

Adenovirus control in small drinking water systems: risks and strategies

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ABSTRACT

Many small drinking water treatment systems disinfect using only ultraviolet light, which provides minimal adenovirus inactivation at common doses. There is uncertainty in jurisdictions around the world about whether such practices should be accepted by regulators, or whether there is a need to specify a treatment target for adenovirus. A preliminary objective of this study was to determine whether adequate adenovirus information exists to conduct a meaningful quantitative microbial risk assessment (QMRA). The study then applied the QMRA framework to draw conclusions about whether there is justification to regulate adenovirus, and to identify weaknesses in those conclusions. A thorough review revealed that the quality of data available to conduct the analysis was poor. Using the (limited) data available, the QMRA suggests that adenovirus may pose risks similar to other viruses that are regulated. A lack of information about the concentration of adenovirus in source waters makes it difficult to propose a compelling evidence-based argument either for or against the need to require adenovirus treatment. Furthermore, management of risk from waterborne adenovirus should take into account an endemic rate of infection from non-waterborne exposures that appears to obscure accepted levels of waterborne risk.

Key words | adenovirus, drinking, QMRA, risk, small, ultraviolet

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INTRODUCTION

The problem of whether to target adenovirus control in the design and regulation of drinking water disinfection systems has been discussed for a number of years, but many jurisdictions are still struggling with the issue. Part of the difficulty is that quantitative information about the multiple factors related to the risk of adenovirus in drinking water is sparse. The purpose of this paper is to consolidate and critically review quantitative adenovirus risk information, and then to provide recommendations about whether regulators could use such information for rule making. Methods to control adenovirus in very small systems, particularly in the absence of chlorine, are also reviewed.

BACKGROUND

Most jurisdictions in North America require that drinking water from surface sources or ground waters under the

direct influence of a surface source (GUDI) be treated to a level that provides a minimum of 2 or 3-log (99–99.9%) reduction of *Cryptosporidium* and *Giardia* (oo)cysts, and 4-log (99.99%) reduction of viruses (USEPA 1989; MOE 2006). The treatment requirements for protected ground waters are more variable, but often do not require any treatment, or may only require 2 to 4-log virus reduction on the assumption that protozoa or bacteria are too large to reach the aquifer (MOE 2006; USEPA 2006a).

Adenovirus is readily inactivated by chlorine at low CT values (chlorine concentration×contact time) (Baxter *et al.* 2007). However, very small and remote systems on their own water supply, such as campgrounds or rest stations, often do not use chlorine. Equipment requirements, safety concerns, footprint and the operational burden of metering a consumable reagent likely contribute to the appeal of alternatives. Chlorination may also be avoided by some private owners owing to individual perspectives on the health

risks of disinfection by-products as well as taste and odour. It is the authors' experience in Canada that ultraviolet light (UV) disinfection is by far the most common alternative disinfectant, with thousands of such systems in the province of Ontario alone.

Small UV systems that are approved by regulators for public drinking water in North America often must meet National Sanitation Foundation (NSF) 55 Class A criteria, which require a minimum UV dose of 40 mJ/cm² (NSF 2009). While this dose is sufficient to provide greater than 4-log inactivation of almost all known waterborne viruses, the various adenovirus serotypes have been shown to require low pressure UV doses in the order of 140–220 mJ/cm² for 4-log inactivation (Nwachuku *et al.* 2005). An NSF 55-approved UV reactor might therefore only achieve approximately 1-log adenovirus reduction. As such, there is concern about whether small water treatment systems employing UV alone at doses of 40 mJ/cm² or UV with micron-size prefiltration (which would not reliably remove viruses) should receive regulatory approval. This question has significant ramifications given the many thousands of UV systems already in place under such circumstances.

RATIONALE FOR THE EXISTING VIRUS REDUCTION REQUIREMENTS: DOES IT APPLY TO ADENOVIRUS?

When considering whether adenovirus should be regulated, it is necessary to first review the rationale behind existing virus regulations to ensure that adenovirus is reviewed in a consistent manner.

