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Risk-based enteric pathogen reduction targets for non-potable and direct potable use of roof runoff, stormwater, and greywater

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ABSTRACT

This paper presents risk-based enteric pathogen log reduction targets for non-potable and potable uses of a variety of alternative source waters (*i.e.*, locally-collected greywater, roof runoff, and stormwater). A probabilistic Quantitative Microbial Risk Assessment (QMRA) was used to derive the pathogen $\log_{10}$ reduction targets (LRTs) that corresponded with an infection risk of either 10^{-4} per person per year (ppy) or 10^{-2} ppy. The QMRA accounted for variation in pathogen concentration and sporadic pathogen occurrence (when data were available) in source waters for reference pathogens in the genera *Rotavirus, Mastadenovirus* (human adenoviruses), *Norovirus, Campylobacter, Salmonella, Giardia* and *Cryptosporidium*. Non-potable uses included indoor use (for toilet flushing and clothes washing) with occasional accidental ingestion of treated non-potable water (or cross-connection with potable water), and unrestricted irrigation for outdoor use. Various exposure scenarios captured the uncertainty from key inputs, *i.e.*, the pathogen concentration in source water; the volume of water ingested; and for the indoor use, the frequency of and the fraction of the population exposed to accidental ingestion. Both potable and non-potable uses required pathogen treatment for the selected waters and the LRT was generally greater for potable use than non-potable indoor use and unrestricted irrigation. The difference in treatment requirements among source waters was driven by the microbial quality of the water – both the density and occurrence of reference pathogens. Greywater from collection systems with 1000 people had the highest LRTs; however, those for greywater collected from a smaller population (~ 5 people), which have less frequent pathogen occurrences, were lower. Stormwater had highly variable microbial quality, which resulted in a range of possible treatment requirements. The microbial quality of roof runoff, and thus the resulting LRTs, remains uncertain due to lack of relevant pathogen data.

1. Introduction

Interest in using alternative waters in community water systems has increased in the United States and worldwide (National Academies of Sciences, 2016). Possible alternative waters include, but are not limited to:

- *Greywater*: wastewater from bathtubs, showers, bathroom sinks, and clothes washing machines, excluding toilet and—in most cases—dishwasher and kitchen sink wastewaters;
- *Roof runoff (rainwater)*: precipitation collected from roof surfaces or other above ground collection surfaces not impacted by human activity; and
- *Stormwater*: precipitation and runoff collected from ground level.

Given the lack of federal recommendations in the United States, communities face a challenge when using alternative waters for non-potable and potable purposes. Many states and communities have adopted standards based on fecal indictor bacteria concentrations in finished water (*e.g.*, the NSF/ANSI Standard 350 for non-potable onsite reuse of greywater (NSF International, 2015)). However, these standards lead to an unknown level of protection of human health for consumers (National Academies of Sciences, 2016).

In previous work, we reviewed the microbial risks associated with non-potable uses of alternative waters as predicted by Quantitative Microbial Risk Assessment (QMRA) (Schoen and Garland, 2015). QMRA is a scientific approach to estimate the potential human health risks resulting from exposures to microbial hazards (*i.e.*, human pathogenic viruses, protozoa, and bacteria) (Haas et al., 1999) and has been applied across multiple water

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regulatory processes (Petterson and Ashbolt, 2016; U.S. EPA, 2014; NRMMC et al., 2006; WHO, 2016). For the waters listed above, the microbial hazards include enteric pathogens resulting from human or animal fecal contamination; opportunistic pathogens (*e.g., Legionella pneumophila*) which may grow within the collection and distribution systems (Chapman et al., 2008; O'Toole et al., 2014; Garner et al., 2016; Ashbolt, 2015); antimicrobial resistant bacteria (including pathogens) (Ashbolt et al., 2013); and possibly endotoxins (Barker et al., 2016). In the previous review of QMRA-derived microbial risks, we concluded that risks associated with non-potable use of untreated or minimally treated alternative waters exceeded previously employed benchmark levels of risk. Yet, risk-based pathogen treatment targets aimed to lower the risk to benchmark levels were widely missing, apart from targets for stormwater for domestic and municipal purposes (Schoen and Garland, 2015; NRMMC et al., 2009).

Pathogen treatment targets, referred to as pathogen $\log_{10}$ reduction targets (LRTs), are the difference between the $\log_{10}$-transformed pathogen concentrations pre-treatment and post-treatment. (This is equivalent to the proportional reduction in the non-log scale.) Pathogen reduction targets that are "risk-based" are calculated to achieve a specific level of health protection for consumers. Please refer to Sinclair et al. (2015) for a discussion of the evolution of risk-based targets for drinking water. The level of health protection is typically expressed as a tolerable burden of disease (*e.g.*, Disability Adjusted Life Years [DALYs], the sum of years of life lost by premature mortality and years lived with disability (Murray and Acharya, 1997)) or as a tolerable/benchmark level of infection or illness risk per person per year [ppy] (*e.g.*, Regli et al., 1999).

The World Health Organization (WHO) and Australian government established risk-based LRTs of enteric pathogens for a limited number of uses for stormwater and municipal wastewater (NRMMC et al., 2006; NRMMC et al., 2009, 2008; WHO, 2006a). For potable water consumption, the WHO used a tolerable burden of disease of 10^{-6} DALYs ppy (WHO, 2011), which was also used for non-potable purposes (NRMMC et al., 2009; WHO, 2006a, 2006b; Health Canada, 2010). This tolerable burden of disease roughly corresponds to an infection risk of 10^{-3} ppy for *Cryptosporidium* spp., 7.2×10^{-4} ppy for *Campylobacter* spp., and roughly 10^{-4} ppy for *Rotavirus* (NRMMC et al., 2009; WHO, 2006a, 2006b). In the United States, an infection risk of 10^{-4} ppy for giardiasis has been used for surface water treatment requirements producing drinking water (Macler and Regli, 1993; U.S. EPA, 2006). As an alternative, the less restrictive illness risk of 10^{-2} ppy, based on the U.S. EPA Recreational Water Quality Criteria (U.S. EPA, 2012), may be applicable for voluntary exposures (Appendix A). Thus, a benchmark risk for non-potable uses in the U.S. likely falls within the range already adopted for potable and recreational exposures.

To support the development of microbial LRTs for the management of alternative waters, we computed risk-based pathogen reduction targets for enteric pathogens suited to both non-potable and potable uses of alternative source waters, assuming a benchmark rate of infection (not illness) of either 10^{-4} or 10^{-2} ppy. We present LRTs in two parts: first, using literature values for the pathogen concentration in each source water (or sources of contamination) accounting for the observed or modeled variation across collection locations and conditions; and second, using a set of alternative pathogen concentration characterizations so that site-specific targets may be estimated.

2. Methods

Schoen and Garland (2015) described the reverse QMRA methods previously used to calculate LRTs. While not adopted by other agencies, due to complications in computation, Schoen and Garland (2015) recommended a stochastic, forward approach, rather than a reverse approach, to allow for the inclusion of factors either missing or difficult to incorporate in the reverse approach. These factors included sporadic and variable pathogen occurrence and concentration, variation in pathogen dose over the course of a year, and occasional accidental ingestion.

