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Assessment of public health risk associated with viral contamination in harvested urban stormwater for domestic applications

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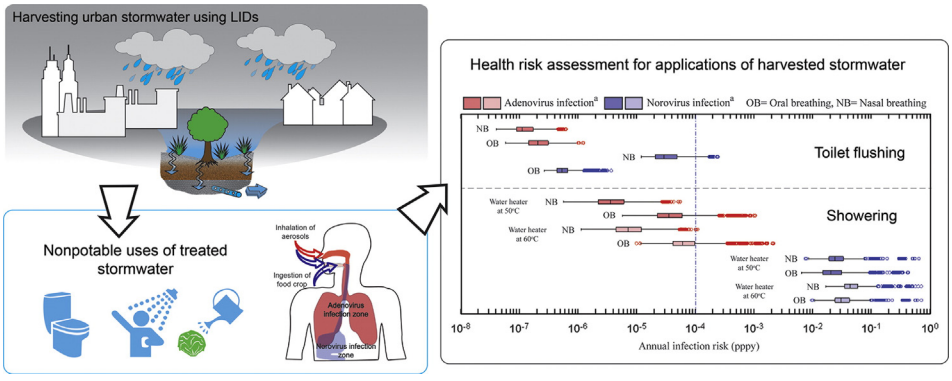
HIGHLIGHTS

- Human health risks for three non-potable uses of treated stormwater are modeled.
- Crop irrigation poses the highest risk, followed by showering and toilet-flushing.
- Only toilet-flushing is deemed acceptable based on the U.S. EPA risk benchmark.
- Both toilet-flushing and showering are within the WHO recommended disease burdens.

GRAPHICAL ABSTRACT



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ABSTRACT

Capturing stormwater is becoming a new standard for sustainable urban stormwater management, which can be used to supplement water supply portfolios in water-stressed cities. The key advantage of harvesting stormwater is to use low impact development (LID) systems for treatment to meet water quality requirement for non-potable uses. However, the lack of scientific studies to validate the safety of such practice has limited its adoption. Microbial hazards in stormwater, especially human viruses, represent the primary public health threat. Using adenovirus and norovirus as target pathogens, we investigated the viral health risk associated with a generic scenario of urban stormwater harvesting practice and its application for three non-potable uses: 1) toilet flushing, 2) showering, and 3) food-crop irrigation. The Quantitative Microbial Risk Assessment (QMRA) results showed that food-crop irrigation has the highest annual viral infection risk (median range: 6.8×10^{-4}–9.7×10^{-1} per-person-per-year or pppy), followed by showering (3.6×10^{-7}–4.3×10^{-2} pppy), and toilet flushing (1.1×10^{-7}–1.3×10^{-4} pppy). Disease burden of each stormwater use was ranked in the same order as its viral infection risk: food-crop irrigation > showering > toilet flushing. The median and 95th percentile risk values of toilet-flushing using treated stormwater are below U.S. EPA annual risk benchmark of $\leq 10^{-4}$ pppy, whereas the disease burdens of both toilet-flushing and showering are within the WHO recommended disease burdens of

Abbreviations: DALYs, disability adjusted life years; GI, genogroup I; GII, genogroup II; LID, low impact development; MNV, murine norovirus; NRMMC–EPHC–NHMRC, Natural Resource Management Ministerial Council–Environment Protection and Heritage Council–National Health and Medical Research Council; OSHA, Occupational Safety and Health Administration; PFU, plaque forming units; pppy, per-person-per-year; QMRA, Quantitative Microbial Risk Assessment; $TCID_{50}$, median tissue culture infectious dose; U.S. EPA, United States Environmental Protection Agency; WHO, World Health Organization; WSUD, water sensitive urban design.

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$\leq 10^{-6}$ DALYs pppy. However, the acceptability of showering risk interpreted based on the U.S. EPA and WHO benchmarks is in disagreement. These results confirm the safety of stormwater application in toilet flushing, but call for further research to fill the data gaps in risk modeling as well as risk benchmarks.
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1. Introduction

Sustainable urban stormwater management is emerging as one of the solutions to alleviate the negative impact of rapid urbanization. Stormwater harvesting systems are receiving attentions from the water sectors following the revived interest in rainwater harvesting in intermittently drought-ridden regions (Fletcher et al., 2008; Grant et al., 2013; Hatt et al., 2006). The rationale for harvesting stormwater for beneficial uses is to capture the excess stormwater before it contaminates the receiving water body and changes the stream hydrology, while providing a new source of water supply that may require less treatment than sewage for various non-potable uses. Development of stormwater harvesting systems as a water source, however, is often impeded by social and institutional barriers resulting from a complicated mix of risk perceptions by multiple stakeholders (Dobbie and Brown, 2012). While there is an increasing recognition that other associated risks such as technical, socio-economics, and environmental risks also play influential roles in risk management, public health risk has been the focal point of technical risk assessment that guides risk management within the water sector in developed countries.

Stormwater is water that is collected by storm drain systems without any engineered treatment, and can include urban runoff from irrigation, car washes, and rainwater that is intercepted by paved surface. In urban settings stormwater carries a large number of chemical and microbiological pollutants, which have a detrimental impact to coastal water quality (e.g., Ahn et al., 2005; Handler et al., 2006; Lipp et al., 2001). Stormwater collection systems are usually underground channels that are separated from—but often in close proximity to—sanitary sewer lines. Many of these systems in older cities suffer leakage, which results in unintended cross-contamination of the two types of water (Brownell et al., 2007; Jiang, 2006; Sidhu et al., 2012). A review of stormwater harvesting practices in Australia (Hatt et al., 2006) identified that most of the stormwater (in ~60% of the large-scale systems) collected using conventional urban drainage techniques such as gutters, pipes, and channels, is contaminated by sewage. In spite of the presence of contaminants, harvested stormwater should require less treatment than sewage if it is to be used for non-potable purposes, such as toilet-flushing, irrigation of lawns, car washing, and laundry. Sustainable urban water management systems, frequently termed low-impact development (LID) systems in the U.S. or water-sensitive urban design (WSUD) in Australia, are presumed to be able to provide passive treatment of stormwater that is needed for its safe non-potable uses with much less energy requirements than conventional water treatment technologies (Fletcher et al., 2013; Hatt et al., 2006). These systems include biofilters, rain gardens, bioswales and filter strips, as well as wetlands and ponds. Ultimately, the main concern of using harvested stormwater for household uses lies in the transmission of pathogens to humans, which may translate to disease outbreak in more severe cases.

Human-specific fecal waste markers have been detected in urban stormwater in the cities of U.S. (Sauer et al., 2011) and Australia (Sidhu et al., 2013, 2012; Tang et al., 2013), in which human enteric viruses generally pose the greatest threat to public health (Scallan et al., 2011). Of these, noroviruses' high potency to cause gastroenteritis (Lopman et al., 2011) and adenoviruses' ubiquitous presence in environmental waters (Jiang, 2006) have rendered them two of the most studied viruses. Adenoviruses, double stranded DNA viruses, contain 51 known serotypes. Illnesses associated with adenoviruses range from acute respiratory disease, pneumonia, conjunctivitis, and gastroenteritis, all of which could potentially be transmitted environmentally through non-potable uses of harvested stormwater (Arnone and Walling, 2007). Noroviruses are frequently reported as the leading cause of viral gastroenteritis outbreaks worldwide, with some literature estimating that they account for ~50% of all gastroenteritis cases (Lopman et al., 2012; Patel et al., 2009).

Direct measurements of viral concentration in stormwater, however, are sparse due to the difficulties facing the quantification technologies, which are often plagued by poor recoveries in environmental water and inhibitory effects of PCR used for detecting viral genomes (Rajal et al., 2007). Our previous work has shown that viruses were more frequently detected in the receiving water affected by urban stormwater flow than directly from the stormwater itself due to the PCR inhibition and co-concentrated suspended solids (Choi and Jiang, 2005; Jiang et al., 2001; Jiang and Chu, 2004; Jiang et al., 2007). In fact, a molecular quantitative analysis of human viruses in stormwater conducted by Rajal et al. (2007) yielded results that effectively comprise of non-detects only.

These challenges in enumerating enteric viruses in stormwater have translated to a very poor understanding of removal by basic treatment processes. In fact, the only removal efficacy study for stormwater treated through a LID system (biofilters in this case) is for the removal efficiency of an indicator virus, F-RNA coliphage (Li et al., 2012), and not human-pathogenic viruses.