The first 4-log virus reduction treatment goal was implemented in the USA, where the Environmental Protection Agency (USEPA) in 1989 applied the requirement to the treatment of surface waters or ground waters under the direct influence of surface waters (USEPA 1989). The goal was based on observations that systems employing chemically assisted filtration and disinfection in practice routinely achieved at least 4-log virus reduction, and no waterborne viral disease had ever been implicated in such systems when functioning properly (USEPA 1987). The 4-log reduction target did not, therefore, represent a specific risk level. It is possible that as 4-log virus reduction resulted

in no observed illness, a less stringent virus reduction would provide the same result.

In 2006, the US EPA introduced the Ground Water Rule (GWR), which set a 4-log virus reduction target for ground waters at risk of faecal contamination. In developing the rule, it was calculated that a 4-log virus reduction when treating ground waters would lead to less than a 1×10^{-4} annual risk of infection by model viruses (rotavirus and enterovirus) for more than 99% of the population (USEPA 2006b). This was used to justify the 4-log reduction target. The work in developing the rule, however, did not explore different levels of virus reduction: costs and benefits were only calculated for 4-log virus reduction compared with maintaining the regulatory status quo (a mixture of sanitation and treatment requirements). While a 4-log reduction target was shown to be sufficiently conservative for the ground waters, there was no explicit analysis of whether a lower target would also be sufficient. Another US rule introduced in 2006 – the Long Term 2 Enhanced Surface Water Treatment Rule (LT2 Rule) – continued to use 4-log virus reduction as a target for surface or GUDI water treatment (USEPA 2006c). The LT2 Rule did not re-evaluate the 4-log requirement: it was directly maintained from the previous 1989 rule. The LT2 Rule did, however, lead to an important decision to use adenovirus inactivation kinetics in the USA as the basis for the doses required when using UV light disinfection for virus inactivation, requiring a UV dose of 186 mJ/cm² for 4-log virus inactivation credit (USEPA 2006d). The decision to use adenovirus for UV dose requirements was based on a purely qualitative analysis. The justification was an argument that adenovirus is found in water subject to faecal contamination, can be transmitted through drinking water, is a common cause of diarrhoeal illness, and that the disease can be severe in some cases. While logical, this approach did not specifically correlate 4-log adenovirus reduction to a desired level of infection risk, as was done for rotavirus and enterovirus in the GWR.

In summary, the information leading to these virus regulations never demonstrated that a 4-log reduction is the minimum necessary to provide public health protection: only that 4-log reduction is sufficient. A lower level of reduction could also be sufficient.

In Canada, a draft policy document was released in 2010 by Health Canada which serves to advise individual provinces on how to regulate viruses (Health Canada 2010). A quantitative microbial risk assessment (QMRA) calculated virus risk in drinking water following methods described by Havelaar & Melse (2003). An important element of the Health Canada approach is the accounting of severity of illness – its duration and effect on quality of life. Health Canada adopted the World Health Organization's drinking water standard that identifies the

maximum tolerable risk level as 1×10^{-6} DALYs (disability adjusted life year) per person per year, and determined the combination of virus concentrations in the source water coupled with different levels of treatment that would allow this risk level to be achieved. The QMRA calculations were based on a 'composite' virus, generally compiling worst-case characteristics of several different viral pathogens. The illness symptoms that were modelled included mild diarrhoea, severe diarrhoea, and death. A summary of the model assumptions is given in Table 1.

