The subtype of avian influenza A virus known as H5N1 is very contagious among birds and can cause significant mortality in some avian species. In the rare instances that the virus is transmitted from birds to humans, H5N1 can cause pneumonia, multi-organ failure and often death. As of 24 March 2006, there have been 186 cases of transmission to humans, with 105 fatalities reported to the WHO. New Zealand cases of highly pathogenic avian influenza are notifiable to the Medical Officer of Health.

The overriding concern with respect to the H5N1 virus is that it may change into a form that is highly infectious for humans and that spreads easily from person to person. This could mark the start of a global outbreak or pandemic. No-one will have immunity to the virus, because no-one will have been exposed to it or have developed antibodies.

Most cases of H5N1 infection in humans to date have occurred as a result of direct contact with poultry or with surfaces and objects contaminated by their faeces. A questionnaire (MAF 2012) showed that NZ poultry workers generally do not take many hygiene precautions when working with the birds, with gumboots being the only common protective measure; many of the workers also have close contact with water fowl. However, concern has recently been expressed about the potential for transmission of the virus to humans through water and sewage, although no definitive cases have been reported to-date.

The fact that waterfowl excrete influenza viruses into water does not confirm waterborne transmission between birds; nor does it offer an indication of the extent of the risk of infection to humans exposed to the water. Some other viruses are likewise excreted into water environments without being transmitted to a meaningful extent via that route.

The frequent occurrence of diarrhoea in infected humans and the detection of viral RNA in most faecal samples tested (and infectious virus in one faecal sample) suggest that H5N1 virus may replicate in the human gastrointestinal tract.

### Treatment of drinking-water

Based on this review (WHO 2006), water supplies receiving treatment as recommended in the WHO *Guidelines for Drinking Water Quality* are unlikely to pose a significant risk of infection even if infected waterfowl are present in source waters.

The efficacy of UV disinfection on different types of viruses is quite variable. For example, a UV dose of 40 mJ/cm2, which is sufficient to achieve compliance with the bacterial and protozoal criteria in the DWSNZ, will only inactivate adenoviruses to about 0.5 log (see Chapter 7 of the Guidelines). Similar studies on influenza viruses have not been done. Therefore, during periods of high incidence of influenza A, water supplies that receive animal or human wastes upstream of the intake should consider supplementing their UV disinfection with chlorination. Chapter 19 (Section 19.3.4) discusses the use of household bleach (eg, Janola, Chlorogene or equivalent) for disinfecting individual supplies.

Due to their structure, influenza viruses are relatively susceptible to chemical disinfectants, including oxidising agents such as chlorine. For effective disinfection of adequately pretreated water, there should be a residual concentration of free available chlorine of at least 0.5 mg/L after at least 30 minutes’ contact time at pH <8.0. They are also readily inactivated by heating, so boiling would also be effective. Recent studies have demonstrated that free chlorine levels recommended by CDC (1–3 mg/L for pools and 2–5 mg/L for spas) are adequate to disinfect avian influenza A (H5N1) virus. It is likely that other influenza viruses such as novel H1N1 virus would also be similarly disinfected by chlorine.

Recently published research (Lenes et al 2010) has shown that conventional water treatment and disinfection are sufficient for the effective removal and/or inactivation of influenza viruses from drinking water.

### SARS

Severe acute respiratory syndrome (SARS) is a serious form of pneumonia. Infection with the SARS virus causes [acute](http://www.ncbi.nlm.nih.gov/pubmedhealth/n/pmh_adam/A002215/) respiratory distress (severe breathing difficulty) and sometimes death. SARS is a dramatic example of how quickly world travel can spread a disease. It is also an example of how quickly a connected health system can respond to a new health threat. The 2003 outbreak had an estimated 8,000 cases and 750 deaths. Since 2004, there have not been any known cases of SARS reported anywhere in the world. It was eventually brought under control in July 2003.