Consideration of risk associated with stormwater reuse needs to look beyond the water quality itself to include the various ways in which the water is likely to be used. Toilet flushing, showering, and food-crop irrigation are three likely uses, yet they represent distinctly different pathogen-human transmission routes and different infection sites (respiratory vs. intestinal system). Variation within such systems can also be significant. For instance, flush energy associated with different types of toilets can result in marked variation in aerosol production, with high-energy toilets generating larger droplets and greater aerosol production (Johnson et al., 2013). Rapid gravitational sedimentation or shrinkage of large aerosol droplets usually occurs in the first 15–30 s immediately after flushing, and the dynamic regime of aerosol concentration in the air translates to inconsistent results across the literature (Johnson et al., 2013; O'Toole et al., 2009). Complicating matters further, the deposition rate of aerosols in the respiratory system varies with physical properties of aerosol, such as size, density, and shape, and also the breathing patterns of humans (e.g., breathing cycle, breathing intensity) (Heyder et al., 1986). While most individuals breathe predominantly through the nose, habitual and obligatory nasal-oral breathers are not uncommon (Warren et al., 1988). These are important considerations as our noses retain and remove large deposited particles through mucociliary clearance (up to 83% for 2.5–10 μm particles) before they reach human's lower respiratory tract (Couch et al., 1966; Fry and B.A., 1973). Particles deposited within macrophages or upon the mucus layer itself are primarily cleared to the gastrointestinal tract, which represents a transmission pathway for pathogens causing gastrointestinal illness (Stuart, 1984).

Similarly, the size distribution of aerosols produced by shower heads varies as a function of the water flow rate, water temperature, relative humidity, and also configuration of the shower room (e.g., ventilation) (Zhou et al., 2007). In addition, an individual's shower temperature preference is greatly influenced by season. Heating shower water can also affect risk. Viruses can be inactivated thermally, the kinetics of which are determined by the water temperature, contact time, and also the types of viruses (Bozkurt et al., 2013; Maheshwari et al.,

2004; Tuladhar et al., 2012). Conventional water heaters, which heat and store hot water in a tank, can be a potential virus inactivation system due to the longer contact time, whereas tank-less, on-demand water heaters might contribute to insignificant reduction of viruses due to the very short contact time with the hot water.

Irrigation of food-crops poses a markedly different situation. Water retained on the food-crops can transmit pathogens in the water to cause human enteric infection through ingestion of the crop. Owing to its capacity to trap water on its surface and its popularity among household growers, lettuce has been the subject of previous risk assessments for other types of contaminated water (Hamilton et al., 2006; Lim and Jiang, 2013; Mok and Hamilton, 2014) but not stormwater.

More broadly, risk modeling for stormwater has received little attention in comparison with other water reuse practices (see Hamilton et al., 2007 and citations within, and more recent examples: Barker et al., 2013; Olivieri et al., 2014; Symonds et al., 2014). QMRA has also been conducted in recreational waters receiving urban storm runoff using screening-level data (Ashbolt et al., 2010), based on the pathogen numbers inferred from indicator bacteria numbers (Tseng and Jiang, 2012), or using stormwater pathogen data inferred from surface water (McBride et al., 2013). These studies suggest that the health risk associated with recreation in stormwater-affected surface water is noteworthy and requires intervention to reduce stormwater impacts on recreational waters. Inherently, harvesting stormwater for non-potable uses also necessitates the evaluation of hazards that are present in stormwater. Given the concern enteric viruses raise to public health, their presence in stormwater, and the rapidly increasing interest in stormwater application for domestic purposes, it is perhaps surprising that to date there has not been a study on health risk assessment of stormwater harvesting practice.

To redress this gap, here we present a QMRA for two viruses of public health significance, norovirus and adenoviruses, for three non-potable applications of LID-treated stormwater: toilet flushing, showering, and food-crop irrigation.

2. Materials and methods

QMRA was conducted following the U.S. National Academy risk assessment framework, which consists of hazard identification, exposure assessment, dose–response assessment, and risk characterization (National Research Council, 1983). The Monte Carlo technique was used to represent the propagation of variability and uncertainties in risk estimation. All calculations were conducted using MATLAB R2012a (The MathWorks Inc., Natick, MA).

2.1. Hazard identification

2.1.1. Viral concentration in stormwater

Having identified adenovirus and norovirus as the important microbial hazard in stormwater uses, we collected virus data in surface waters affected by urban stormwater runoff as an indirect measure of viral concentration in stormwater after failed attempts to compile meaningful data for stormwater virus load directly (see Section 1 Introduction). The definition for surface water herein is urban tributaries and rivers, which may function as source water for drinking water treatment plants and recreational waters. Urban surface waters are usually affected by storm drain flow. Stormwater can be idealized as undiluted surface water, as in a recent QMRA study (McBride et al., 2013). A concentration factor can then be used to estimate viral water quality of stormwater based on that of surface water,

$$C_{virus,storm} = C_{virus,surface} \times F_{conc} \times \frac{1}{R_{eff}}, \quad (1)$$

where $C_{virus,storm}$ is the estimated viral concentration in stormwater (genome copies/L), $C_{virus,surface}$ is the measured viral concentration in surface water (genome copies/L), F_{conc} is the viral concentration factor from surface water to stormwater (unitless) that is adopted from the study of McBride et al (2013), and R_{eff} is the recovery efficiency of virus quantification method (unitless).

With the acknowledgement of the existing limitations and uncertainties, we compiled quantitative virus data in urban surface water for inferring viral concentration in stormwater based on two criteria: 1) quantitative PCR (qPCR) as the detection method and 2) surface water that receives storm-runoff. Accepted data are derived from different countries and also varied in viral concentration methods, and the qPCR primers and probes used (Table 1 and citations within). In the absence of the seasonal data on viral concentration from most regions, seasonal variability was not included in the QMRA and the viral concentrations from all relevant literature were used to provide a broader view of the risk.

2.1.2. Distribution fit for virus data

A portion of adenovirus and norovirus genogroup I and II (noroviruses GI + GII) data compiled from the literature were reported as non-detects. Instead of applying the commonly used strategy of replacing non-detects with single values (i.e., detection limit value or half of detection limit value) (e.g., Helsel, 2005), which is known to create biased results, we applied a left-censored data regression technique (Tobit regression) for estimating parameters that characterize the viral concentration distribution (Lubin et al., 2004). Following inspection of each virus data histogram and based on the knowledge that most environmental and microbial measurement data are distributed log-normally (Hirano et al., 1982; Loper et al., 1984), we assumed that adenovirus data follow a unimodal $\log_{10}$-transformed normal distribution and norovirus a bimodal $\log_{10}$-transformed normal distribution. Thus, the concentrations of adenoviruses ($C_{AdV,surface}$) and noroviruses ($C_{NoV,surface}$) in surface waters (genomic copies/L) are respectively given as

$$\log_{10} C_{AdV,surface} = N(\mu, \sigma), \quad (2)$$

and

$$\log_{10} C_{NoV,surface} = \alpha \times N(\mu_1, \sigma_1) + (1-\alpha) \times N(\mu_2, \sigma_2). \quad (3)$$

Non-detects are treated as latent continuous variables, $C_{virus,surface}{}^*$, which have been left-censored, and where

$$C_{virus,surface} = C_{virus,surface}{}^* \quad \text{if} \quad C_{virus,surface}{}^* > \rho$$

and

$$C_{virus,surface} = \text{missing} \quad \text{if} \quad C_{virus,surface}{}^* \leq \rho,$$

and where ρ is the detection-limit parameter, which can take different values depending on the virus detection method used in study at hand. There are five different detection-limit values for the compiled adenovirus data. For the purposes of our study, we set the lowest observed value to be the deterministic detection limit value and applied the Tobit regression on the virus data to generate the best-fit.

The maximum likelihood distribution fits for adenovirus and norovirus concentration in surface water (Fig. 1) indicate that theoretical and empirical probability distribution curves of the data are visually mismatching due to the presence of non-detects and the arbitrarily selected bin sizes for the histograms. Cumulative probability plots of the theoretical (10,000 iteration values) and empirical distribution were thus used to justify the appropriate distribution assumption and good fit of the data. The best-fit parameters are presented in Table 2.

In estimating the viral concentration in LID systems-treated stormwater, a 5-$\log_{10}$ viral reduction value was assigned to the estimated viral concentration in harvested stormwater. This reduction value is

Table 1
Summary of references used for collecting concentration of viruses in surface water.

Virus	Reference	Virus concentration method	No. of samples	No. of samples below DL	Types of data	Recovery efficiency[a]	Recovery target	Primers and probes reference
Adenovirus	Albinana-Gimenez et al. (2006)	Adsorption–elution	2	0	Observed value	25%	HAdv2	Hernroth et al. (2002)
	Albinana-Gimenez et al. (2009)	Adsorption–elution	2	0	Observed value	4.2% (2–6.9%)	HAdv2	Hernroth et al. (2002)
	Haramoto et al. (2010)	Adsorption–elution	18	7	Observed value	–	–	Ko et al. (2005)
	Aslan et al. (2011)	Adsorption–elution	2	0	Geometric mean	–	–	Xagoraraki et al. (2007)
	Choi and Jiang, 2005	Ultrafiltration	12	4	Median	54% (<0–>100%)	Bacteriophage ΦHSIC	He and Jiang (2005)
	Dong et al. (2010)	Ultrafiltration	14	10	Observed value	41% (21–89%)	MS2 coliphage	Heim et al. (2003)
	Calgua et al. (2013)	Skim milk flocculation	12	0	Observed value	65% (24–94%)	HAdV	Hernroth et al. (2002)
	Kishida et al. (2012)	Adsorption–elution	52	30	Observed value	–	–	Ko et al. (2005)
Norovirus GI	Kishida et al. (2012)	Adsorption–elution	52	23	Observed value	–	–	Kageyama et al. (2003)
Norovirus GII	Kishida et al. (2012)	Adsorption–elution	52	20	Observed value	–	–	Kageyama et al. (2003)
	Calgua et al. (2013)	Skim milk flocculation	7	0	Mean	53% (22–74%)	NoV GII	Jothikumar et al. (2005)

[a] Unbracketed numbers are mean recovery efficiency, whereas bracketed numbers are the range of recovery efficiency.

based on Li et al. (2012)'s experimental study of a biofilter's removal efficiency for virus indicators (>4-$\log_{10}$ removal), plus an additional $\log_{10}$ removal of virus assigned to the polishing step (i.e., microfiltration) to produce the finished water for domestic applications.