2.1. QMRA model

The forward QMRA included the traditional QMRA steps used to calculate the annual probability of infection (Haas et al., 1999), but rearranged to solve for the LRT. To solve for the pathogen $\log_{10}$ reduction target (LRT) for a set of activities, the annual probability of infection ($\text{Pinf}_{\text{annual}}$) was set equal to the benchmark infection risk as follows:

$$\text{Pinf}_{\text{annual}} = \text{Benchmark infection risk} = S * \left(1 - \prod_{n_i}\left[1 - DR\left(V_i * 10^{(log_{10}(C) - LRT)}\right)\right]\right) \quad (1)$$

where

- S is the fraction of people in the exposed population susceptible to each reference pathogen.
- DR(…) is a dose-response function for the reference pathogen.
- V_i is the volume of water ingested per day for the activity set i.
- n_i is the number of days of exposure over a year for activity set i.
- C is the pathogen concentration in the untreated, freshly collected source water.

The annual probability of infection for an activity set in Eq. (1) was calculated assuming independent, daily risks; each daily risk was computed from a daily accumulated pathogen dose from all relevant activities (*e.g.*, clothes washing and toilet flushing). Eq. (1) was modified to include accidental ingestion of treated non-potable water (or cross-connection with potable water) by summing the annual probabilities of infection for populations with and without accidental ingestion, weighted by the relevant fraction of the population.

Pathogen concentrations were characterized using probability distributions based on literature values (described in Section 2.5) or alternative characterizations (described in Section 2.6.1). The remaining exposure and dose-response assessment parameters (described in Sections 2.2 and 2.4) were fixed at expected or best-estimate values. Please refer to the Supporting Information (SI) Table SI1 for a summary of how the input variables in Eq. (1) were treated.

2.2. Exposure routes

The selected uses included: (1) potable consumption; (2) toilet flush water; (3) unrestricted irrigation use (*i.e.*, dust suppression and municipal irrigation, excluding food crops); and (4) indoor use (*i.e.*, toilet flush water, clothes washing, and accidental cross-connection of treated water to potable water or accidental ingestion of treated water). The assumed volume of water consumed during each activity (for healthy adults), the frequency of use, and the fraction of the population exposed are presented in Table 1.

The volume of water inhaled after toilet flushing is potentially very small, *e.g.*, 10^{-9} L (Lim et al., 2015). The total volume of water ingested due to other routes of exposure such as hand contact with bathroom surfaces during cleaning or repair activities; hand contact during clothes washing; and unrestricted irrigation remains uncertain due to lack of data. We selected best-estimate,

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Table 1
Use characteristics for healthy adults[a].

Activity	Ingested volumes per day (L)	Use days per year	Fraction of population
Toilet flush water	3×10^{-5}	365	1
Clothes washing	1×10^{-5}	365	1
Unrestricted irrigation and dust suppression	1×10^{-3}	50	1
Cross-connection of treated water with potable water	2	1	0.1
Potable consumption	2	365	1

[a] Values adopted from NRMMC et al. (2006); we assumed increased frequency of clothes washing (to simplify the calculations) and increased volume and fraction of the population for the cross-connection event

fixed ingestion volumes from the QMRA literature and explored alternative values in a separate analysis (described in Section 2.6.1).

Using the de Man et al. (2014a) estimates of the water ingested from wet hand to mouth exposure for children, we estimated that one second of exposure could result in ingested volumes of 2×10^{-5} to 3×10^{-4} L. We adopted values from the Australian guidelines for water recycling (Table 1; NRMMC et al., 2006); being 3×10^{-5} L per day for toilet use (3 flushes $\times 10^{-5}$ L per flush) and 1×10^{-5} L per day for clothes washing. For unrestricted irrigation and dust suppression, we assumed that 1×10^{-3} L was ingested 50 times per year. The frequency was selected for a dry climate (*e.g.*, San Francisco) and may not be applicable to a climate with less irrigation needs. The ingested volume of 1×10^{-3} L is equivalent to 10–100 s of hand to mouth exposure (de Man et al., 2014a) or one drop of water (de Man et al., 2014b). We assumed 100% partitioning and/or recovery for aerosol or fomite-hand-mouth exposures, and thus a partitioning coefficient was not included in Eq. (1).

The volume consumed during a cross-connection or accidental ingestion event corresponds to one day of potable consumption. The number of days with accidental ingestion or cross-connection and fraction of the population exposed is highly uncertain and likely variable. Approximately 6.5% of the population is under the age of five, a potential target age group for accidental ingestion (United States Census Bureau, 2011). We selected uncertain input values from the low end of the possible ranges and explored alternatives in a separate analysis (described in Section 2.6.1).

2.3. Reference pathogens

Reference hazards represent classes of pathogens with potential adverse health impacts. Potential reference pathogens have been reviewed for wastewater (Soller et al., 2010a), blackwater and greywater (WHO, 2006a, 2006b), stormwater (NRMMC et al., 2009), and rainwater (Chapman et al., 2008). For a screening-level assessment, we initially considered a number of human-infectious enteric viruses, bacteria and protozoa (*i.e.*, human adenoviruses (*Mastadenovirus*), *Norovirus, Rotavirus, Campylobacter jejuni, Salmonella enterica, Escherichia coli O157: H7, Giardia lamblia*, and *Cryptosporidium* spp.) from human or animal fecal contamination. However, the final reference pathogens for each water type were limited to those with available observations. Although opportunistic pathogens are also important to manage in engineered water systems using alternative waters (Garner et al., 2016; Ashbolt, 2015), we limited our analysis to enteric pathogens since opportunistic pathogens largely proliferate post-treatment and need different management strategies (*e.g.*, ASHRAE 188-2015 (2015)).

2.4. Pathogen dose-response

We selected commonly used dose-response models that relate healthy adults' dose to a probability of infection based on ingestion or for adenoviruses, ingestion and inhalation (Table 2). There is only one peer-reviewed model option for *Giardia lamblia* (Haas et al., 1999). For *Rotavirus*, the various options are relatively similar (Teunis and Havelaar, 2000). For *Campylobacter* spp., two dose-response relationships (discussed by Schmidt et al. (2013a)) were initially considered. The dose-response proposed by Teunis et al. (2005), incorporating outbreak data, was not selected given the problem of structural identifiability when using outbreak data to estimate the dose-response parameters when dose is also uncertain (described by Schmidt (2015)) and the exposed population was largely children rather than healthy adults. For the remaining pathogens, there are multiple dose-response models, with different mechanistic assumptions and dose-response data, but which may not necessarily address the range in pathogen or host effects that impact the dose-response. Please refer to Messner and Berger (2016), Van Abel et al. (2016), Bambic et al. (2011), and U.S. EPA (2014) for a full discussion and description of the issues related with selecting and using these models.

For *Norovirus,* human adenoviruses, and *Cryptosporidium* spp., we adopted dose-response models that (at the time of publication) are less commonly applied in QMRA, in addition to those routinely used. The *Mastadenovirus* dose-response model for oral ingestion proposed by Teunis et al. (2016) combines dose-response information from inhalation, oral ingestion, intranasal, and intraocular droplet inoculation by *Mastadenovirus* Types 4, 7, and 16. The model is described and compared to preceding models in the Supporting Information, Table SI2. For *Norovirus*, two dose-response models were selected to represent the lower and upper bounds of predicted risk across the range of available models (Van Abel et al., 2016). The upper bound, developed by Teunis et al. (2008), is generally used in QMRA and predicts relatively high risks among the available models in the relevant dose range. The lower bound, proposed by Messner et al. (2014), predicts similar risks as the majority of the published *Norovirus* dose-response models with good empirical fit to the available data (reviewed in Van Abel et al. (2016)) and is easy to compute. However, neither model includes data for the low-dose exposures anticipated from non-potable water use, and the true dose-response relationship at these levels of exposure remains uncertain.

