The use of Quantitative Microbial Risk Assessment to estimate the health risk from viral water exposures in Sub-Saharan Africa: A review

Nicole Van Abel[a*] and Maureen B Taylor[a,b]

[a]Department of Medical Virology, School of Medicine, Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa
[b] School of Health Systems and Public Health, Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa

*Corresponding author
Dr NA Van Abel
Department of Medical Virology
Faculty of Health Sciences
University of Pretoria
Private Bag X323
Arcadia
0007 Pretoria
South Africa
Telephone: +27 (012) 319 2534
Email: nvanabel@uw.edu

Short Title: Review of QMRA to assess risk of water exposure to viruses in Sub-Saharan Africa

Highlights

- Viral contamination in water in Sub-Saharan Africa (SSA) is summarized.
- Summary of QMRAs from exposure to virally-contaminated water in SSA.
- Viral concentration data as a main input for QMRA is summarized.
- QMRA is not widely adopted in Sub-Saharan Africa.

ABSTRACT

Access to microbiologically safe water is not a reality for many people throughout Sub-Saharan Africa where there is widespread occurrence of viruses in water sources. Exposure to this water can lead to adverse health risks including diarrhoeal disease. To a limited extent in Sub-Saharan Africa, the quantification of the human health risk associated with exposure to virally contaminated water has been done through the use of quantitative microbial risk assessment (QMRA). To understand the scope of the information available on this region, two systematic reviews were done to collect previously published literature from Sub-Saharan Africa on (1) prevalence and quantification of viral contamination in water and (2) QMRAs assessing the risk from exposure to water contaminated by viruses. The results of the 2 reviews were then summarised including, for the QMRAs, exposure and dose-response assumptions, input parameters, and risk outcomes. The results found the prevalence of 10 viruses (1-100%) in drinking, ground, irrigation, surface, and waste waters from eight countries with South Africa having the most information on water contamination by viruses. Quantified viral concentration data was reported for ~50% of the papers, for 6 viruses (entero-, human adeno-, noro-, rota-, sapo- and Hepatitis A virus), and ranged from (10^{-4}-10^{11} viruses/liter). Additionally, 22 QMRAs were identified for 6 viruses (entero-, human adeno-, noro-, rota-, coxsackie B, and Hepatitis A virus) from 4 countries demonstrating that QMRA has not been used extensively in this region. The majority of these QMRAs concluded that the risk of infection, illness, or Disability Adjusted Life Year (DALY) was exceptionally high and in excess of acceptable risk limits indicating a public health concern. In conclusion, water is contaminated with viruses, risk from exposure to viruses in water was extremely high for these 4 Sub-Saharan Africa countries, and QMRA is not a widely adopted methodology. Finally, some QMRA limitations were observed such as the need for more viral concentration data, collection of site- or region-specific exposure data, application of commonly used dose-response models, addressing susceptible populations such those with human immunodeficiency virus (HIV) infection in the risk characterisation, and access to free software.

Keywords: QMRA, water, enteric viruses, Sub-Saharan Africa, South Africa

INTRODUCTION

Worldwide numerous people do not have access to microbiologically safe water for drinking, cooking, or other domestic purposes (Gibson et al., 2011). It is estimated that ~1.8 billion people use a drinking water source that has faecal contamination and, as of 2014, ~700 million people did not have access to an improved water source with over 50% of those people residing in Sub-Saharan Africa (World Health Organization and UNICEF, 2014).

Exposure to contaminated water is an important route for the transmission of diarrhoeal pathogens and is considered to be one of the major causes of diarrhoeal disease deaths occurring annually (Enger et al., 2012; Lopez et al., 2006; Lulani et al., 2008). In 2012, there were approximately 1.5 million diarrhoeal deaths in low- and middle-income countries (LMICs) worldwide. Approximately 842,000 of the deaths were caused by inadequate water,

sanitation, and hygiene (WASH), which represents about 58% of total diarrhoeal deaths and about 1.5% of the total global disease burden (World Health Organization, 2014).

Diarrhoeal disease and waterborne disease outbreaks (WBDOs) from recreational, treated drinking, and ground water are often caused by pathogens such as waterborne viruses, which tend to be more persistent in the environment than bacteria (Gibson, 2014; Silverman et al., 2013; World Health Organization, 2011). While the presence of pathogens in water worldwide is recognised, the majority of microbial data collected is from high income countries as opposed to low income countries providing a potentially important limitation in understanding the extent of the issue (Gibson, 2014). Furthermore, the magnitude of microbial risks to human health from pathogens, especially waterborne viruses, remains largely unknown particularly in developing countries (Katukiza et al., 2013a).