2.2. Exposure assessment

As the dose–response model for adenoviruses is based on the serotype 4, which causes respiratory infection and transmitted through

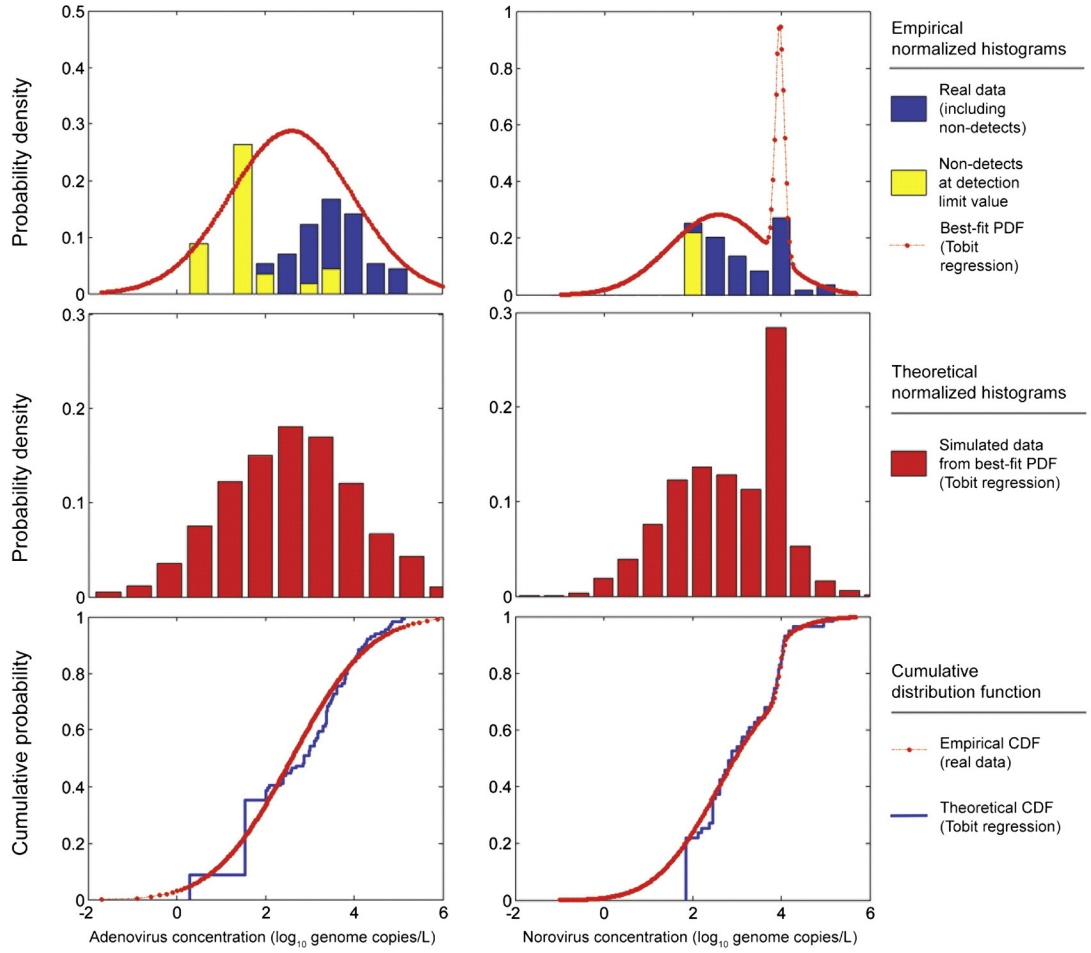

Fig. 1. Distribution fit for adenovirus and norovirus concentration in surface water based on data reported in literature and compiled in Table 1. Left-censored regression technique (Tobit regression) is used to treat the data reported as non-detects.

Table 2
List of parameters used in hazard identification of the study.

Description	Unit	Symbol	Point estimate	Probability distribution	Reference
Concentration of adenovirus in surface water	$\log_{10}$(genomes/L)	$C_{AdV,surf}$		N(2.588,1.385)	
Concentration of norovirus in surface water	$\log_{10}$(genomes/L)	$C_{NoV,surf}$		Bimodal normal, $0.792 \times N(2.578, 1.114) + 0.208 \times N(3.959, 0.100)$	
Viral concentration factor from surface water to stormwater	Unitless	F_{conc}	30		McBride et al. (2013)
Recovery efficiency of virus quantification method	Unitless	R_{eff}	0.1		
Log_{10} reduction of virus by LID systems[a]	Unitless	$\log_{LID}$	5		Li et al. (2012)

[a] Value is justified by assumptions made in Section 2.1.2.

inhalation route, we estimated adenovirus risk based on the viruses' deposition in human respiratory system. Conversely, norovirus risk was estimated based on inhalation–ingestion route through mucociliary action (assuming all aerosols trapped by our nose are cleared to gastrointestinal tracts) as noroviruses mainly cause gastrointestinal infection. Furthermore, due to the differential deposition efficiencies of aerosols in extrathoracic (nasal and laryngeal) region through nasal versus oral breathing, a distinction was made between the two in our risk assessment.

Only noroviruses were accounted for in the risk associated with food-crop irrigation. Although certain serotypes of adenovirus also cause gastroenteritis, there has not been a dose–response model for enteric adenovirus to be used in the QMRA.

2.2.1. Toilet-flushing scenario

The deposition efficiency of aerosols in the human body during toilet flushing is considered based on breathing pattern as indicated by U.S. EPA, which is represented by the inhalation rate for individuals engaging in light activities (U.S. EPA, 2011). These deposition efficiencies were derived empirically by Heyder et al. (1986) as a function of particle size and breathing patterns. A breathing rate of 15 L of air/min, an 8-second breathing cycle period (4 s each for inspiration and expiration), and 1 L of tidal volume were applied. Duration of exposure to the aerosols is defined as the time an individual would stay in the room after flushing the toilet. For simplicity, this exposed duration is set at 1 min and 5 min to represent a range of exposure scenarios.

The dose of adenoviruses ($Dose_{AdV,toilet}$) and noroviruses ($Dose_{NoV,toilet}$) inhaled and deposited in human's system (genomic copies) after flushing the toilet were estimated as

$$\begin{aligned} Dose_{AdV,toilet} &= \sum_{i=1}^{n} C_{AdV,treated} \times AerosolDose_{diam_i} \times MFR_{air} \times Duration_{toilet} \\ &= \sum_{i=1}^{n} \left[C_{AdV,storm} \times 10^{-\log_{LID}} \right] \\ &\quad \times \left[C_{aero,diam_i} \times V_{aero,diam_i} \times DE_{B+A,diam_i} \right] \times MFR_{air} \\ &\quad \times Duration_{toilet} \end{aligned} \tag{4}$$

and

$$\begin{aligned} Dose_{NoV,toilet} &= \sum_{i=1}^{n} C_{NoV,treated} \times AerosolDose_{diam_i} \times MFR_{air} \times Duration_{toilet} \\ &= \sum_{i=1}^{n} \left[C_{NoV,storm} \times 10^{-\log_{LID}} \right] \\ &\quad \times \left[C_{aero,diam_i} \times V_{aero,diam_i} \times DE_{ET,diam_i} \right] \\ &\quad \times MFR_{air} \times Duration_{toilet}, \end{aligned} \tag{5}$$

where $C_{AdV,treated}$ and $C_{NoV,treated}$ are the concentration of adeno- and noroviruses in treated stormwater (genomic copies/L), $\log_{LID}$ is the $\log_{10}$ reduction of adeno- and noroviruses by LID systems (unitless), $C_{aero,diam_i}$ is the concentration of aerosols (according to median diameter size, i) in the air generated after a single toilet flush (# of aerosols/m^3 of air) and $V_{aero,diam_i}$ is the volume of spherical aerosol (L/aerosol), $DE_{B+A,diam_i}$ and $DE_{ET,diam_i}$ are the deposition efficiencies of aerosols on bronchial and alveolar region, and extrathoracic region, respectively (unitless), MFR_{air} is the mean flow rate of air breathed after toilet flushing (m^3 of air/min), and $Duration_{toilet}$ is the time spent in the room after toilet flushing (min).