World Health Organization (WHO) physician Dr. Carlo Urbani identified SARS as a new disease in February 2003. He diagnosed it in a 48-year-old businessman who had traveled from the Guangdong province of China, through Hong Kong, to Hanoi, Vietnam. The businessman and the doctor who first diagnosed SARS both died from the illness.

SARS (SARS-CoV) is caused by a member of the coronavirus family of viruses (the same family that can cause the [common cold](http://www.ncbi.nlm.nih.gov/pubmedhealth/n/pmh_adam/A000678/)). It is believed the epidemic started when the virus spread from small mammals in China. While the spread of droplets through close contact caused most of the early SARS cases, SARS might also spread by hands and other objects the droplets has touched. Airborne transmission is a real possibility in some cases. Live virus has even been found in the stool of people with SARS, where it has been shown to live for up to four days. The virus may be able to live for months or years when the temperature is below freezing (*Pub Med Health* 2011).

The SARS outbreak was the first major international health emergency in which the WHO was able to take full advantage of the Internet age. It was detected early because of recently established Web-based systems trawling for unusual health events. It occurred at a time when health authorities around the world were on the alert and ready to respond. History will determine the final impact on global health security of the decisions made 10 years ago. The evidence so far is good: SARS was contained, and countries now freely report suspected threats like avian influenza or Ebola and cull their animals accordingly. From *The New York Times Opinion Pages*, 14 March 2013.

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Further information is available at:

* WHO Avian influenza factsheet: <http://www.who.int/mediacentre/factsheets/avian_influenza/en/>
* WHO Avian influenza in humans: <http://www.who.int/influenza/human_animal_interface/avian_influenza/en/>
* WHO Influenza – Information resources: <http://www.who.int/influenza/resources/en/>
* WHO – Influenza at the Human-Animal Interface (HAI): <http://www.who.int/influenza/human_animal_interface/en/>
* WHO – Avian influenza: food safety issues: <http://www.who.int/foodsafety/areas_work/zoonose/avian/en/index1.html>
* FAO Avian Influenza – Food and Agriculture Organization of the United Nations: <http://www.fao.org/avianflu/en/index.html>
* WHO Avian Influenza – A(H7N9) Information: <http://who.int/influenza/human_animal_interface/influenza_h7n9/en/index.html>

# Rotavirus and Reovirus

### Maximum Acceptable Value

No MAV has been set for the presence of reoviridae in drinking-water. If sought specifically, reoviridae should not be detected. If detected, advice should be sought from the relevant health authority.

### Sources to drinking-water

The family Reoviridae contains two important genera, *Reovirus* and *Rotavirus*. These are among the most widespread of all viruses in nature. They are passed in the faeces of infected individuals and may find their way into water by faecal contamination or by discharge of sewage effluents (Basch et al 1988, Bates et al 1984, Dahling et al 1989).

Rotaviruses and orthoreoviruses have been detected in sewage, rivers and lakes, and treated drinking-water. Cases of infection tend to be sporadic but several large waterborne outbreaks have been reported. The human rotaviruses (HRVs) have considerable public health significance as a common cause of acute diarrhoea, particularly in young children. They infect and multiply in mature or differentiated enterocytes located on the villi of the duodenum and small intestine. They are excreted in large numbers with as many as 1011 virus particles per gram of faeces for approximately eight days after onset of symptoms. This implies that domestic sewage and any environments polluted with the human faeces are likely to contain large numbers of HRVs. Orthoreoviruses generally occur in wastewater in substantial numbers.

### Health considerations

*Rotavirus* and *Orthoreovirus* are the two genera of the family Reoviridae typically associated with human infection.

Members of the genus *Orthoreovirus* infect many humans, but they are typical “orphan viruses” and not associated with any meaningful disease.

Members of the genus *Rotavirus* consist of a segmented double-stranded RNA genome in a non-enveloped icosahedral capsid with a diameter of 50–65 nm. This capsid is surrounded by a double-layered shell, giving the virus the appearance of a wheel, hence the name rotavirus. The diameter of the entire virus is about 80 nm.