Even with these limitations in developing countries, Quantitative Microbial Risk Assessment (QMRA) can be used to estimate health risks, describe the potential risks from the water supply, and determine water safety management strategies (Howard et al., 2006). QMRA has 4 steps of hazard identification, exposure assessment, dose-response assessment, and risk characterisation and can be deterministic, estimating point risk estimates, or probabilistic, accounting for variability and uncertainty in risk estimates (Haas et al., 2014). Risk estimates are calculated as either Disability Adjusted Life Years (DALYs), which is an overall measure of disease burden, or daily/annual probability of infection or illness values. While QMRA is used extensively in developed countries such as the Netherlands, Australia, and the United States (US) (Bichai and Smeets, 2013; US Environmental Protection Agency, 2014), developing countries, with limited data and resources, have greater challenges when applying QMRA. Thus, the objective of this paper was to (1) describe the extent of viruses in water through the collection of prevalence and quantitative viral concentration data in Sub-Saharan Africa and (2) to describe the use of QMRA in Sub-Saharan Africa as well as to identify data gaps that may limit adoption of this methodology.

METHODS

Two systematic literature reviews were completed to identify all peer-reviewed literature and grey literature available relating to (a) viral prevalence and concentration in water and (b) QMRAs estimating the risk from virally-contaminated water in Sub-Saharan Africa. For both systematic reviews, a two-step process was used to identify papers for inclusion. First, titles and abstracts of papers were initially screened for relevance and then full text articles were reviewed for inclusion.

Both systematic literature reviews were conducted in PubMed and Web of Science to identify relevant papers published from 1990 to June 2017. The database search was supplemented with grey literature found in Google Scholar or after review of included paper bibliographies. Keywords included *water* (waste, irrigation, surface, drinking) and *viruses* and *Africa* and then either *occurrence* (prevalence, detection, quantitative) or *risk* (burden, QMRA). For prevalence and concentration data, 909 papers were identified in the systematic literature search, 736 were screened after removing duplicates, and 81 full-text articles were accessed

for review. In the end, 40 papers were included. Papers were excluded if (a) only clinical data were presented, (b) only summary data was available, (c) indicator data was reported, but not pathogen data, (d) if the paper was purely a methods paper, and (e) the data was collected outside Sub-Saharan Africa. For QMRA data, 961 papers were identified and screened. Of those, 35 were reviewed and 24 were included so 11 were excluded because (a) the QMRA was for indicators and not pathogens, (b) the geography was not Sub-Saharan Africa, or (c) it was an unspecific or comparative risk assessment.

RESULTS AND DISCUSSION

Hazard Identification

Prevalence data and/or concentration data from 40 papers was identified and summarised for a variety of water sources in Sub-Saharan Africa including drinking, ground, irrigation, surface, and waste water (septage and latrines). This data represented 32 independent studies. As observed in Table 1, only 8 of the 48 Sub-Saharan countries (17%) had viral prevalence data. South Africa had the most comprehensive picture of viral contamination (n=21 studies) with numerous viruses and water sources being reported in the literature. Other countries reporting on viral prevalence were Ghana (n=3), Kenya (n=2), Uganda (n=2), and n=1 for Benin, Chad, Nigeria, and Tanzania. The low percentage of viral prevalence data in this region is consistent with another report that found the occurrence of enteric viruses, especially norovirus (NoV), is mostly unknown worldwide (Gibson, 2014).

Viral prevalence data was most abundant for human adenovirus (HAdV) (n=16) followed by rotavirus (RV), NoV, and HAV (all with n=10). The reason these viruses were the most reported on maybe because (a) RV and HAdV species F serotype 40 and 41 are the leading causes of childhood diarrhoea (Mwenda et al., 2010; World Health Organization, 2011) or (b) the second most important viral agent causing gastroenteritis after RV in children in South Africa is NoV (Mans et al., 2010). Other viruses with viral prevalence data included enterovirus (EV) (n=9), sapovirus (SaV) (n=4), human astrovirus (HAstV) and Hepatitis E virus (HEV), and Hepatitis B virus (HBV) and human polyomavirus (HuPyV) (all n=1). Prevalence data varied by water type with viruses found in 1 to 2% of drinking water or in 100 % of surface water or sewage. In general, lowest prevalence values were observed in drinking water while the highest were observed in surface or wastewater.