For *Cryptosporidium* spp., we adopted the fractional Poisson model proposed by Messner and Berger (2016) based on a comparison of six possible model forms, including exponential that address *C. parvum, C. hominis*, and *C. muris*. This model (along with the updated dose-response dataset) results in risks that are much greater than previously predicted using the exponential model from the U.S. EPA Long Term 2 Enhanced Surface Water Treatment Rule (LT2) Economic Analysis (Messner and Berger, 2016). However, Schmidt and Chappell (2016) raised concerns about the biological assumptions and model fit of the model proposed by Messner and Berger (2016). The final pathogen reduction targets address model uncertainty for *Norovirus* and *Cryptosporidium* spp. by presenting an upper bound LRT, lower bound LRT, and an "averaged" target that randomly weights the lower and upper bound models using a uniform distribution of weights (Soller et al., 2016a).

We adopted the conservative assumption that 100% of the population of healthy adults is susceptible to all pathogens (*i.e.*, $S = 1$

Table 2
Pathogen dose-response relationships.

Reference pathogen	Model	Parameters	Parameter values	Units	Reference	Susceptible fraction
Norovirus (GI)	hypergeometric	alpha	0.04	genome copies	(Teunis et al., 2008)	1
		beta	0.055			
Norovirus (GI & GII.4)	fractional Poisson	*P*	0.72	genome copies	(Messner et al., 2014)	1
		U	1106			
Mastadenovirus Type 4, 7, and 16	hypergeometric	alpha	5.24	TCID50	(Teunis et al., 2016)	1
		beta	2.95			
Rotavirus	approximate beta-Poisson	alpha	0.2531	FFU	(Haas et al., 1999)	0.06
		beta	0.4265			
Campylobacter jejuni	approximate beta-Poisson	alpha	0.145	CFU	(Medema et al., 1996)	1
		beta	7.589			
Salmonella enterica	approximate beta-Poisson	alpha	0.3126	CFU	(Haas et al., 1999)	1
		beta	2884			
Giardia lamblia	exponential	*r*	0.0199	cysts	(Rose et al., 1991)	1
Cryptosporidium spp.	fractional Poisson	*P*	0.737	oocysts	(Messner and Berger, 2016)	1
Cryptosporidium spp.	exponential	*r*	0.09	oocysts	(U.S. EPA, 2005)	1

in Eq. (1)), with one exception. For *Rotavirus*, we used a dose-response model for healthy adults but assumed that only young children were susceptible (WHO, 2011). The assumed susceptible fraction (Table 2) is likely high given widespread vaccination of young children. Note that we assumed that 100% of the population is susceptible to noroviruses given that susceptibility is not fully understood across genotypes (*e.g.*, Se– individuals have been infected from genotypes GII.4 and GI.8) (Van Abel et al., 2016).

2.5. Characterization of pathogens in waters using observations

We used multiple techniques to estimate pathogen concentrations in waters using the available literature, depending on the type of information available for each water source. Pathogen observations were used to characterize pathogen concentration and occurrence in the untreated source waters when the data met the following criteria:

1 Analytical methods used to enumerate the pathogens were comparable to those used in the dose-response studies (*i.e.*, "conventional" methods for all reference pathogens except *Norovirus*); and
2 If a large fraction of the samples were non-detects, the limit of detection was specified.

In addition, approaches that modeled the pathogen concentration in source waters were used (described below). The data used to characterize the pathogen concentrations are reported in Table 3.

2.5.1. Greywater

In the absence of a greywater study with sufficient pathogen monitoring data described in the literature, we adopted an epidemiology-based approach to describe distributions of pathogen concentrations in laundry, bathroom sink, and shower/bath greywaters (Jahne et al., 2016). The epidemiological approach used data describing population illness rates (as a surrogate for infection) and shedding characteristics. Two different collection system sizes were selected, the five and 1000-person collection systems, since pathogen occurrence and densities in local greywater have scaling effects due to sporadic pathogen occurrence and lack of dilution effects in smaller populations. The probabilistic approach captured the variation of fecal contamination, pathogen shedding, and pathogen incidence across collection locations and/or shedding individuals.

2.5.2. Stormwater

Four studies with quantitative data on pathogen concentrations in stormwater were identified (NRMMC et al., 2009; Lim et al., 2015; de Man et al., 2014a; Bambic et al., 2011; McBride et al., 2013), with one from the United States, reported in Bambic et al. (2011) and McBride et al. (2013). We selected a subset of the watersheds in McBride et al. (2013): residential, commercial/light industrial, mixed use, and dry season urban runoff (described in Table SI3); and two characterizations of the pathogen concentration: the hockey stick (see McBride et al. (2013) Appendix A for a full description) and lognormal distributions (presented in Table SI4).

To compliment the LRTs based on pathogen observations, we also estimated the LRTs for a range of dilutions of municipal wastewater; those representative of the LRTs for the monitored watershed types were selected. By modeling stormwater in this way, we have attempted to reduce dependency on the available monitoring data; in addition, this generalization aids decision makers with the range of likely impact from human contamination in their stormwater.

We adopted the lognormal characterization of *Norovirus* GII in raw wastewater by Pouillot et al. (2015) based on 566 observations from wastewater influent from across the world and across seasons. Note that Pouillot et al. (2015) characterized *Norovirus* GII and GI separately, possibly resulting in a lower treatment requirement than for a combined GI and GII characterization.

For the remaining pathogens, we adopted the $\log_{10}$uniform characterizations by Soller et al. (2016b), which targeted wastewaters in North America. In contrast to *Norovirus*, the limited number of observations in peer-reviewed publications of the remaining reference hazards, particularly within North America, made it difficult to select data-driven characterizations. Log_{10}uniform distributions seemed reasonable given that we are exploring the range of pathogen occurrence (and resulting treatment requirements) in human-impacted stormwater, rather than characterizing pathogen occurrence with the purpose of specifying LRTs for wastewater.

There was limited data for the human adenoviruses and the bacterial reference pathogens in North American wastewater; therefore, international data was also used (Table 3). We modified the *Salmonella* spp. distribution from Soller et al. (2016b) by increasing the upper bound based on data reported by Bonadonna et al. (2002) in samples collected after an activated-sludge biological tank. In addition, we decreased the upper bound of the *Cryptosporidium* spp. distribution (using data from Madore et al. (1987)) after finding an error in the reporting of data from Gennaccaro et al. (2003) across manuscripts (Soller et al., 2016b; Yang et al., 2015; Crockett, 2007).

2.5.3. Roof runoff

Site-specific roof runoff pathogen concentrations are critical to estimate accurate LRTs for roof runoff, given the variety of

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Table 3
Pathogen concentration inputs.