The genus *Rotavirus* is serologically divided into seven groups, A–G, each of which consists of a number of subgroups; some of these subgroups specifically infect humans, whereas others infect a wide spectrum of animals. Groups A–C are found in humans, with group A being the most important human pathogens.

Human rotaviruses (HRVs) are the most important single cause of infant death in the world, and they also cause gastroenteritis in the elderly. Typically, 50–60 percent of cases of acute gastroenteritis of hospitalised children throughout the world are caused by HRVs. The viruses infect cells in the villi of the small intestine, with disruption of sodium and glucose transport. Acute infection has an abrupt onset of severe watery diarrhoea with fever, abdominal pain and vomiting; dehydration and metabolic acidosis may develop, and the outcome may be fatal if the infection is not appropriately treated. The burden of disease of rotavirus infections is extremely high.

The para-rotaviruses have been responsible for major outbreaks in adults in China.

HRVs are transmitted by the faecal–oral route. Person-to-person transmission and the inhalation of airborne HRVs or aerosols containing the viruses would appear to play a much more important role than ingestion of contaminated food or water. This is confirmed by the spread of infections in children’s wards in hospitals, which takes place much faster than can be accounted for by the ingestion of food or water contaminated by the faeces of infected patients. The infectious dose may be <100 virus particles. The role of contaminated water in transmission is lower than expected, given the prevalence of HRV infections and presence in contaminated water. However, occasional waterborne and foodborne outbreaks have been described. Two large outbreaks in China in 1982–1983 were linked to contaminated water supplies.

### New Zealand significance

Reoviridae have not been detected in New Zealand drinking-water, and there is little information on the occurrence of rotavirus in Australian drinking water supplies either. This may be because of the difficulties associated with detection and the absence of studies carried out in this country. Rotaviruses are ubiquitous, infecting over 90 percent of all children up to three years of age in the USA. New Zealand, however, does not have a human rotavirus surveillance programme so the prevalence and rotovirus disease burden cannot be estimated.

The USEPA has included RoV as a pathogen likely to be in drinking-water or drinking-water sources on the preliminary contaminant candidate list (PCCL) in the Drinking-Water Contaminant Candidate List 3 – Draft in the Federal Register: 21 February 2008 (Volume 73, Number 35; <http://www.epa.gov>), suggesting that RoV is likely to be of significance to New Zealand drinking-water too.

### Method of identification and detection

The concentration of the virus in treated water is likely to be low. Detection requires concentration of the virus from large volumes of water, often exceeding 1,000 litres. The final volume of the concentrate should be as small as possible. This can then be inoculated into cell cultures. The infected cells form plaques, and from the number of these the concentration of virus in the original sample and typing can be calculated. There are a number of PCR-based detection methods available for environmental samples.

Electron microscopy of faeces, or other samples, may also be used to detect the presence of viruses.

### Treatment of drinking-water

Conventional water treatment should result in water that is essentially virus-free except where the intake water has a high virus load. UV disinfection at doses that satisfactorily inactivate bacteria and protozoa are sometimes inadequate for inactivation of viruses; dosing chlorine as well is recommended. This would occur where the intake water receives partially treated or untreated sewage and thus control measures should focus on prevention of source water contamination. In such cases other processes, such as some of the membrane technologies, may have to be used to ensure removal of the viruses (Gerba et al 1984). The effectiveness of treatment processes to remove human rotaviruses still requires validation.

### Derivation of Maximum Acceptable Value

The infectious dose for many viruses may be as low as one particle. Many tentative guidelines give figures of one particle per 1,000 litres of water, but testing for viruses is difficult and results can be variable. *E. coli* has been generally used as an indicator of water quality but owing to the higher resistance of the viruses to disinfection it may not be a reliable indicator. The use of enteroviruses and F-RNA coliphages has been proposed as indicators but are not widely used.

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