Insert Table 1

The widespread prevalence of viruses in water highlights a potentially important public health problem (Kiulia et al., 2010). To estimate the extent of this public health burden, QMRA has been used and a total of 24 papers were reviewed describing 22 different QMRAs (Table 2), which assessed the risk of exposure to viral pathogens in a various water sources. Of the 48 Sub-Saharan African countries, only 4 (~8%) including Democratic Republic of the Congo, Ghana, South Africa, and Uganda, had published QMRAs. The QMRAs were done for a variety of viruses with the majority being RV (n=11) followed by NoV (n=8), HAdV and enterovirus (EV) (n=3), and Hepatitis A virus (HAV) and coxsackie B virus (CB-V) all having n=2 QMRAs.

Insert Table 2

Exposure Assessment
The predominant exposure pathways investigated in the QMRAs were drinking and recreation (swimming and immersion) followed by ingestion of raw produce, incidental ingestion while playing by water, incidental ingestion while working, and ingestion during domestic activities (i.e. laundry).

Viral concentration data (Table 1) was found in ~50% of papers representing 4 countries and a variety of water types. The limited viral concentration data in a water samples could be due to the complexity and expense of such analyses (Silverman et al., 2013). Measured viral concentration data varied depending on the type of water with the highest concentration (GC or viruses/L) found in surface water (10^{11}) followed by wastewater effluent (10^{9}), raw wastewater or septage (10^{8}), irrigation water (10^{7}), and drinking water (10^{4}). Measured viral concentration data also varied widely within water type with the widest range (in GC or viruses/L) reported for surface water (10^{-4} to 10^{11}) followed by drinking water (10^{-4} to 10^{4}), wastewater effluent (10^{1} to 10^{9}), raw wastewater or septage (10^{2} to 10^{8}), and irrigation water (10^{2}-10^{7}).

Viral concentration (GC or viruses/L) was most often estimated for HAdV (n=11, range: 10^{-4} to 10^{6}), HAV (n=5, range: 10^{-3}-10^{5}), RV (n=4, range:10^{1}-10^{11}), NoV (n=4, range:10^{1}-10^{8}), EV (n=3, 10^{1}-10^{6}), and SaV (n=2, range: 10^{5}-10^{9}). The highest viral concentration values were reported for RV, SaV, and NoV while the lowest were reported for HAdV and HAV with these both reporting concentration values less than 1 virus/L.

Measured viral concentration data is important because QMRAs rely on this data to estimate dose and risks. However, the majority of the QMRAs (15 of 22, 68%) did not directly quantify the concentration of virus in water. Instead, 11 extrapolated the viral concentration from a surrogate such as *E. coli*, faecal coliforms, total coliforms, or somatic coliphages (Antwi-Agyei et al., 2015; Barker et al., 2014; Fuhrimann et al., 2016; Howard et al., 2006; Hunter et al., 2009; Labite et al., 2010; Lulani et al., 2008; Machdar et al., 2013; Mohammadi, 2014; Seidu et al., 2008) while 4 estimated the viral concentration from presence/absence data obtained from PCR (Grabow et al., 2004; van Heerden et al., 2005c; Venter et al., 2007; Vivier et al., 2004). One QMRA predicted the viral concentration from epidemiological data (Enger et al., 2012). The issue is that indicators are only a proxy of pathogen concentrations in water and when viral concentrations are extrapolated from indicators, these viral concentrations are only an approximation of what is in the water. In fact, a QMRA comparing the risk results from measured quantitative virus concentration data to viral concentration data extrapolated from indicators found that the World Health Organization (WHO) DALY threshold was not met for measured data, but was met when extrapolating from indicator data (Owusu-Ansah et al., 2017). This conclusion highlights how more conservative risk results and management decisions might be made when data is measured versus extrapolated from indicator data.