2.2.2. Showering scenario

Only conventional water heaters were considered. Shower temperature preferences of 50 °C and 60 °C were chosen in an attempt to represent variation in this parameter. Assumptions about shower duration, flow rates, and thermal reduction are given in Table 3 along with sources to justify these choices. The doses of adenoviruses ($Dose_{AdV,shower}$) and noroviruses ($Dose_{NoV,shower}$) inhaled and deposited in a person's system (in genomic copies) during showering were estimated as

$$\begin{aligned} Dose_{AdV,shower} &= C_{AdV,T_{shower}} \times \frac{AerosolDose_{B+A}}{\rho_{water}} \times Duration_{shower} \\ &= \left\{ C_{AdV,storm} \times 10^{-\log_{LID}} \times \frac{(100 - \%_{hot}) + \%_{hot} \times 10^{-\log_{T,hot}^{AdV}}}{100} \right\} \\ &\quad \times \frac{AerosolDose_{B+A}}{\rho_{water}} \times Duration_{shower} \end{aligned} \tag{6}$$

and

$$\begin{aligned} Dose_{NoV,shower} &= C_{NoV,T_{shower}} \times \frac{AerosolDose_{ET}}{\rho_{water}} \times Duration_{shower} \\ &= \left\{ C_{NoV,storm} \times 10^{-\log_{LID}} \times \frac{(100 - \%_{hot}) + \%_{hot} \times 10^{-\log_{T,hot}^{NoV}}}{100} \right\} \\ &\quad \times \frac{AerosolDose_{ET}}{\rho_{water}} \times Duration_{shower}, \end{aligned} \tag{7}$$

where $C_{AdV,T_{shower}}$ and $C_{NoV,T_{shower}}$ are the concentration of adeno- and noroviruses in shower water (genomic copies/L), $\%_{hot}$ is the percentage of hot water used for mixing with ambient temperature water to produce shower water at the desired temperature, $\log_{LID}$ is the $\log_{10}$ reduction of adeno- and noroviruses by LID systems (unitless), $\log_{T,hot}^{AdV}$ and $\log_{T,hot}^{NoV}$ are the $\log_{10}$ reductions of adeno- and norovirus at the temperature of the hot water used for shower water mixing (unitless), $AerosolDose_{B+A}$ and $AerosolDose_{ET}$ are the mass of water aerosol deposited in the bronchial–bronchiolar + alveolar-interstitial region and extrathoracic region (g/min), respectively, and ρ_{water} is the density of water (g/L), and $Duration_{shower}$ is the showering time (min).

Table 3
List of parameters used in exposure assessment of the study.

Description		Unit	Symbol	Point estimate		Probability distribution	Reference
Toilet-flushing scenario							
Concentration of aerosol in air after each toilet flush (at different sampling heights)		# of aerosols/cm^3 air	$C_{aero,diam,i}$				O'Toole et al. (2009)
Median diameter size, *i*	0.6 μm (42 cm above toilet)					Uniform(0, 106.9)	
	2.5 μm (42 cm above toilet)					Uniform(0, 11.6)	
	2.5 μm (5 cm above toilet)					Uniform(0, 24.5)	
Deposition efficiency of aerosols in extrathoracic region		Unitless	$DE_{ET,i}$	Oral breathing	Nasal breathing		Heyder et al. (1986)
Aerosol size, *i*	0.6 μm			0	0.04		
	2.5 μm			0.01	0.42		
Deposition efficiency of aerosols in bronchial and alveolar region		Unitless	$DE_{B+A,i}$	Oral breathing	Nasal breathing		
Aerosol size, *i*	0.6 μm			0.17	0.18		
	2.5 μm			0.61	0.41		
Mean flow rate of air during a minute of breathing cycle		L of air/min	MFR_{air}	15			U.S. EPA (2011)
Duration spent in restroom after flushing toilet	Mean scenario	Min/flush	$Duration_{toilet}$	1			
	Worst-case scenario			5			
Showering scenario							
Log_{10} reduction of Adenovirus through heat inactivation by hot water		Unitless	$log^{AdV}{}_{T,heat}$				Maheshwari et al. (2004)
	At T = 50oC			Inf			
	At T = 60oC			Inf			
Log_{10} reduction of Norovirus through heat inactivation by hot water		Unitless	$log^{NoV}{}_{T,heat}$				Gibson and Schwab (2011)
	At T = 50 °C			1.7			
	At T = 60 °C			5.2			
Percentage of hot water used for mixing during summer[a]		%	$\%Summer^{hot}{}_{,T}$				
Hot water	at T = 50 °C			15			
	at T = 60 °C			11.3			
Percentage of hot water used for mixing during winter[a]		%	$\%Winter^{hot}{}_{,T}$				
Hot water	at T = 50 °C			81.9			
	at T = 60 °C			64.1			
Mass of water deposited in the extrathoracic region per minute of shower		mg/min	$AerosolDose_{ET}$	Oral breathing	Nasal breathing		Zhou et al. (2007)
Hot shower (T = 43.5 °C) at flowrate of:	5.1 L/min			0.659	0.951		
	6.6 L/min			0.637	0.994		
	9.0 L/min			0.852	1.211		
Cold shower (T = 24.5 °C) at flowrate of:	5.1 L/min			0.004	0.001		
	6.6 L/min			0.007	0.018		
	9.0 L/min			0.02	0.029		
Mass of water deposited in the bronchial and alveolar region per minute of shower		mg/min	$AerosolDose_{B+A}$	Oral breathing	Nasal breathing		
Hot shower (T = 43.5 °C) at flowrate of:	5.1 L/min			0.297	0.036		
	6.6 L/min			0.357	0.049		
	9.0 L/min			0.364	0.044		
Cold shower (T = 24.5 °C) at flowrate of:	5.1 L/min			0.005	0.002		
	6.6 L/min			0.008	0.003		
	9.0 L/min			0.007	0.001		
Duration of each shower		min/shower	$Duration_{shower}$	20			
Food crop irrigation scenario							
Mass of raw lettuce intake per unit body weight per day		g of lettuce/kg-day	$M_{lettuce:body}$			Empirical distribution of consumer-only intake for all age-groups	U.S. EPA (2011)
Body weight of U.S. population		kg	M_{body}			Empirical distribution of body weight from populations of all age-groups	Kahn and Stralka (2009)
Volume of water retained on per unit weight of lettuce		L/g of lettuce	$V_{lettuce}$			Uniform (0.24,0.48) × 10^{-5}	Shuval et al. (1997)
Withholding time (between last irrigation and harvesting/eating)		Days	$T_{withhold}$			Uniform (0,3)	Hirneisen and Kniel (2013)
Environmental decay rate of norovirus		log_{10}/day	log_{decay}	0.192			

[a] Assuming that the temperature of tap water is 20 °C and 14 °C during summer and winter, respectively.

2.2.3. Food-crop irrigation scenario

Lettuce was modeled as the representative vegetable. We assumed that lettuce is watered every two to three days, between which the environmental decay of microbes deposited on the surface of lettuce leaves will occur. Considering the growing period and high perishability of lettuce and also the varying expertise of home growers, it is unlikely

that homegrown lettuce will be consumed daily throughout a year. Thus, we assessed only how the risk varies from one lettuce meal to 90, 180, and 270 meals per year. The environmental decay rate of norovirus GII on savoy spinach was used as a surrogate for estimating the reduction of norovirus on homegrown lettuce during the withholding period between last irrigation and harvesting/consumption of lettuce (Hirneisen and Kniel, 2013). Assumptions and relevant sources relating to consumption and water capture on leaf surfaces are given in Table 3.

The dose of noroviruses ($Dose_{NoV}$) ingested through intake of raw lettuce (in genomic copies) was estimated as

$$Dose_{NoV} = C_{NoV,treated} \times 10^{-\log_{decay} \times T_{withhold}} \times M_{lettuce:body} \times M_{body} \times V_{lettuce}, \quad (8)$$

where $\log_{decay}$ is the $\log_{10}$ reduction of norovirus due to environmental decay ($\log_{10}$/day), $T_{withhold}$ is the duration of environmental decay, $C_{NoV,treated}$ is the concentration of noroviruses in treated stormwater $= C_{NoV,treated} \times 10^{-\log_{decay}}$, which include the 5-$\log_{10}$ reduction values (genomic copies/L) by LID treatment. The daily intake of lettuce is calculated as a function of body mass $= M_{lettuce\,:\,body} \times M_{body}$, where $M_{lettuce\,:\,body}$ is the mass of raw lettuce intake per unit body weight per day (grams of lettuce/kg-day) and M_{body} is the body weight of U.S. population (kg). The volume of water retained on per unit weight of lettuce is $V_{lettuce}$ (L/g of lettuce).

2.3. Dose–response assessment

The risk or probability of getting infected through intake of pathogens was estimated using dose–response models based on clinical trial data. The adenovirus dose in genome copies was converted to median tissue culture infective dose ($TCID_{50}$), using 1 $TCID_{50}$ equals 700 genomes, to be consistent with that of clinical trial data (Couch et al., 1966; McBride et al., 2013). All adenovirus genomic copies are included in the assessment to yield the maximal estimate of risk, although only a sub-portion of the 51 adenovirus serotypes are known to cause respiratory illnesses (Mena and Gerba, 2009). The dose–infection model is characterized by an exponential function (Haas et al., 1993)

$$P_{\inf,AdV} = 1 - e^{-r \times Dose_{AdV}^{TCID_{50}}}. \quad (9)$$

$P_{\inf,AdV}$ is the estimated infection risk, and r represents infectivity of the virus and is the best-fit parameter of the model, and $Dose_{AdV}^{TCID_{50}}$ represents the dose of adenovirus in $TCID_{50}$.