Table 1 | Quantitative microbial risk analysis comparing adenovirus with rotavirus and a 'composite virus' (line numbers on the left are used for reference in the text; data sources are listed in the footnotes)

| | | World Health Organization Drinking Water Target: 1×10^{-6} | | |
|-----------------------------------|--|---|---|--------------------------------------|
| Tolerable risk (DALY/person/year) | | Health Canada 'composite virus' ^a | Rotavirus ^b | Adenovirus |
| Disease burden | | | | |
| 1 | Death: outcome fraction | 0.0001 | 0.00015 | 0.0001 ^c |
| 2 | Death: severity weight | 1 | 1 | 1 |
| 3 | Death: life years lost | 41.52 yr | 80 | Assume 80 |
| 4 | Death: life years lost (DALY/case) | 4.15×10^{-3} | 1.2×10^{-2} | 8×10^{-3} |
| 5 | Mild diarrhoea: outcome fraction (duration) | 0.50 (7 days) | 0.975 (7 days) | Unknown |
| 6 | Mild diarrhoea: severity weight | 0.067 | 0.1 | 0.1 |
| 7 | Years lived with mild diarrhoea (DALY/case) | 6.43×10^{-4} | 1.9×10^{-3} | Unknown |
| 8 | Bloody diarrhoea: outcome fraction (duration) | 0.49 (7 days) | 0.025 (7 days) | Unknown |
| 9 | Bloody diarrhoea: severity weight | 0.39 | 0.23 | Assume 0.39 |
| 10 | Years lived with bloody diarrhoea (DALY/case) | 3.67×10^{-3} | 1.1×10^{-4} | Unknown |
| 11 | Total disease burden (DALY/case) | 8.46×10^{-3} | 1.4×10^{-2} | 8×10^{-3} +morbidity |
| Risk of illness | | | | |
| 12 | Proportion of population susceptible to infection | 100% ^a | All children <4 | Assume 100% |
| 13 | Proportion of illnesses developed per infection | 88% | 88% in <3 yr old | 47% ^d |
| 14 | Dose-response model ^{e,f} | $P = 1 - \left(1 + \frac{N_d}{0.4415}\right)^{-96.7}$ | $P = 1 - \left(1 + \frac{N_d}{0.4415}\right)^{-96.7}$ | $P = 1 - e^{-1523(N_d)}$ |
| 15 | P = probability of infection per year per person (1 L/day) | | | |
| 16 | N_d = number of organisms ingested per day (1 L/day) | | | |
| 17 | Viruses in source water requiring 4-log reduction to achieve tolerable risk (1×10^{-6} DALY/person/year) | 0.613 viruses/100 L | 0.375 viruses/100 L | 0.18 viruses/100 L (mortality alone) |

^aHealth Canada 2010.

^bHavelaar & Melse 2003. Analysis limited to children <4 yr old: older persons assumed to be immune.

^cBennett *et al.* 1987.

^dFox *et al.* 1977.

^eWard *et al.* 1986 (rotavirus dose-response model).

^fRose *et al.* 1996 (adenovirus dose-response model), based on data reported by Couch *et al.* (1966) using exposure to adenovirus 4 in aerosols.

The overall result of Health Canada's QMRA analysis was that when more than 0.62 'composite' viruses per 100 L are present, treatment must provide a minimum of 4-log reduction to meet the risk target of 1×10^{-6} DALY/person per year. This is a significant result: surface waters can be expected to routinely exceed this concentration of viruses (McGuire *et al.* 2002) and therefore it may be reasonable to require a minimum of 4-log virus reduction. Importantly, however, surveys of virus concentrations in wells, compiled by the USEPA, suggest that wells that do not show any evidence of faecal contamination (presence of coliform, etc.) may also have average infectious virus concentrations in the order of 0.1–1 virus/100 L (Abbaszadegan *et al.* 2003; USEPA 2006b). This implies that wells without clear evidence of faecal contamination might also require multiple-log virus reduction.

Both the EPA and Health Canada conclude, by different methods, that when model viruses (i.e. the representative virus used for risk calculations) are present at concentrations greater than about 1 per 100 L, a 4-log reduction (or more) is appropriate. Such model viruses, however, are 'worst-case' viruses: those presumed to be more dangerous than others. Logically, less dangerous viruses would require lower levels of treatment to achieve the same risk reduction. Given the unique resistance of adenovirus to UV, we undertook a quantitative risk analysis based specifically on adenovirus properties to see if: (1) adequate information existed to conduct a meaningful QMRA of adenovirus; and then (2) using whatever information existed, to determine if a similar level of treatment is warranted if adenovirus were present at a similar concentration to other regulated viruses.