Pathogen or indicator	Source[a]	Unit	$\log_{10}$- transformed concentration #/g or #/L[b]	No. positive (total) or rate[c]	Reference
Norovirus GII	WW	genome copies	N(3.9,1.4)	360 (566)	(Pouillot et al., 2015)
Mastadenovirus	WW	infectious units	U(1.75,3.84)	24 (30), 10 (10)	(Soller et al., 2016b; Hewitt et al., 2011; Hurst et al., 1988)
Cryptosporidium	WW	oocysts	U(−0.5,3.72)	NR	(Soller et al., 2016b; Madore et al., 1987; Yang et al., 2015; Crockett 2007; Nasser, 2015)
Giardia	WW	cysts	U(0.5,4.0)	NR	(Soller et al., 2016b; Wallis et al., 1996; Sykora et al., 1991; Harwood et al., 2005)
Campylobacter	WW	MPN	U(2.95,4.60)	15 (15)	(Soller et al., 2016b; Stampi et al., 1993)
Salmonella	WW	MPN	U(0.47,5.70)	9 (9)	(Soller et al., 2016b; Bonadonna et al., 2002; Lemarchand and Lebaron, 2003)
Norovirus	GW-5	genome copies	Empirical(3.11,5.82)	2.8%	(Jahne et al., 2016)
	GW-1000		Empirical(5.53,4.57)	99.7%	(Jahne et al., 2016)
Rotavirus	GW-5	FFU	Empirical(5.56,4.59)	0.1%	(Jahne et al., 2016)
	GW-1000		Empirical(3.32,4.82)	19.8%	(Jahne et al., 2016)
Cryptosporidium	GW-5	oocysts	Empirical(2.84,1.83)	0.1%	(Jahne et al., 2016)
	GW-1000		Empirical(0.99,1.87)	11.3%	(Jahne et al., 2016)
Giardia	GW-5	cysts	Empirical(2.06,2.05)	0.6%	(Jahne et al., 2016)
	GW-1000		Empirical(0.84,2.02)	69.8%	(Jahne et al., 2016)
Campylobacter	GW-5	CFU	Empirical(2.28,2.25)	0.1%	(Jahne et al., 2016)
	GW-1000		Empirical(1.13,2.85)	27.3%	(Jahne et al., 2016)
Salmonella	GW-5	CFU	Empirical(3.04,1.93)	0.1%	(Jahne et al., 2016)
	GW-1000		Empirical(1.11,2.16)	23.2%	(Jahne et al., 2016)
Fecal coliforms	RR	CFU	N(2.80,0.92)	12 (12)	(Chapman et al., 2008)
Fecal coliforms	gull feces	CFU	N(8.30,0.95)	25 (25)	(Lévesque et al., 2000)
Campylobacter	gull feces	CFU	U(3.3,6.0)	25 (25)	(Schoen and Ashbolt, 2010; Lévesque et al., 2000)
Salmonella	gull feces	CFU	U(2.3,9.0)	24 (25)	(Schoen and Ashbolt, 2010; Lévesque et al., 2000)

[a] Wastewater (WW), Greywater (GW) 5 or 1000-person collection system, roof runoff (RR). Stormwater is presented in Supplemental Information Table SI4
[b] The $\log_{10}$ transformed concentrations were used to characterize the Normal (N) with parameters mean and standard deviation; Uniform (U) with parameters upper and lower bound; and empirical with summary statistics median when occurring, and net mean including non-occurrences
[c] The positive rate is 100 ×(No. positive samples/total samples) from the Monte Carlo analysis; NR = not reported

climatic conditions across the U.S., the range of pathogen occurrence across sites, and potential differences in animal populations (Ahmed et al., 2012). A literature review of roof-collected rainwater revealed no study within the continental U.S. with sufficient pathogen and/or fecal indicator monitoring data to be useful for our analysis. The most comprehensive pathogen datasets were from Southeast Queensland, Australia (Ahmed et al., 2010, 2012). However, the data did not meet our adopted criteria since non-conventional analytical and enumeration methods were used and a large percentage of samples were non-detects (95–90%, depending on pathogen), for which the limit of detection in roof runoff was not specified. Previous risk assessments using this data (by Ahmed et al. (2010) or Lim and Jiang (2013)) assumed either that non-detects represented an absence of pathogens or wrongly assumed a detection limit of 5 gc L^{-1}. In addition, our aim was to characterize fresh roof runoff, rather than stored roof runoff, to provide a conservative (or protective) LRT. Moreover, only two rainfall events were captured during the observation period; in comparison, there were five to seven storm events for each stormwater type.

Given the lack of roof runoff data, a conservative modeling approach was adopted to estimate the mass of fresh animal fecal contamination in roof runoff using fecal indicator measurements. The use of indicators to estimate fecal mass has been employed in QMRA given the absence of pathogen observations (*e.g.*, see Jahne et al. (2016) for greywater and Petterson et al. (2016) for stormwater). We consider this approach to be conservative because fecal indicators are consistently detected in fresh roof runoff, whereas, pathogens are not (Ahmed et al., 2011). Furthermore, we assumed that all of the fecal indicators in the roof runoff resulted from fresh fecal contamination. However, this approach is limited in ability to predict pathogen densities given differences in inactivation (or growth) between indicators and pathogens (Ahmed et al., 2014) and the aforementioned differences in occurrence. Therefore, our approach is most applicable to bacterial pathogens when using fecal bacterial indictors due to potential differences in die-off of protozoan and viral pathogens on the roof (Ahmed et al., 2014).

Thus, using an approach adopted from Schoen and Ashbolt (2010), we calculated the pathogen concentration in roof runoff ($C_{RR,p}$) as:

$$C_{RR,p} = \frac{C_{RR,FIB}}{C_{F,FIB}} \times C_{F,p} \times I_{F,p} \quad (2)$$

where

$C_{RR,FIB}$ and $C_{F,FIB}$ are the fecal indicator bacteria (*FIB*) concentrations in roof runoff (*RR*) or feces (*F*)
$C_{F,p}$ is the pathogen (p) concentration in feces (*F*)
$I_{F,p}$ is the fraction of human-infectious pathogenic strains in feces (*F*)

We characterized the $\log_{10}$-transformed fecal coliform concentration in roof runoff as normal using data from six rainwater tanks in Sydney, Australia up to a week after rainfall (Chapman et al., 2008). We assumed the FIB in the roof runoff resulted from the deposition of feces from one animal type, and all of the pathogens in the feces were human-infectious ($I_{F,p} = 1$).

Ideally, the approach in Eq. (2) should be implemented for all relevant animals that are likely contributors of fecal contamination to rooftops in North America, *e.g.*, seagulls, possums, squirrels, rodents, and bats. Of this list, we were able to characterize pathogen concentrations in the feces of seagulls. We adopted the pathogen concentration characterization from Schoen and Ashbolt (2010) and characterized the $\log_{10}$-transformed fecal coliform concentration in gull feces based on composite samples from Lévesque et al. (2000), representing a population of possible infected and non-infected individual gulls. They collected fresh fecal samples six or seven times across three bird colonies near Quebec, Canada from April to mid-July. Since pathogens were detected in nearly all of the composite samples (Table 3), we assumed that pathogens were always present in roof runoff from birds. Normal distributions of the fecal coliform concentrations were selected after visual inspection of

the normal q-q plot showed minimum variation from the theoretical quantiles. Overall, the lack of pathogen concentration data in fresh roof runoff and feces across relevant animals, limits our ability to draw conclusions about protective LRTs for roof runoff.