Dose–response model for monodispersed norovirus as also used by other norovirus QMRA to maximize the infection risk outcome and the margin was adopted. This dose–infection was characterized by a confluent hypergeometric function (Teunis et al., 2008)

$$P_{\inf,NoV} = 1 - {}_1F_1(\alpha, \alpha+\beta, -Dose_{NoV}). \quad (10)$$

Similar to the dose–infection model for adenovirus, $P_{\inf,NoV}$ is the infection risk caused by norovirus, whereas α and β are the fitting parameters of the model. $Dose_{NoV}$ is the dose of norovirus in genome copies.

Both Eqs. (9) and (10) estimate infection risk, wherein infection does not always translate to illness (symptomatic infection) and is dependent on many factors such as an individual's immunity status, age, medical conditions, and nutrient intake. Higher pathogen dose generally results in higher probability of illness. In the absence of dose–illness data, as is the case for adenoviruses, probability of illness is estimated as a fixed portion of probability of infection, which is multiplied by a coefficient representing the percentage of illness cases in every infection case. In this study, a value of 0.5 for this coefficient is used for adenoviruses (Table 4). For norovirus, a dose–illness model has been developed as a function of pathogen dose intake (Teunis et al., 2008), where conditional dose-dependent norovirus illness risk is expressed as

$$P_{ill,NoV}|P_{\inf,NoV} = 1 - (1 + \eta \times Dose_{NoV})^{-r_{ill,NoV}}. \quad (11)$$

The best-fit parameters, η and $r_{ill,NoV}$, which describe the effects of initial pathogen dose and host's defenses, are also based on that for monodispersed norovirus genome copies.

The general illness risk equation, which applies for both adenoviruses and noroviruses, is expressed as

$$P_{ill,virus} = P_{\inf,virus} \times \left(P_{ill,virus}|P_{\inf,virus}\right). \quad (12)$$

2.4. Risk characterization

Two widely-used health risk benchmarks, the acceptable annual infection risk level proposed by the U.S. EPA (2005) and the acceptable disability-adjusted life years (DALYs) by WHO, were used for interpreting the magnitude of risk assessment outcomes. The U.S. EPA benchmark is $\leq 10^{-4}$ infection cases per-person-per-year (pppy), and the WHO benchmark is $\leq 10^{-6}$ DALYs pppy (World Health Organization, 2008).

Table 4
List of parameters used in dose–response assessment and risk characterization of the study.

Description	Unit	Symbol	Point estimate	Reference
Dose–response assessment				
Dose–infection parameter for adenovirus	–	r	0.4172	Haas et al. (1999)
Dose–infection parameters for norovirus		α	0.04	Teunis et al. (2008)
		β	0.055	
Adenovirus dose conversion factor	$TCID_{50}$/genome copies	$C^{AdV}_{TCID50/GC}$	1/700	McBride et al. (2013)
Conditional probability of illness given an infection due to adenovirus		$P(ill\|inf)_{AdV}$	0.5	
Conditional dose–illness parameters for norovirus		η	2.55×10^{-3}	Teunis et al. (2008)
		$r_{ill,NoV}$	0.086	
Risk characterization				
Frequency of shower in a day	Times	$Freq_{shower}$	1	
Frequency of flushing toilet in a day	Times	$Freq_{flush}$	4	
Frequency of eating lettuce in a year	Times	$Freq_{meal}$	90, 180, or 270	
DALYs per illness case of Adenovirus disease[a]		*DALYs/illness case*	0.05340	Gaunt et al. (2011)
DALYs per illness case of Norovirus disease[b]		*DALYs/illness case*	0.00095	Kemmeren (2006)

[a] Dataset in Table 2 of reference was used. Adenovirus disease burden per 1000 population for age-groups <5, 6–15, 16–64, and >65 years old was summed up.
[b] DALYs/illness case is computed by dividing total DALYs per year by the total number of incidence cases. The values in Table 27 of Kemmeren, 2006.

The annual infection risk metric is computed based on the theorem of independence of probability as (Haas et al., 1999)

$$P_{\inf,annual_{scenario,virus}} = 1 - \prod_{i=1}^{n=365\times Freq_{scenario}} \left(1 - P_{\inf,virus_i}\right), \quad (13)$$

where $Freq_{scenario}$ represents the number of times an activity is engaged during a day (e.g., one shower event per day), and n represents the total number of times an activity is engaged in a year. For food-crop irrigation, $n = Freq_{meal}$ as it is highly unlikely that an individual would eat the crop he/she grown every day.

Eq. (13) is also used to compute the annual illness risk, $P_{\inf,annual_{scenario,virus}}$, by substituting per-event illness risk (Eq. (12)) for per-event infection risk. Subsequently, the DALYs metric can be computed from the annual illness risk as (Mara and Sleigh, 2010)

$$DALY_{scenario,virus} = \frac{DALY}{\text{illness case}_{virus}} \times P_{ill,annual_{scenario,virus}}. \quad (14)$$

3. Results

3.1. Toilet flushing scenario

The viral infection risks from flushing toilet using treated stormwater water are mostly negligible (Fig. 2). Infection risks for all scenarios are typically order(s) of magnitude (median range: 1.1×10^{-7}–3.3×10^{-5} pppy, 95th percentile range: 2.7×10^{-7}–1.4×10^{-4} pppy) less than the U.S. EPA annual infection benchmark of $\leq 10^{-4}$ pppy. It is noted that norovirus infection risks are up to two orders-of-magnitude or 2 $\log_{10}$ higher than adenovirus infection risk. In terms of breathing style, adenovirus infection risks are within a two-fold difference between oral and nasal breathers. However, norovirus infection risks for nasal breathers are much higher than oral breathers (median: 3.3×10^{-5} pppy vs. 5.3×10^{-7}, 95th percentile: 1.4×10^{-4} pppy vs. 1.6×10^{-6} pppy) due to the nasal breathers' higher indirect ingestion rate of norovirus through mucociliary action. Duration of exposure to aerosols generated by toilet flushing has negligible influence on predicted annual risk, where the difference in risk between one minute exposure and five minutes exposure is within an order of magnitude (See APPENDIX A, Table A.1).

Disease burdens associated with toilet flushing (median range: 1.0×10^{-20}–5.4×10^{-9} DALYs pppy, 95th percentile range: 5.3×10^{-19}–1.4×10^{-8} DALYs pppy) are all far below the WHO's recommended threshold of $\leq 10^{-6}$ DALYs pppy (Fig. 3).

3.2. Showering scenario

Showering risk using treated stormwater differs depending on the virus inhaled, where norovirus infection risks clearly far exceed the U.S. EPA annual infection benchmark and are much higher than adenovirus infection risk (median range: 3.4×10^{-4}–4.3×10^{-2} pppy vs. 3.6×10^{-7}–6.0×10^{-5} pppy, 95th percentile range: 1.6×10^{-3}–2.9×10^{-1} pppy vs. 1.3×10^{-6}–3.5×10^{-4} pppy) (Fig. 2). In