ADENOVIRUS RISK ANALYSIS

There have been no QMRA analyses conducted to date to predict the disease burden associated with adenovirus in drinking water. Adenovirus illness rates (without consideration of severity of illness) have, however, been estimated in three previous studies (Crabtree *et al.* 1997; van Heerden *et al.* 2005; Mena & Gerba 2008). The general consensus of these studies was that when adenovirus is present in source water at concentrations in the range of 0.1–1 per 100 L,

4-log reduction is required to reduce the infection rate to less than 1×10^{-4} per person per year – the USEPA target. To advance the quantitative microbial risk analysis, however, illness severity rates should also be considered, such that the overall risk can be expressed in terms of a total burden of disease (DALYs per person per year).

There is little information relating adenovirus infection to the probability of experiencing symptoms of different severities. The literature contains mostly anecdotal reports that characterize the types of illness that may be caused by adenovirus, which usually include gastroenteritis, respiratory illness, and conjunctivitis, but may also target other organs such as the bladder and pancreas. The reader is referred to Mena & Gerba (2008) or Jiang (2006) for excellent summaries. The range of illnesses extends from very mild symptoms, to rare deaths in the case of (almost exclusively, but not entirely) infants (CDC 2001).

It is instructive to compare illness caused by adenovirus with illness caused by rotavirus and the 'composite' virus created by Health Canada, as the rotavirus and composite virus were used by the USA and Canada, respectively, to justify virus regulations (although rotavirus illness is typically limited only to gastroenteritis, in contrast to the greater variety of illnesses caused by adenovirus). A simple point-estimate QMRA analysis of rotavirus and composite virus disease is shown in Table 1. Some of the terminology and methods in Table 1 are explained in Table 2, but it is beyond the scope of this paper to explain QMRA procedures in detail. Instead, the reader is referred to Havelaar & Melse (2003) or Health Canada (2010).

The risk levels from rotavirus and the 'composite virus' are very similar: 4-log reduction through treatment requires that source water virus concentrations be in the order of 0.1–1 per 100 L to maintain a disease burden below 1×10^{-6} DALYs/person/year when considering the rates and severity of disease due to mild diarrhoea, severe diarrhoea, and death (refer to line 17 in Table 1). Perhaps more importantly, the data illustrate that half or more of the predicted disease burden is from the very rare instances of mortality (line four, the disease burden from mortality, as a fraction of line 11, the total disease burden). The remaining disease burden is due to mild and severe diarrhoea (lines seven and 10, respectively). This may be helpful information when assessing the risk from adenovirus. While

Table 2 | Explanation of selected terms and methods used in Table 1

| | |
|--|---|
| DALYs | Disability adjusted life year: a unit of measure for risk. A risk of 0.01 DALYs means a 1% risk of one year of life lost per person per year of exposure to that risk, or the illness <i>equivalent</i> of 1 yr of life lost. Severity weights (see below) are used to compare non-lethal illnesses (e.g. diarrhoea) with death |
| Disease burden | A measure of the expected DALYs per case of infection |
| Outcome fraction | The fraction of infections that lead to the specified outcome (e.g. death, or bloody diarrhoea) |
| Severity weight | An agreed-upon and arbitrary measure of the severity of a type of outcome relative to death (e.g. death = 1.0, mild diarrhoea = 0.067 which implies that 1 yr of life lost due to death is equivalent to living approximately 15 yr (= 1 yr ÷ 0.067) with mild diarrhoea) |
| Total disease burden = Line 4+Line 7+Line 10 (disease burden due to death plus both mild and severe diarrhoea) | |