2.6. Risk characterization

A Monte Carlo analysis was implemented to capture the natural variation in pathogen concentration across time and collection locations (the remaining exposure and dose-response parameters were fixed at the best-estimate values; refer to Table SI1). For each combination of health benchmark, water type, pathogen, and water use, we simulated 10,000 Monte Carlo iterations in R 3.2.3 (R Core Team, 2015) using Eq. (1). For each iteration, we simulated 365 daily pathogen concentrations (for greywater, the daily concentration could be zero). A random subset of the 365 concentration values were used based on the number of use days each year (Table 1). The R function uniroot was used to solve Eq. (1) for the LRT. This function searches a user defined interval for a root of the selected nonlinear function. The 95th percentile LRT (upper tail LRT), rounded to one decimal point, was reported. The LRTs were then used as input into Eq. (1) to double check the predicted annual risk; LRTs resulted in annual infection risks less than or equal to 1.1×10^{-4}, due to rounding.

The 95th percentile LRTs do not account for uncertainty in the dose-response relationships. Instead, we recalculated the LRTs using two dose-response models for *Norovirus* and *Cryptosporidium* spp. to capture the range of prediction (Section 2.4). A separate analysis was performed to evaluate the change in the indoor use LRT for selected source waters due to changes in the fraction of the population exposed to accidental ingestion (or cross-connection of potable water with treated source water) and the number of event days per year.

2.6.1. Alternative exposure scenario analysis

To facilitate alternative assumptions and/or site-specific use, we recalculated the LRT for *Norovirus, Rotavirus, Cryptosporidium* spp., and *Campylobacter jejuni*, for a single use (*e.g.*, toilet flush or unrestricted irrigation) using Eq. (1) with alternative fixed values for ingested volume; number of days of exposure (*e.g.*, 1, 10, and 365); as well as alternative pathogen concentration characterizations. Pathogen LRTs were then plotted as a function of these parameters to allow determination of LRTs for alternative input values (Section 3.3). The ingested volume range of 1×10^{-7} L (*i.e.*, possible aerosolized volume) to 1×10^{-3} L (*i.e.*, roughly one drop) was selected based on the literature reviewed in Section 2.2; these values may not capture the full range of non-potable ingestions and remain highly uncertain. We assumed that the $\log_{10}$-transformed pathogen concentration in the source water was normally distributed. The selected pathogen means and standard deviations captured the range observed across pathogens and waters in the literature reviewed in Section 2.5, but also included densities less than those observed and corresponding to a predicted LRT of less than or equal to zero (*i.e.*, the critical density characterization for which no treatment is required 95% of the time). The analysis of alternative scenarios is applicable to any type of water with the selected pathogen characterization.

3. Results

The 95th percentile $\log_{10}$ pathogen reduction targets (LRTs) for each use, using the available monitoring data accounting for pathogen concentration variation across time, are presented in Table 4 for healthy adults given the 10^{-4} ppy (infection) benchmark and Table SI5 for selected waters and pathogens given the 10^{-2} ppy (infection) benchmark. The simulated pathogen concentrations in source waters used to calculate the LRTs in Table 4 are further described in Supporting Information, Tables SI7-9. Alternatively, LRTs for alternative exposure scenarios are presented in Figures SI1–6 for the benchmark infection risk of 10^{-4} ppy.

3.1. LRTs using observed or modeled pathogen concentrations

LRT results in Table 4 are presented for greywater for a 1000-person collection system, greywater for a 5-person collection system, stormwater with low dilution (10-fold) of raw municipal wastewater, stormwater with moderate dilution (1000-fold) of raw municipal wastewater, and roof runoff. LRTs for human-impacted stormwaters observed across the United States reported by McBride et al. (2013) are presented in Supporting Information Table SI5. The low dilution (*i.e.*, 10^{-1}) LRTs for stormwater roughly align with the maximum LRTs for *Norovirus* and *Giardia lamblia* (using the lognormal pathogen characterization) for stormwaters observed across the United States (Table SI5); whereas, the high dilution (*i.e.*, 10^{-3}) LRTs roughly align with the maximum LRTs using the hockey stick distribution (the recommended distribution by McBride et al. (2013)) for *Norovirus, Cryptosporidium, Giardia lamblia*, and *Salmonella*. Therefore, we believe that the 10^{-1} dilution could be considered a conservative LRT for stormwater.

The predicted 95th percentile LRTs indicated that treatment was required across waters, uses, and health benchmarks. The 95th percentile LRTs corresponding to the 10^{-2} ppy (infection) benchmark (Table SI6) were approximately 2 units less than the LRTs corresponding with the 10^{-4} ppy (infection) benchmark (Table 4). The predicted LRTs generally increased in magnitude across the selected uses for toilet flush water, unrestricted irrigation, indoor use, and drinking. Based on the selected assumptions, the only water with no 95th percentile treatment requirement (*i.e.*, water is acceptable for use without treatment 95% of the time) across uses was the greywater from a 5-person collection system for reference protozoa and bacteria; however, the 99th percentile LRT for these reference pathogens was approximately that of the greywater 1000-person collection and thus greater than zero. The 99th percentile LRTs, calculated (but not presented) for a subset of reference pathogens and waters, were nearly identical to the 95th percentile LRTs for *Cryptosporidium* and *Campylobacter jejuni* in stormwater, and less than 1 unit different for *Norovirus* in these waters.

The LRTs for indoor use using alternative scenarios of accidental ingestion or potable water cross-connection events are presented in Table 5 for selected source waters and the benchmark infection risk of 10^{-4} ppy. The indoor use LRT was lower than, but approaching, the LRT for potable use when we assumed that half of the population was exposed to accidental ingestion (or cross-connection) for half of the year.

3.2. Reference pathogens

Comparing reference pathogens within each class, the LRTs for *Campylobacter jejuni* were generally greater than *Salmonella* across waters (with the exception of roof runoff) and uses using the dose-response models proposed by Haas et al. (1999). For parasitic protozoa, the LRTs for *Cryptosporidium* using two selected dose-response models (Table 2) were generally greater than targets for *Giardia lamblia* using the dose-response model recommended by Rose et al. (1991). Infectious *Rotavirus*, and for some uses, adenoviruses targets fell between the lower and upper bound *Norovirus* targets (based on genome copy number qPCR estimates).

The LRTs for *Norovirus* and *Cryptosporidium* were sensitive to the selected dose-response function. The LRTs for *Norovirus* calculated using the upper bound dose-response, the hypergeomet-

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Table 4
95th percentile Log_{10} pathogen reductions targets for healthy adults corresponding to the 10^{-4} ppy (infection) benchmark.