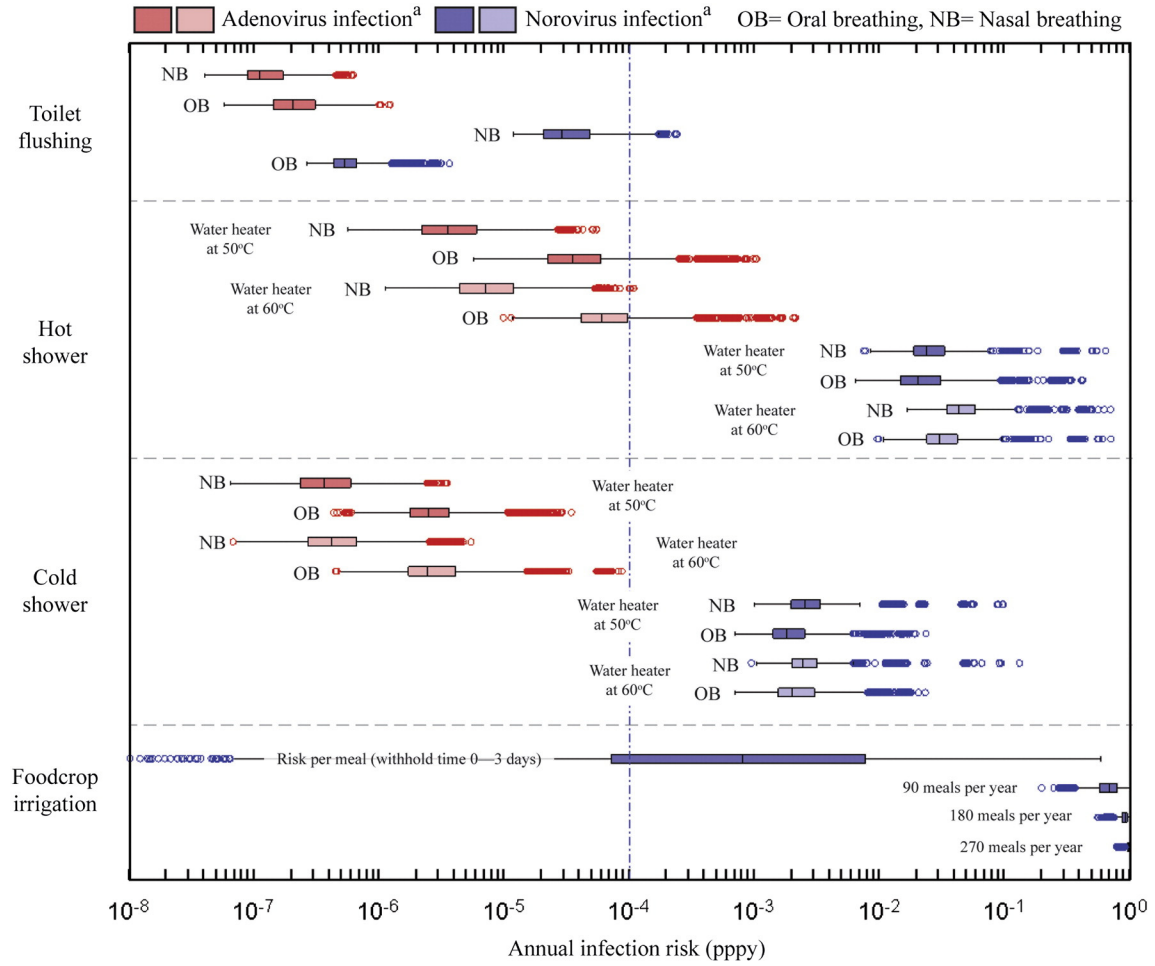

Fig. 2. Box-and-whisker plot showing the annual adenovirus and norovirus infection risks from using treated stormwater for various water applications. Each box represents the lower, median, and upper quartile (e.g., 25th, 50th, and 75th percentile values) of the distribution, where the whiskers extend 1.5 × (75th percentile value − 25th percentile value) from each end of the box. Markers graphed outside of each whisker are considered as outliers. The vertical dashed line represents the U.S. EPA annual infection risk benchmark of $\leq 10^{-4}$ pppy.

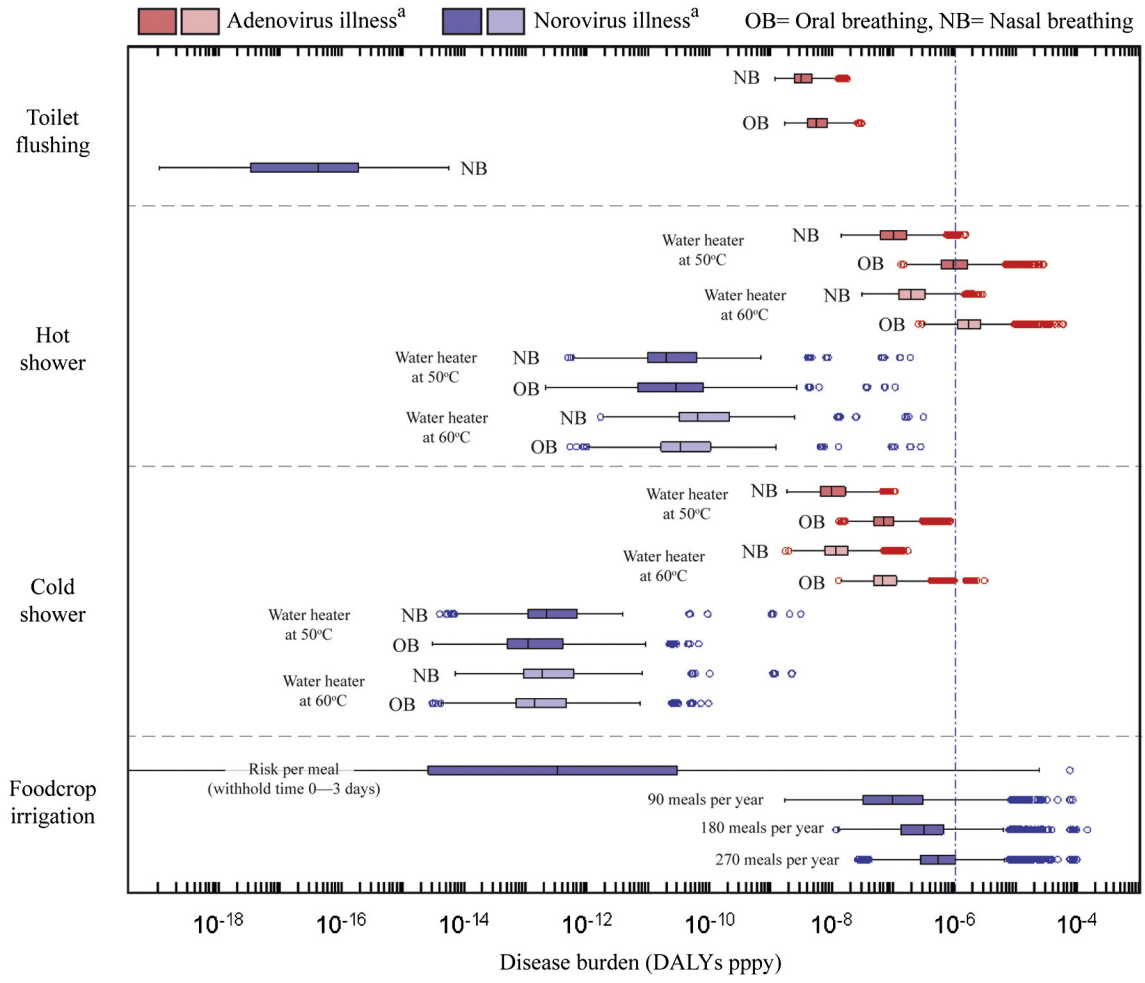

Fig. 3. Box-and-whisker plot showing the disease burdens of adenovirus- and norovirus-related illnesses due to using treated stormwater for various water applications. The vertical dashed line represents the WHO recommended benchmark of $\leq 10^{-6}$ DALYs pppy. Disease burden of norovirus for an oral breather flushing toilet is too low to be graphed.

comparison, the infection risks of hot showers are a $\log_{10}$ lower than those of cold showers when all else is equal. The breathing style of an individual does not alter the norovirus infection risk (within a $\log_{10}$ difference), whereas the adenovirus infection risk of an oral breather is typically a $\log_{10}$ higher than that of a nasal breather. Risk prediction also is not influenced by different shower water flow rates (See Appendix A, Table A.2).

When the infection risks of showering were translated to disease burdens, the opposite trend was observed (Fig. 3). The disease burdens of norovirus (median range: 4.1×10^{-15}–6.3×10^{-11} DALYs pppy, 95th percentile range: 3.5×10^{-8}–6.1×10^{-8} DALYs pppy) all fell below the WHO's benchmark, whereas portion of the disease burdens of adenovirus (median range: 9.6×10^{-9}–1.6×10^{-6} DALYs pppy, 95th percentile range: 3.5×10^{-8}–9.3×10^{-6} DALYs pppy) exceeded the benchmark.

3.3. Food-crop irrigation scenario

Norovirus infection risks from the consumption of stormwater-irrigated raw lettuce varied little (median range: 0.681–0.973 pppy, 95th percentile range: 0.881–0.995 pppy) when a range of 90 to 270 meals per year intake frequency was considered (Fig. 2 see Appendix A, Table A.3). The per-event risk had a median of 8.0×10^{-4} pppy and 95th percentile value of 5.2×10^{-2} pppy. Despite such a wide range on the event scale, the annual risk (multiple intakes) converged rapidly to the 10^{-1} range.

Again, disease burdens of the food-crop irrigation shed a very different light on the risk interpretation, where the DALYs computed for the different intake frequency (median range: 9.5×10^{-8}–5.1×10^{-7} DALYs pppy, 95th percentile range: 2.3×10^{-6}–1.8×10^{-5} DALYs pppy) pppy) frequently fall below that of the WHO's benchmark (Fig. 3).

4. Discussion

4.1. Implications

Models developed in this study conceptualize the health risks associated with LID-treated stormwater in three domestic applications and identify the uncertainties for a more accurate risk assessment. The QMRA predictions rank the viral risks of toilet flushing the lowest while food-crops irrigation the highest. Two of the three stormwater uses are generally above the U.S. EPA annual infection risk benchmark, while toilet flushing is well below the benchmark. It should be noted that U.S. EPA does not enforce the risk benchmark as a legal requirement, which is primarily established for assessment of safe drinking water. Nevertheless, the existence of the benchmark proposed by the authoritative government agency inevitably demands attention from water practitioners, and may also be relevant in a legal context when demonstrating due diligence. In fact, the U.S. EPA benchmark is used in Dutch regulatory processes, which require water authorities to comply with under a QMRA framework (Bichai and Smeets, 2013). Instead of a yes-or-no compliance, water utilities in the Netherlands use QMRA as a tool for discussion with the regulatory bodies to support decisions about water systems through acknowledging the uncertainties in QMRA. In the same way, the risk assessment outcomes presented

here could also be of value in assisting the adoption of alternative water resources for various applications.