there is little or no quantitative information available about most forms of adenovirus symptoms (mild diarrhoea, pneumonia, etc.), there is a reported adenovirus mortality rate in the USA of 0.0001 (Bennett *et al.* 1987) which is very similar to the rate for rotavirus and the composite virus (0.0001 or 0.00015). Furthermore, the rate of developing an illness upon infection by rotavirus, the composite virus, and adenovirus, is within the same order of magnitude (88 and 47% for rotavirus/composite virus and adenovirus, respectively; Table 1, line 13). This suggests that the burden of disease (i.e. DALYs) per infection caused by adenovirus due to mortality alone is in the same order of magnitude as that due to all rotavirus/composite virus symptoms (i.e. line four (adenovirus) vs. line 11 (rotavirus/composite virus)). When it is then considered that adenovirus can also cause symptoms beyond death and diarrhoea that are included in calculations in Table 1 (such as respiratory illnesses), in contrast to rotavirus which causes primarily gastroenteritis, it is perhaps conservative to conclude that an infection by adenovirus is at least as severe as an infection by rotavirus.

Taking this analysis one step further, the rate of infection experienced upon ingestion of a rotavirus or the composite virus is similar to the infection rate upon ingestion of an adenovirus to within approximately 10–20%, as can be calculated using the dose-response models shown in Table 1 (calculation not shown). This logic suggests that if adenovirus and rotavirus or the composite virus are present in a source water at similar concentrations, then the log reduction of adenovirus must be at least similar to the log reduction of rotavirus or the composite virus to achieve the same level of risk reduction based solely on mortality,

and may need to be greater to account for other mild or severe symptoms associated with adenovirus illness.

There are two caveats to this analysis, however, that deserve attention:

1. *Uncertainty in the data:* The assumptions used in the adenovirus risk model contain considerable uncertainty and simplifications. The most significant uncertainty is with the mortality rate of 0.0001 reported by Bennett *et al.* (1987). This data point appears in a table in the Bennett *et al.* paper that is derived from ‘experts in the various divisions of the [United States] Center for Infectious Diseases and the Center for Prevention Services’ in 1985. No further information is given about whether this mortality rate is based on numerical evidence, or personal judgment. In fact, this figure is very similar to the ‘overall virus mortality rate’ reported in the same document (17,000 estimated fatalities in the USA per year per 207,329,000 viral infections = 0.00009). Attempts to clarify this issue with the original authors of the paper were unsuccessful. It is possible that the adenovirus fatality rate, which is the controlling factor in assessing the overall adenovirus disease burden calculated here, was simply assumed to be the same as the fatality rate from viruses in general. If this is the case, and if the true adenovirus mortality rate is substantially different from 0.0001, then our conclusion that the disease burden due to adenovirus infection is similar to that of rotavirus or composite virus infection is correspondingly in error.

Other problems with this risk analysis involve the accuracy of using dose-response models based on

inhalation of adenovirus type 4 to represent illness rates from drinking water ingestion of other serotypes (see note 'f' in Table 1), the lack of consideration of secondary infection, the lack of consideration of vulnerable sub-populations, etc. It is beyond the scope of this discussion to address these issues, but these factors are likely less important than the accuracy of the adenovirus mortality rate, which controls the current risk analysis.

2. *Drinking water exposure to adenovirus vs. other routes of exposure:* The QMRA approach that was used as a template to assess adenovirus risk, as summarized in Table 1, may not be appropriate for this purpose. The goal of reducing the burden of disease due to waterborne adenovirus was selected as 1×10^{-6} DALYs per person per year, and was adapted from the World Health Organization's risk analysis protocol for drinking water (WHO 2008). The WHO protocol cautions against setting a strict risk target for a contaminant when the overall burden of disease from other sources of the contaminant is very high when compared with water. In the USA, the annual illness rate due to adenovirus for the population was estimated to be 4% (Bennett *et al.* 1987). Given that the vast majority of the population drinks chlorinated water which very rapidly inactivates adenovirus (Baxter *et al.* 2007; AWWA 2008), it can be assumed that almost all of this infection and illness is attributable to sources other than drinking water. As such, attempting to limit adenovirus disease from drinking water to a level of one in one million DALYs per person per year (or 1 in 10,000 extra infections per year) when the illness rate from other sources is in the range of 400 per 10,000 (i. e. 4%), may be, according to the WHO, an incorrect interpretation of its risk management protocol.