	Norovirus (genome copies)[a]	*Mastadenovirus* (TCID50)	*Rotavirus* (FFU)	*Cryptosporidium* (oocysts)[b]	*Giardia* (cysts)	*Campylobacter* (CFU)	*Salmonella* (CFU)
Greywater 1000-person collection							
Toilet flush water	7.8/7.5/5.0	NR	5.8	4.0/3.7/3.1	2.8	3.2	0.6
Unrestricted irrigation	8.4/8.1/5.6	NR	6.4	4.5/4.2/3.6	3.4	3.7	1.2
Indoor use[c]	8.8/8.5/6.0	NR	6.4	4.5/4.2/3.6	3.8	3.7	1.6
Drinking	12.6/12.3/9.8	NR	10.6	8.8/8.5/7.9	7.6	8.0	5.4
Greywater 5-person collection[d]							
Toilet flush water	7.6/7.3/4.8	NR	NR	0/0/0	0	0	0
Unrestricted irrigation	7.7/7.4/4.9	NR	NR	0/0/0	0	0	0
Indoor use	7.8/7.5/5.0	NR	NR	0/0/0	0	0	0
Drinking	12.4/12.0/9.5	NR	NR	0/0/0	0	0	0
Stormwater[e] *– 10^{-1}*							
Toilet flush water	7.3/7.0/4.5	4.1	NR	3.8/3.7/2.8	2.5	3.4	1.8
Unrestricted irrigation	8.0/7.7/5.1	4.8	NR	4.5/4.4/3.6	3.3	4.1	2.6
Indoor use	8.3/7.9/5.4	5.9	NR	5.7/5.5/4.8	4.5	5.1	3.8
Drinking	12.1/11.7/9.3	8.9	NR	8.6/8.5/7.7	7.4	8.2	6.6
Stormwater[e] *– 10^{-3}*							
Toilet flush water	5.3/5.0/2.5	2.1	NR	1.8/1.7/0.8	0.5	1.4	0
Unrestricted irrigation	6.0/5.7/3.2	2.8	NR	2.5/2.4/1.6	1.3	2.1	0.6
Indoor use	6.2/5.9/3.4	3.9	NR	3.7/3.5/2.8	2.5	3.1	1.8
Drinking	10.1/9.8/7.3	6.9	NR	6.6/6.5/5.7	5.4	6.2	4.6
Roof runoff[f]							
Toilet flush water	NR	NR	NR	NR	NR	2.4	2.9
Unrestricted irrigation	NR	NR	NR	NR	NR	3.1	3.5
Indoor use	NR	NR	NR	NR	NR	3.3	3.5
Drinking	NR	NR	NR	NR	NR	7.3	7.7

[a] Hypergeometric model (HG)/averaged results of HG and FP/fractional Poisson (FP) model results; NR is not reported due to either lack of pathogen data or unstable Monte Carlo simulation results
[b] Fractional Poisson/averaged results/exponential model results
[c] Assumes 10% of the population is exposed to accidental ingestion of treated water or a cross-connection event each year
[d] The 99th percentile log_{10} reduction for protozoa and bacteria is greater than zero and approximately equal to the 95th percentile target log_{10} reductions for the 1000-person system
[e] Calculated using dilutions of municipal wastewater with dilution values selected based on predicted LRTs from the observational data presented in Table SI5
[f] Calculated using the animal feces approach (Eq. (2)) with seagulls as the selected animal and fecal indicator concentration in stored roof runoff

Table 5
Sensitivity analysis of 95th percentile log_{10} pathogen reduction targets for indoor use to accidental ingestion and potable water cross-connection events, for the infection benchmark of 10^{-4} ppy.

Fraction of the population exposed	Number of days with cross-connection to or accidental ingestion of treated alternative water		
	1	50	182
Norovirus (genome copies)[a]			
Greywater – 1000 people			
0.5	9.4/9.1/6.5	11.4/11.1/8.5	12.0/11.7/9.2
0.1	8.8/8.5/6.0	10.7/10.4/7.9	11.3/11.0/8.5
Campylobacter (CFU)			
Stormwater – 10^{-1} dilution			
0.5	5.8	7.1	7.6
0.1	5.1	6.4	6.9

[a] Hypergeometric model/averaged results/fractional Poisson model results.

ric model proposed by Teunis et al. (2008), were approximately three units greater than the LRTs calculated using the lower bound, the fractional Poisson model proposed by Messner et al. (2014). Whereas the LRTs for *Cryptosporidium* calculated using the fractional Poisson model proposed by Messner and Berger (2016) were approximately one unit greater than the LRTs calculated using the exponential model (U.S. EPA, 2005).

3.3. Alternative scenario analysis

A sample analysis of alternative exposure scenarios is presented in Fig. 1 for *Campylobacter jejuni* and the benchmark infection risk of 10^{-4} ppy (additional reference hazards and conditions are presented in Figures SI1-6). The reductions were calculated assuming that the log_{10}-transformed pathogen concentration was distributed normally, and do not apply when a pathogen is characterized by sporadic occurrence over time (*e.g.*, for the 5-person greywater collection) or for a use that affects only a fraction of the population. The y-axis plots the 95th percentile pathogen LRT using the best-estimate dose-response parameters. The x-axis intersection for each line is the critical concentration (*i.e.*, mean pathogen concentrations below this value do not require treatment for the selected assumptions). For example, for activities that occur daily with an ingested volume of about a drop of water (*i.e.*, 10^{-3} L), the critical density for *Campylobacter jejuni* was between 3×10^{-2} and 3×10^{-5} CFU L^{-1}, for waters without and with pathogen variation, respectively. The critical densities for *Rotavirus* were similar to *Campylobacter jejuni*; less for *Cryptosporidium* (using both dose-response models); and highly variable for *Norovirus*, depending on the selected dose-response model (Figs. SI1–6).

Based on Fig. 1 and SI1–6, there was approximately a three log_{10} unit increase in LRT when the standard deviation (sd) of the log_{10} transformed *Campylobacter* concentration increased from 0.0 (no variation) to 1.5 (*i.e.*, the highest observed pathogen sd in source material (Table 3)). Whereas, an order of magnitude increase in the number of exposures, the volume ingested, the mean pathogen concentration or the benchmark risk (refer to Table 4 *vs.* Table SI6) resulted in roughly a one unit increase in the 95th percentile pathogen LRT.

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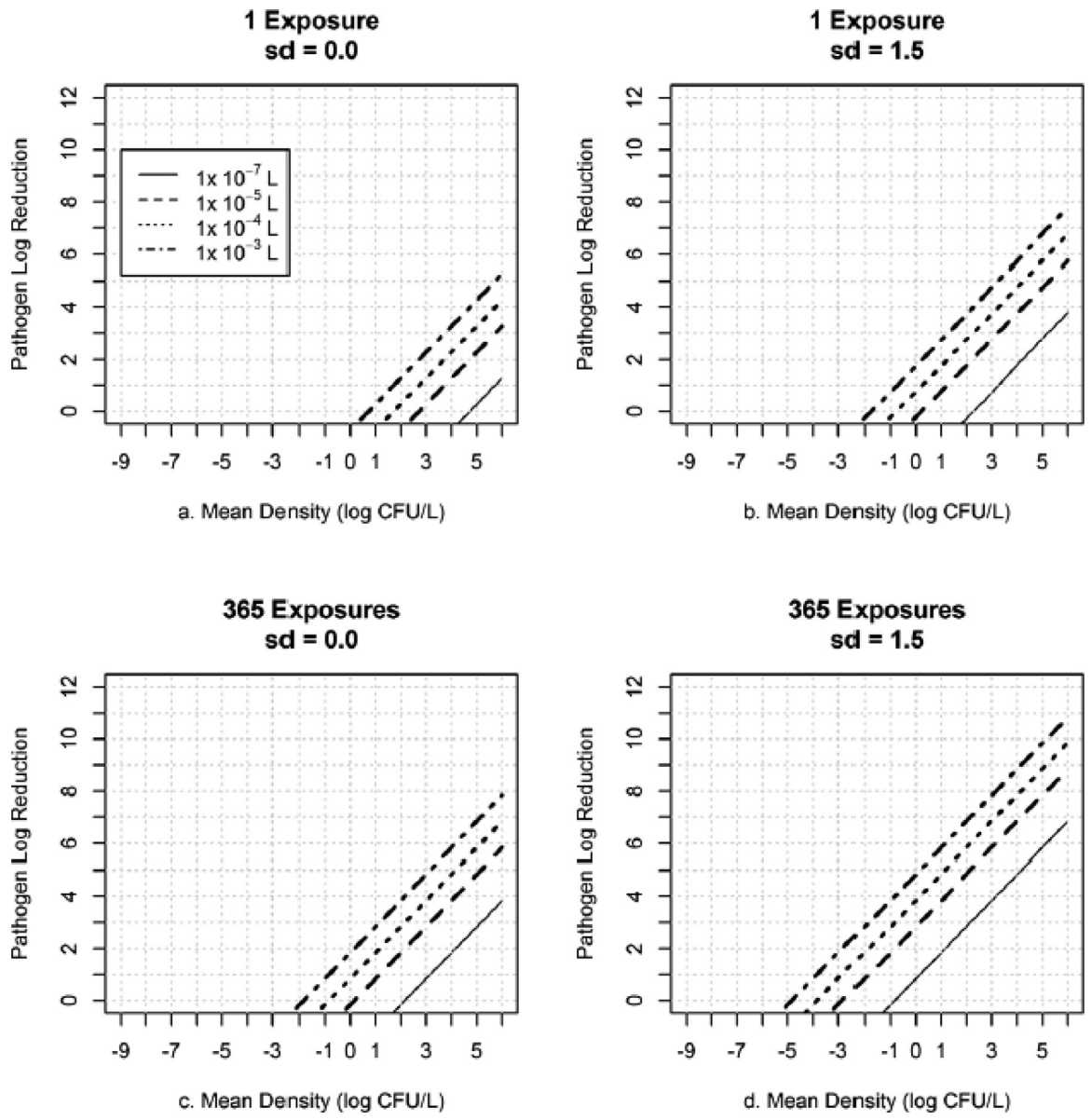