Interpreting QMRA results usually draws interesting comparison with how waterborne disease risks are perceived and regulated in different states and countries. Toilet flushing is generally the most acceptable to the public (Dobbie and Brown, 2012; Wu et al., 2012). Flushing the toilet using non-potable water (i.e., seawater, reclaimed water, treated gray water) is practiced in many parts of the world (Leung et al., 2012) and is supported as being an acceptably safe practice by our risk assessment. Interestingly, the criteria for toilet flushing vary across states and countries. California, for example, has adopted the most stringent microbial standard for toilet flushing with reclaimed water (California Law, Title 22), which specifies a 7-day median of ≤2.2 total coliforms/100 mL of reclaimed water. This is three orders of magnitude lower than Japan's reuse criterion of 1000 total coliforms/100 mL of reclaimed water (Ogoshi et al., 2001). It should be noted that the microbial risk of flushing toilet is mostly derived from the aerosolization of human waste and vomitus rather than from the flushing water itself (Caul, 1994; Lopman et al., 2012). The disease transmission in public toilet facilities through aerosols (carrying human waste) generated by flushing water has not been investigated.

Showering using water that is not designated for potable-use is not a readily embraced idea for people due to the close contact of showering water with human. The annual risk profiles of showering suggest that viral infection risks are higher during winter, when individuals are more likely to take a hot shower than a cold shower. Aerosols produced using hot water are not only larger in size and quantity, but also more likely to reach infection sites in human body than when cold water is used. This phenomenon, combined with the depressed human immune systems during the cold seasons and tendency for people to stay indoors (i.e., secondary spread), are predicted to lead to higher infection risks during winter (Lavoy et al., 2011). In fact, many norovirus outbreaks had occurred in various geographical locations during winter and is so common in UK that norovirus is sometimes referred to as “winter vomiting bug” (Hall et al., 2011; Lopman et al., 2011; Siebenga et al., 2009).

Crop irrigation using reclaimed water is a well-accepted practice, which also makes harvested stormwater a potential water resource for crop irrigation. However, the annual risk profiles of this stormwater practice tell a very different story, where the infection risks exceeded the corresponding risk using non-disinfected secondary effluent for the same purpose (Asano et al., 1992; Hamilton et al., 2006; Olivieri et al., 2014; Petterson et al., 2001a; Tanaka et al., 1998). This finding, however, is not a total surprise as norovirus is much more infectious and resistant to environmental decay than the enteric virus used in the previous QMRA studies for crop irrigation. Many of these enteric virus studies have also relied on the use of bacteriophages as surrogate. However, the use of bacteriophages is inappropriate for norovirus as the dose–response model of norovirus is expressed in terms of genome copies, which might be vastly different from the plaque forming units (PFU) for bacteriophages due to the different principles of science involved behind each quantification method (McBride et al., 2013). In fact, norovirus quantified using genome copies are five times more resistant to environmental decay than bacteriophages under comparable experimental conditions (Hirneisen and Kniel, 2013; Petterson et al., 2001b). This comparative analysis of risk outcomes offers a basis for judging the safety and adequacy of new water applications. It also implies the need for incorporating updated science into risk assessment, which can be used to revise the findings in past research, and therefore, the current health risk benchmark.

4.2. Model uncertainties

A large number of factors can influence the model predictions. Firstly, viral concentrations in urban stormwater were deduced from surface water based on dilution factor, quantification recovery efficiency, and PCR inhibitions. The dilution factor of stormwater to surface water could be more accurately assessed with additional hydrological data inputs that usually become available with the development of a stormwater harvesting project (Inamdar et al., 2013). The recovery efficiency of virus in stormwater could be further improved since there is a lack of agreement among the literature values. The value as used in this study is representative of a worst case estimate for public health protection, while the viral concentration data as collected from literature were quantified using different primers and probes that targeting different serotypes of the virus, which may not best inform us of the likely disease/illness it may cause. This factor is reluctantly put aside, but was considered as an uncertainty compiled in the viral concentration distribution. It is recommended that future studies of viral concentration in environmental waters use standardized and uniform quantification methods, so as to produce/reproduce comparable results across different laboratories (Wyn-Jones et al., 2011). This would enable more informative statistical analysis, including Bayesian methods, as recommended by (Wu et al., 2014) for such circumstances. In spite of the aforementioned data inconsistencies, the virus data as used represent the range of uncertainties that are credible for risk analysis and are reducible with improved knowledge.

Uncertainties associated with virus removal efficiency of LID-treated water were considered by incorporating a safety factor based on a preliminary experimental value of column biofilters' removal efficiency for adenovirus (>4-log removal) (data not published) and F-RNA coliphages (3.1 to 4.6 log removal) (Li et al., 2012). Thus, our risk analysis presents a “what-if” scenario for treating harvested stormwater. However, more comprehensive studies related to the virus removal efficiency of biofilters and its dynamics (i.e., removal efficiency during wet or dry season) are warranted. The findings of these new studies should then be incorporated into the risk models as poor functioning/maintenance of LIDs could lead to inadequately treated stormwater for its intended usages and may heighten public health risks considerably.

Model uncertainties are also derived from components of exposure models. For example, most individuals are nasal–oral breathers engaging in both types of breathing instead of strictly nasal or oral, which places the annual risk of a typical individual between that of exclusive nasal or oral breathers. The wide range of annual risks observed in our results would reasonably be expected given the large differences in the scenarios considered, and it is likely that actual reuse situations would pose risks somewhere along this continuum.

Dose–response models used for estimating the probability of infection and/or illness are, perhaps, the most important source of uncertainties due to the end point result it generates for risk characterization. Considerable care has to be taken in the culmination of valid inputs for the models and, thus, correct interpretation of the result. In this regard, the complexity of norovirus dose–response model poses a number of data gaps to be filled by future research (Teunis et al., 2008). In particular, the aggregation state of norovirus in the finished water must be addressed. Dose–infection models for norovirus developed from clinical studies considered the virus aggregation factor (Teunis et al., 2008). The model that accounts for the aggregation factor treat viruses as aggregates and have higher ID_{50} than the model for monodispersed viruses (due to the higher efficiency of monodispersed viruses in reaching infection sites to cause infection). Most norovirus QMRAs conducted have ignored the aggregation factor, citing the lack of knowledge of virus aggregation states in water (McBride et al., 2013; Schoen and Ashbolt, 2010; Soller et al., 2010; Viau et al., 2011). In fact, aggregation of norovirus is likely rare in the environment due to the high stability of norovirus against aggregation in water near neutral pH and high ionic strength (da Silva et al., 2010), which is characteristic of stormwater (NRMMC–EPHC–NHMRC, 2009). Although aggregated noroviruses are not as infectious as when they are in monodispersed suspended form, the former are much more likely to cause illness in a person they successfully infected (e.g., higher DALYs). Neglecting the aggregation state of norovirus can result in widely different risk results, which potentially contradict risk management decisions

depending on the risk metric/benchmark (annual infection or DALYs) used. A more accurate risk assessment can also be aided through understanding the relationship of norovirus quantified in genome copies and infectious units, which currently cannot be assessed due to the lack of a sensitive cell line. The relationship may vary depending on the types of water (e.g., non-disinfected effluent vs. tertiary effluent). As a first start, a study by Hirneisen and Kniel (2013) comparing the environmental decay of norovirus GII in genome copies and MNV in PFU showed minor difference (within a $\log_{10}$ difference) between the two virus quantification methods.

Uncertainties as discussed herein are important in risk characterization. They should be used to guide any future risk assessment for improvement. Uncertainties should also adhere to individual circumstances, which could be unique to each case in the risk analysis.

4.3. Comparison of annual infection risks and disease burdens

Disease burdens are at times used for a broader cost-benefit analysis of microbial risks that encompasses socio-economic terms. The WHO recommends a benchmark of $\leq 10^{-6}$ DALYs pppy for safe drinking water, but has generated inconsistencies in regard to its adoption in different countries and its comparability with the U.S. EPA annual infection risk benchmark. As a matter of fact, both benchmarks are considered to be overly conservative and impractical by some risk assessors for evaluating the safety of using non-potable water for various water-related activities (Mara, 2011; Mara and Sleigh, 2010).

The metrics used for both approaches are directly related to each other: annual infection risks are converted to DALYs through incorporation of disease surveillance data such as severity and duration of illness attributable to identified target pathogen. These disease surveillance data are often regionally-bounded and therefore may not be representative of the whole population. The DALYs approach is not commonly used for risk assessment studies in the U.S., and therefore disease burden data specific to the U.S. are less readily available.

In our study, the disease burden of adenovirus is affected by the lack of surveillance data to characterize the true impacts of adenovirus-related illnesses. As presented in Fig. 3, the DALYs associated with adenovirus-related disease from showering are based on DALYs per illness case derived solely from hospitalized patients, which are heavily scaled upwards (i.e., people who are infected and ill, but with only mild disease symptoms would not visit a hospital) (Gaunt et al., 2011). As a result, adenoviruses risks frequently exceeded the WHO's DALYs benchmark, while looked much more “acceptable” in terms of U.S. EPA annual infection benchmark.