DISCUSSION OF THE QUANTITATIVE RISK ANALYSIS

The implications of the quantitative risk analysis for adenovirus, summarized in Table 1, can best be discussed in the separate contexts of surface waters and protected ground waters.

For protected ground waters, some jurisdictions do not require any form of treatment on the assumption that such water can be free from all faecal contamination (USEPA 2006a). Evidence, however, suggests that it may be difficult to identify such instances, as Abbaszadegan *et al.* (2003) detected enteric viruses in ground waters meeting common criteria for being free from faecal contamination. Other studies (e.g. Borchardt *et al.* 2003, 2004; Locas *et al.* 2007) have also reported enteric viruses, including adenovirus, in water that would normally be considered free from other indicators of faecal contamination. The issue of whether to allow any water to be delivered to the public without treatment is beyond the scope of this paper. Instead, the quantitative risk assessment model reported in Table 1 suggests that even a 'low' adenovirus concentration in the source water – arbitrarily selected as 0.005 per 100 L for the sake of this argument, which is one or two orders of magnitude lower than the concentration of *total* enteric viruses reported by Abbaszadegan *et al.* (2003) in their survey of protected ground water wells – would still require 2 or 3-log reduction to reduce infection rates or the burden of disease to the 1 in 10,000 or 1×10^{-6} DALY/person/year level, respectively. It is unknown whether the actual concentrations of adenovirus in ground waters would be higher or lower than our hypothesized 0.005 per 100 L, and without such data, an appropriate quantitative risk assessment cannot be completed. The value of this assessment, however, is to demonstrate that at plausible adenovirus concentrations in ground water, a health risk may exist at a level that would warrant treatment for adenovirus according to some regulatory precedence.

For surface or GUDI waters, it is assumed that faecal contamination occurs. To determine the amount of treatment required to control adenovirus through a QMRA approach, knowledge of the expected concentration in the source water is required. The sole study identified that has reported adenovirus concentrations in surface waters indicated average infectious concentrations ranging from approximately 0.01–0.5 per 100 L, based on 51 samples from two surface sources in South Africa (van Heerden *et al.* 2005). Many other non-quantitative studies have shown adenovirus to be ubiquitous in both surface and GUDI sources (e.g. Chapron *et al.* 2000; Jiang *et al.* 2001; Choi & Jiang 2005), with adenovirus in some cases detected

more frequently than rotavirus (Verheyen *et al.* 2009). The ubiquitous nature of adenovirus is to be expected, as most adenovirus serotypes from infected humans are shed faecally (Feigin *et al.* 2004), and can survive in the environment quite readily (Enriquez *et al.* 1995). Without better knowledge of the expected concentration of adenovirus in source waters, however, a quantitative risk assessment cannot be conducted. As with the case of protected ground waters, however, the value of this quantitative risk analysis is to demonstrate that plausible concentrations in source water might require significant log reductions to meet typical health targets.

The conclusion that treatment specifically targeting adenovirus reduction might be required based on realistic source water concentrations is contingent, however, on the reported 0.0001 mortality rate, which we question. If we choose to distrust this value, then we are left with no quantitative basis on which to propose treatment targets for adenovirus. In this case, it might be best to review historical treatment practices: is there evidence of a lack of disease in communities that did not treat adenovirus, but did control other pathogens? Such communities would likely be limited to communities employing only UV disinfection, perhaps with micron-size filtration. Public health reports from such situations could not be identified for this paper, but would offer valuable insight into the actual risk of adenovirus in drinking water. Such a retrospective study of public health records might be an alternate approach to resolving the adenovirus problem if quantitative risk assessment approaches such as taken here remain impractical because of a lack of data.

TREATMENT FOR ADENOVIRUS IN SMALL SYSTEMS

If regulators choose to set treatment requirements for adenovirus in small drinking water systems, the main options include chlorination, UV disinfection, and cartridge and/or membrane filtration.