Fig. 1. *Campylobacter jejuni* 95th percentile $\log_{10}$ reduction targets for daily ingestion of a specified volume (lines), corresponding to the 10^{-4} ppy infection risk benchmark. The x-axis corresponds to the mean of the $\log_{10}$-transformed *Campylobacter jejuni* concentration. (a)/(c) include no pathogen concentration variation (*i.e.*, standard deviation (sd) of the $\log_{10}$-transformed concentration); (b)/(d) include variation. (a)/(b) correspond to exposure frequencies of one day and (c)/(d) of 365 days.

4. Discussion

As expected, the difference in treatment requirements among source waters was driven by the microbial quality of the water – both the density and occurrence of the pathogens. The treatment requirements for greywater from collection systems with 1000 people generally had the greatest LRTs; however, those for greywater collected from smaller populations, which have less frequent pathogen occurrences, were lower. Stormwater had highly variable microbial quality, which resulted in a range of possible treatment requirements. The microbial quality of roof runoff, and thus the resulting LRTs, remains uncertain due to lack of relevant data for protozoan and bacterial pathogens. The predicted pathogen densities for locally-collected wastewater in a 1000-person system in Jahne et al. (2016) were roughly two orders of magnitude greater than those for greywater (Table SI 20). Thus, the LRTs for locally-collected wastewater were roughly two orders of magnitude greater than greywater (Table SI 21).

4.1. What level of protection does a LRT ensure?

If a treatment system can maintain the level of treatment performance specified by the proposed LRTs (Table 4) at all times, the predicted probability of infection across the population of healthy adults will be less than 10^{-4} ppy for each pathogen for 95% of years for the given input assumptions. The LRT does not express the average treatment efficiency of a process; rather, the treatment efficiency of a process should be greater than or equal to the LRT at all times. The predicted LRTs do not ensure a tolerable level of infection for exposed sensitive populations, which may include those with increased exposures, due to occupation or behavior, or those with increased susceptibility to the hazards, *e.g.*, immunocompromised individuals or pregnant women. Generally, dose-response relationships and exposure volumes for sensitive groups/life-stages are missing for the selected reference hazards; thus, defining a LRT for these more sensitive groups using Eq. (1) was not possible.

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The LRTs for indoor use (Table 4) protect against accidental ingestion of treated water (assuming that 10% of the population is exposed to one event each year). The LRTs did not account for cross-connections between untreated waters and potable or non-potable waters. Similarly, the LRT did not account for sporadic treatment malfunction. If these additional "event" scenarios were included in the LRT calculation, the LRTs would increase in magnitude.

4.2. Under what conditions do the LRTs apply?

The LRTs reported in Table 4 apply to freshly collected waters (*i.e.*, not stored), since we did not account for pathogen die-off or growth in collection, for waters with pathogen concentrations similar to those assumed here. Site-specific stormwater LRTs may differ from those presented in Table 4 based on land use and the associated types of fecal contamination (Table SI3-5). Similarly, we may expect variation in fecal contamination and pathogen occurrence across collected roof runoff, particularly when impacted by animals other than seagulls. Greywater LRTs will also be affected by the types of water collected (*e.g.*, from sinks, showers, or laundry) and specific source characteristics (*e.g.*, use by children) (Jahne et al., 2016).

The LRTs for greywater may differ by region depending on the prevalence of respective pathogen infections across the community and other factors, much like municipal wastewater (Pouillot et al., 2015). Pouillot et al. (2015) reported a large variation in the mean *Norovirus* GII concentration in raw wastewater across locations with a 95th percentile mean concentration of roughly 5.9 $\log_{10}$ genome copies L^{-1} (the 50th percentile mean was 3.9 $\log_{10}$ genome copies L^{-1}). This example highlights the difficulty in prescribing one risk-based LRT for a type of source water due to variation across collection sites. Alternatively, LRTs could be adopted using Figs. 1 and SI1–6 for site-specific pathogen characterizations or for "bins" based on local pathogen monitoring data.

The LRTs reported in Table 4 apply to use scenarios with exposures similar to those assumed here. Figures SI 1–6 and Table 5 could also be used to select LRTs for different exposure assumptions (*i.e.*, different volume ingested, number of exposure events per year, and fraction of the population exposed to accidental ingestion/cross-connection).

4.3. Uncertainty

The LRTs for both potable and non-potable use remain uncertain due to limited data for certain exposure and dose-response inputs. Below, we discuss the major sources of uncertainty and suggest ways to reduce it through additional data collection.

4.3.1. Exposure assessment

The alternative exposure scenarios in Section 3.3 confirmed that variation in the pathogen density greatly influenced the LRTs. Generally, the pathogen density in all source waters was difficult to characterize using probability distributions due to lack of data, with the exception of noroviruses in wastewater. In addition, we assumed that all the pathogen strains or groups that were detected were human infectious due to lack of information. Updated characterization of these pathogen densities for a particular location could shift the LRTs, as demonstrated in Section 3.3. An estimate of the human-infectious pathogen density has been shown to decrease the predicted risk in waters with low human impact (Schoen and Ashbolt, 2010; Soller et al., 2010b; Lapen et al., 2016) and thus could decrease the treatment requirements for roof runoff or some stormwaters.

The ideal dataset for conducting QMRA from observations of pathogen density (or fecal contamination) in alternative waters would:

1. monitor multiple locations over time;
2. be freshly-collected (*i.e.*, sampled before entering storage);
3. use (or be translated into) conventional units;
4. target human infectious strains/groups; and
5. report raw data (Schmidt et al., 2013b; Schmidt and Emelko, 2011; Pouillot et al., 2013), the recovery, and limit of detection to properly characterize occurrence distributions.