The conversion of DALYs from annual infection risks is perhaps most problematic because it requires the knowledge of the portion of ill subjects out of the infected subjects. Many risk assessments have used point-estimate of conditional illness probability to compute DALYs as a simple but not necessarily correct solution. Only Teunis et al. have put forth the idea that illness risk is a function of the dose of target pathogens took in by an individual (Teunis et al., 2008, 1999). In this regard, we have shown that the computation of DALYs is prone to being influenced significantly by the risk model of choice, where using point-estimate of conditional illness probability can greatly overestimate illness risk, and therefore, DALYs. In contrast, using a dose-dependent illness probability model has shown to moderate the high infection risk of norovirus, which would most likely translate to illness rate that is characteristic of a disease outbreak if point-estimate of conditional illness probability would be used instead. This observation offers a new perspective to evaluate the risk of norovirus (using DALYs), which is disastrous when only infection risk is considered.

The DALYs approach has the potential in adding values to risk management, but is blighted by the lack of data to support its development in the United States. More research is necessary to develop the DALYs approach before it can be used reliably for risk management. The approach should be treated cautiously in a similar manner to U.S. EPA benchmark, and the two should be used as complements rather than in opposition.

5. Conclusion

QMRA offers a useful tool for estimating the public health risk associated with stormwater harvesting and its applications in domestic households. Among the three non-potable use scenarios assessed in this study, toilet flushing presents the lowest health risk, being negligible in relation to both the U.S. EPA and WHO benchmarks. Showering presents a health risk that clearly exceeds the U.S. EPA benchmark, but complies with the WHO benchmark under certain settings. Consumption of fresh produce irrigated with treated stormwater exceeds both benchmarks. The results also showed the inconsistencies in risk interpretation based on different risk models and acceptable health risk benchmarks. Further improvements in data collection and model refinement are necessary to reduce the uncertainties and inconsistencies associated with the risk outcome. Ultimately, the outcomes of the risk assessment should be used as an educational tool to narrow the gap between perceived risk and estimated risk, instill stakeholders' confidence in stormwater harvesting practice, and protect public health.

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Appendix A. Tabulated summary descriptors for annual infection risk and disease burdens

Table A.1
Summary descriptors of the annual infection risk and disease burden for toilet flushing scenario.

	Toilet flushing	Median		Median		95th percentile		95th percentile	
		Adenovirus		Norovirus		Adenovirus		Norovirus	
		Nasal	Oral	Nasal	Oral	Nasal	Oral	Nasal	Oral
Annual infection risk	Typical (1 min)	1.11E-07	2.05E-07	3.28E-05	5.26E-07	2.72E-07	5.16E-07	1.37E-04	1.58E-06
	Worst (5 mins)	6.63E-07	8.89E-07	1.27E-04	4.76E-06	8.74E-07	1.17E-06	1.91E-04	8.88E-06
DALYs	Typical (1 min)	2.98E-09	5.43E-09	1.10E-16	1.00E-20	7.17E-09	1.37E-08	3.15E-15	5.27E-19
	Worst (5 mins)	1.77E-08	2.37E-08	1.54E-16	1.69E-18	2.34E-08	3.13E-08	1.25E-15	5.91E-18

Note: The median value of disease burdens of norovirus to oral breathers under typical condition is imputed using 10^{-20} DALYs pppy, as the real value computed using MATLAB® is zero due to the very low illness risk.

Table A.2
Summary descriptors of the annual infection risk and disease burden for showering scenario.

	Showering		Median					95th percentile			
			Adenovirus			Norovirus		Adenovirus		Norovirus	
			Shower water flow rate (L/min)	Nasal breathing	Oral breathing	Nasal breathing	Oral breathing	Nasal breathing	Oral breathing	Nasal breathing	Oral breathing
Annual infection risk	Hot shower (winter)	Hot water temp. at 50 °C	5.1	3.5E-06	2.6E-05	1.9E-02	1.5E-02	1.7E-05	1.2E-04	8.6E-02	1.9E-01
			6.6	3.9E-06	2.9E-05	1.8E-02	1.3E-02	1.9E-05	1.9E-04	8.7E-02	5.7E-02
			9.9	3.6E-06	3.5E-05	2.3E-02	2.0E-02	1.3E-05	3.5E-04	2.9E-01	9.4E-02
		Hot water temp. at 60 °C	5.1	6.9E-06	4.6E-05	3.8E-02	2.2E-02	3.3E-05	1.5E-04	1.6E-01	4.2E-02
			6.6	7.8E-06	5.3E-05	3.4E-02	2.4E-02	3.8E-05	2.0E-04	8.7E-02	1.1E-01
			9.9	7.1E-06	6.0E-05	4.3E-02	2.6E-02	2.6E-05	3.2E-04	1.9E-01	1.2E-01
	Cold shower (summer)	Hot water temp. at 50 °C	5.1	7.4E-07	2.0E-06	8.6E-04	3.4E-04	6.1E-06	1.4E-05	7.1E-03	1.7E-03
			6.6	1.0E-06	3.2E-06	1.6E-03	6.3E-04	4.0E-06	1.5E-05	7.5E-03	1.1E-02
			9.9	3.6E-07	2.5E-06	2.5E-03	1.8E-03	1.3E-06	1.1E-05	1.3E-02	8.4E-03
		Hot water temp. at 60 °C	5.1	7.7E-07	1.8E-06	8.2E-04	3.5E-04	5.2E-06	1.7E-05	2.0E-03	6.8E-03
			6.6	1.3E-06	3.5E-06	1.6E-03	6.3E-04	4.4E-06	1.3E-05	7.5E-03	1.6E-03
			9.9	4.2E-07	2.4E-06	2.4E-03	2.0E-03	2.0E-06	1.5E-05	1.2E-02	9.0E-03
Disease burden	Hot shower (winter)	Hot water temp. at 50 °C	5.1	9.2E-08	7.0E-07	1.2E-11	1.3E-11	4.5E-07	3.3E-06	2.4E-09	2.2E-08
			6.6	1.0E-07	7.8E-07	9.8E-12	6.8E-12	5.1E-07	5.1E-06	2.6E-09	1.1E-09
			9.9	9.6E-08	9.3E-07	1.9E-11	2.7E-11	3.5E-07	9.3E-06	6.1E-08	2.0E-09
		Hot water temp. at 60 °C	5.1	1.8E-07	1.2E-06	5.2E-11	1.6E-11	8.8E-07	3.9E-06	7.8E-09	1.4E-10
			6.6	2.1E-07	1.4E-06	3.8E-11	1.8E-11	1.0E-06	5.5E-06	1.4E-09	3.7E-09
			9.9	1.9E-07	1.6E-06	6.3E-11	2.2E-11	6.7E-07	8.8E-06	1.2E-08	6.1E-09
	Cold shower (summer)	Hot water temp. at 50 °C	5.1	2.0E-08	5.5E-08	2.6E-14	4.1E-15	1.6E-07	3.8E-07	1.6E-07	3.8E-07
			6.6	2.7E-08	8.5E-08	7.6E-14	1.4E-14	1.0E-07	4.1E-07	1.0E-07	4.1E-07
			9.9	9.6E-09	6.6E-08	2.2E-13	1.1E-13	3.5E-08	3.0E-07	3.5E-08	3.0E-07
		Hot water temp. at 60 °C	5.1	2.0E-08	4.9E-08	1.9E-14	4.2E-15	1.4E-07	4.5E-07	1.4E-07	4.5E-07
			6.6	3.4E-08	9.3E-08	8.1E-14	1.1E-14	1.2E-07	3.5E-07	1.2E-07	3.5E-07
			9.9	1.1E-08	6.5E-08	1.8E-13	1.4E-13	5.3E-08	4.1E-07	5.3E-08	4.1E-07

Table A.3
Summary descriptors of the annual infection risk and disease burden (norovirus) for food-crop irrigation scenario.

Food crop irrigation		Median				95th percentile		
		Withhold time	(0–3 days)	0 day	3 days	(0–3 days)	0 day	3 days
Annual infection risk	Meals per year	1	8.04E-04	1.60E-03	4.01E-04	5.15E-02	8.81E-02	2.55E-02
		90	6.81E-01	8.49E-01	4.68E-01	8.83E-01	9.56E-01	7.39E-01
		180	9.07E-01	9.79E-01	7.38E-01	9.75E-01	9.96E-01	8.98E-01
		270	9.73E-01	9.97E-01	8.69E-01	9.95E-01	1.00E + 00	9.58E-01
Disease burden	Meals per year	1	3.20E-13	1.20E-12	7.95E-14	1.40E-09	4.28E-09	3.32E-10
		90	9.46E-08	2.55E-07	2.88E-08	1.68E-06	2.33E-06	4.36E-07
		180	3.04E-07	6.83E-07	9.76E-08	7.62E-06	1.05E-05	8.59E-07
		270	5.15E-07	1.12E-06	1.73E-07	9.49E-06	1.78E-05	1.55E-06

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