Adenovirus is reported to be very sensitive to free chlorine. CT requirements for 2-log inactivation of adenovirus types 4, 40, and 41 have been reported to be in the order of 0.2 mg-min/L under worst-case chlorination conditions

(5 °C, pH 8–8.5) (Thurston-Enriquez *et al.* 2003; Baxter *et al.* 2007). Monochloramine, in contrast, inactivates adenovirus types 5 and 41 quite slowly with CT values of 300 mg-min/L required for 2-log inactivation (Baxter *et al.* 2007).

The application of chlorine instead of UV to control adenovirus in small systems should be considered with some caution. In very small systems, chlorine doses tend to be very high 'to be safe', and lead to elevated chlorination by-product concentrations. Regulators would have to weigh the uncertain risks associated with adenovirus against the more certain adverse health effects of chlorination by-products.

UV light at 254 nm (i.e. a low pressure lamp) requires a dose of 186 mJ/cm² for 4-log adenovirus inactivation (USEPA 2006d). Typical NSF 55 Class A UV systems are required to deliver a dose of 40 mJ/cm² under worst-case conditions (normally 70% cm⁻¹ UV transmittance, maximum flow rate). Under more normal operating conditions (e.g. UV transmittance >90% cm⁻¹), a much higher dose is actually delivered, but it is not possible to reliably measure such higher doses. It has been proposed that installing UV reactors in series would allow addition of the delivered doses, such that 4 or 5 reactors in series could provide 4-log adenovirus inactivation. A recent study, however, suggests that the total reduction equivalent dose from UV reactors in series can be less than the sum of the doses from each reactor (Ducoste & Alpert 2011). These differences can be due to inadequate mixing between each reactor in series.

Adenovirus types 2 and 40 have been shown to be more susceptible to polychromatic UV light from medium pressure lamps than the monochromatic light from low pressure lamps, with 4-log inactivation exhibited at medium pressure doses in the order of 40 mJ/cm² (Linden *et al.* 2007). It may be difficult, however, to develop medium pressure UV systems appropriate for small drinking water plants because of the difficulties inherent in monitoring the dose delivered by polychromatic lamps, and significant safety and reliability issues related to the much hotter medium pressure lamps. Furthermore, NSF does not have a protocol available to validate medium pressure UV systems. As such, small systems may be unable to use medium pressure UV lamps in the short term.

Cartridge and membrane filters come with a wide range of specifications. The most common point of use (POU) or point of entry (POE) cartridge filters have nominal pore sizes in the range of 1–5 µm, and as such, could not reliably remove viruses which are typically in tens or hundreds of nanometers in size. Challenge tests on new ultrafiltration (<0.01 µm), nanofiltration, and reverse osmosis membranes demonstrate several logs of virus removal, but there is some concern with the lack of ability to monitor virus removal on an ongoing basis. It is common, however, to reduce monitoring requirements for small systems.

CONCLUSIONS

This paper identifies important information gaps in our understanding of the risk of adenovirus in drinking water. In general, there is little compelling information available either for or against the need to regulate adenovirus treatment. The quantitative risk analysis, albeit simplistic due to the lack of data, does suggest that if a mortality rate of 0.0001 upon adenovirus infection is to be believed, then at plausible concentrations of adenovirus in both ground waters and surface waters, several logs of adenovirus reduction might be warranted. The exact level of log reduction required cannot be calculated without knowledge of the source water concentration – something that has very rarely been reported, and that is also likely to vary by orders of magnitude both spatially and temporally within a water source. This is a common challenge associated with using a QMRA approach to evaluating drinking water risks.

An alternative to a QMRA approach to deal rationally with the perceived risk of adenovirus in drinking water is to gather health data from communities that have not employed treatment that would control adenovirus. The authors are aware of communities in Europe that have used UV alone (with no chlorine) to treat ground water in community systems. A retrospective analysis of public health records in those communities could conceivably help to determine the need for adenovirus control.

While the inability to resolve this issue using QMRA may be disappointing, this study is valuable in that it informs regulators that, given the data currently available, they must resort to making a decision about adenovirus control in the

absence of adequate health risk information, using other decision-making methods.

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