Jahne et al. (2016) discussed additional suggestions for locally-collected greywater. In addition, if conducting QMRA using the animal fecal approach with indicators, the indicators should be selected to have similar fate and transport in the source water as each reference pathogen.

The exposure frequencies and ingested volumes were also identified as important inputs (Section 3.3) with outstanding uncertainty (as discussed in Section 2.2). Additional data is required to inform the dominant routes of exposure, frequencies, and volumes for indoor use and unrestricted irrigation. Sinclair et al. (2016) demonstrated a method for estimating non-potable exposures from sprays that could be used to improve the volume assumptions. As additional information about exposure becomes available, *e.g.*, pathogen density or volume ingested, the alternative scenario analyses in Figs. SI 1–6 could be used to update the LRTs.

The pathogen partitioning between source water and aerosols and/or recovery from surfaces to fingers (to mouth) may also be important factors for non-potable exposure routes. Partitioning and recovery of pathogens can greatly reduce the pathogen dose ingested or inhaled when only a small fraction of the pathogens are transferred between media (Lopez et al., 2013; Schoen and Ashbolt, 2011). Given the overall lack of information about the dominant routes of exposure, we adopted best-estimate ingested volumes and assumed 100% partitioning/recovery. Due to the assumptions of 100% partitioning and human-infectious potential of pathogens, we generally consider the exposure assessment to be conservative (*i.e.*, resulting in protective LRTs) for the general population (see Section 4.2 for sensitive life-stages) for toilet flushing and unrestricted irrigation, especially for regions with less frequent irrigation needs, given a selected pathogen characterization. However, the exposure assessment for indoor use may not be conservative.

LRTs for indoor use in Table 4 were calculated assuming that accidental ingestion of treated non-potable water (or cross-connection) occurred one day a year for 10% of the population. The indoor use LRT was sensitive to the uncertainty associated with the frequency and occurrence of cross-connections or accidental ingestion (Table 5). This uncertainty is difficult to address given the limited adoption and monitoring of non-potable water systems (Storey et al., 2007).

4.3.2. Dose-response assessment

In the dose-response assessment, there are uncertainties that are difficult to resolve in the near term. Primarily, we estimated pathogen doses well below those tested in the dose-response studies. There remains great uncertainty about the dose-response relationship at these low doses (*e.g., Cryptosporidium* and *Norovirus* (Schmidt, 2015; Messner and Berger, 2016)). Furthermore, we assumed that the environmental strains or groups observed in the source waters had similar dose-response relationships to the reference pathogens listed in Table 2.

Although *Norovirus* is an important hazard in terms of illness prevalence in the United States (Scallan et al., 2011), there remains

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concern about the application of the *Norovirus* dose-response to environmental conditions when the ratio of infectious to non-infectious genome copies may differ from that in the inocula used in the dose-response studies (Van Abel et al., 2016). This concern seems most relevant for QMRA of stored (or treated) source waters, for which the age of the contamination varies considerably. In addition, as discussed in Section 2.4, limited *Norovirus* dose-response data has resulted in uncertainty surrounding the preferred dose-response relationship (Schmidt, 2015). Hence, we presented a spectrum of interpretations of the dose-response relationship using both an upper and lower bound LRT for *Norovirus* along with other reference viruses. Refer to Supporting Information (Table SI2) for discussion of available dose-response relationships for human adenoviruses and their impact on predicted LRTs.

4.4. Sporadic pathogen occurrence and greywater collection scale

The predicted LRTs for greywater were similar for the 1000 and 5-person collection systems for viruses, even though the pathogen characterization differed between scales (Jahne et al., 2016). The protozoan and bacterial LRTs for the 5-person collection system were 0.0 at the 95th percentile. However, the 99th percentile values were comparable to the 1000-person collection system 95th percentile LRTs. The lack of a 95th percentile treatment requirement for the 5-person collection system should not be interpreted as an absence of risk; rather, it is a result of infrequent predicted pathogen occurrence based on reported illness rates (Table 3).

We evaluated the effect of rare and sporadic pathogen occurrence on potable and non-potable LRTs (example in Figure SI7). Generally, a pathogen occurrence of 5% of days lowered the 95th percentile LRT by one unit compared to an occurrence of 100%. Pathogen occurrence could be captured in the Monte Carlo simulations of pathogen concentration used in Eq. (1) for roof runoff or stormwater given additional data and a clear limit of detection (see Section 4.3.1).

4.5. Other LRT efforts

For a complete discussion and summary of LRTs, please refer to Schoen and Garland (2015) for non-potable uses and Soller et al. (2016b) for potable use of wastewater. The potable LRTs based on a 10^{-4} ppy infection risk (Table 4) for alternative source waters were roughly equal to or less than the risk-based "12-10-10 Rule" from California's groundwater regulations concerning the potable reuse of wastewater which requires a 12-log removal of enteric viruses and a 10-log removal of *Cryptosporidium* and *Giardia* (CDPH, 2011). The LRTs for non-potable uses of greywater (1000-person) and stormwater (low dilution) based on a 10^{-4} ppy infection risk were greater than the 99.999% (5-log) removal for virus required under Title 22 in California (CDPH, 2014) for reuse of wastewater (5 to 6-log removals for virus are required by Australia (NRMMC et al., 2006)).

The non-potable LRTs for alternative source waters (NRMMC et al., 2009, 2008; WHO, 2006a) summarized in Schoen and Garland (2015) were based on a tolerable burden of disease of 10^{-6} DALYs ppy, which roughly corresponds to an infection risk of 10^{-3} ppy for *Cryptosporidium*, 7.2×10^{-4} ppy for *Campylobacter*, and roughly 10^{-4} ppy for *Rotavirus*. Therefore, direct comparison was not possible, except for the treatment requirements for enteric viruses. The predicted non-potable LRTs for enteric viruses for stormwater based on a 10^{-4} ppy infection risk (Table 4) are much greater than those proposed in NRMMC et al. (2009). This may be due to different LRT methods (see Section 2), virus dose-response, or pathogen characterization. Enteric viruses were not considered for roof runoff.

5. Conclusions

There are several implications from this work:

- The pathogen $\log_{10}$ reduction targets for direct potable use are likely overly conservative if applied to non-potable uses such as unrestricted irrigation or indoor use for toilet flushing and washing clothes;
- The pathogen $\log_{10}$ reduction targets across pathogens for municipal wastewater are likely overly conservative if applied to alternative waters such as stormwater, greywater, and roof runoff;
- The pathogen $\log_{10}$ reduction targets derived from QMRA for roof runoff remain highly uncertain due to the lack of available pathogen density data in the peer-reviewed literature;
- Defining general LRTs for a particular source water is complicated by the pathogen density variability across communities or collection systems; and
- To better characterize pathogen treatment needs, particularly for non-potable uses of alternative waters, additional data are needed on: 1) *in situ* pathogen density measurements; 2) the frequency and occurrence of accidental ingestion; 3) the volume of water ingested for non-potable purposes; and 4) pathogen dose-response relationships at low doses.

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Appendix A

The alternative benchmark annual risk of illness was calculated from the health benchmark for recreational water exposure of 32 illness cases per 1000 exposures by solving for the probability of illness per exposure x in $32/1000 = 1 - (1 - x)^{1000}$ and then finding the annual probability of illness $1 - (1 - x)^{365}$. The benchmark annual risk of infection was set equal to the calculated annual probability of illness. This is a conservative simplification given that a fraction of infections result in illness.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.mran.2017.01.002.

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