Thus, measured viral concentration data is ideal and has the advantage of providing site-specific information. Viral concentration was directly measured in 6 QMRAs (Chigor et al., 2014; Genthe et al., 2013; Genthe and Rodda, 1999; Katukiza et al., 2013a; Le Roux et al., 2012; Rodda et al., 1993; Tsai, 2014; Van Abel et al., 2017a) although two QMRAs presented unusable data either in a graph or with contrasting units for the same concentration values (Genthe et al., 2013; Le Roux et al., 2012). Lack of viral concentration data was indicated by others as a major source of uncertainty and it was highlighted that more data, especially in source waters, is needed to refine QMRA estimates of disease burden (Barker et al., 2014; Enger et al., 2012). Ultimately, more measured viral concentration data is needed from this region for use in QMRA.

Published QMRAs do not correspond to the available measured concentration data. For example, 11 RV QMRAs (Table 2) were identified with only 2 using measured RV concentration data (Chigor et al., 2014; Katukiza et al., 2013a). Lack of measured concentration data is supported by Table 1 where RV concentration data was only available from 4 of 40 papers (10%). Thus, most of the RV concentration data was extrapolated from a surrogate. The reason for numerous RV QMRAs even without measured concentration data could be because RV has been identified by the WHO as a potential viral reference pathogen due to the well-defined dose-response model, occurrence in developing countries, low infectious dose, and severe disease burden (Chigor et al., 2014; Haas et al., 2014; World Health Organization, 2011). Moreover, RV is the most important cause of gastrointestinal infection in children in the developing world with almost half of worldwide RV-induced deaths occurring in Africa (Chigor et al., 2014; Katukiza et al., 2013a; Mwenda et al., 2010).

Recovery efficiency was often not reported or used because much of the viral concentration data was extrapolated from a surrogate. Three QMRAs used published recovery efficiencies for NoV (Barker et al., 2014; Mohammadi, 2014; Owusu-Ansah et al., 2017). Another QMRA assumed both high and low recovery efficiencies based on published data (Van Abel et al., 2017a). Four QMRAs estimated the viral concentration from presence-absence data and assumed a recovery efficiency of 40% for low turbidity water and 30% for high turbidity, which were determined from published and unpublished data (Grabow et al., 2004; van Heerden et al., 2005c; Venter et al., 2007; Vivier et al., 2002). Another QMRA assumed a published recovery efficiency of 56 ± 32% for the adsorption-elution method (Chigor et al., 2014). Two QMRAs that directly measured the concentration did not assume a recovery efficiency (Genthe et al., 2013; Katukiza et al., 2013a; Le Roux et al., 2012). Recovery efficiency should be reported alongside reported concentration data.

The degree of infectivity in the QMRAs had to be estimated or assumed because detection and quantification of viral concentration in water (in genome copies, GC) was predominantly done by PCR (polymerase chain reaction) or qPCR (quantitative PCR), respectively. A limitation of this molecular-based approach is that it only estimates the presence of pathogens and cannot distinguish between infectious and non-infectious viruses. Thus, the infectivity of the estimated concentration is unclear and when used in QMRA creates uncertainty in the

health risk estimates (Bambic et al., 2011; Girones et al., 2010; Topping et al., 2009; Van Abel et al., 2017c; World Health Organization, 2011). The degree of infectivity was described in 9 QMRAs. The majority made no assumptions (n=5) about virus infectivity assuming 100% of the estimated concentration of viruses was viable and infectious (Katukiza et al., 2013a; Van Abel et al., 2017a; van Heerden et al., 2005c; Venter et al., 2007; Vivier et al., 2002). One QMRA assumed the infectivity was constant, i.e. no reduction viral concentration in the estimation of dose, because both the exposure assessment (concentration) and dose-response used PCR methods (Tsai, 2014). One QMRA assumed 75% of the viruses were infectious so as to not overestimate the health risk (Grabow et al., 2004) and another assumed 50% (Owusu-Ansah et al., 2017). One QMRA assumed a ratio of infectious to non-infectious particles for each virus (HAdV, HAV, RV, and EV) collected from other published literature (Chigor et al., 2014). Making accurate assumptions about the fraction of infectious particles is important because the exposure outcome is dependent on how many viral particles ingested have the capability of causing infection. Assuming 100% are infectious is a conservative assumption that will not under estimate the risk of infection (Van Abel et al., 2017b). Assumptions about viral infectivity should be stated clearly and be virus-specific. In addition, when possible, comparisons between cell culture and PCR methods should be completed to understand the relationship between genome copies and infectious units.

The volume of water consumed varied in the QMRAs. For drinking water, the assumed intake volume ranged from 100 mL/day to 2.9 L/day with some data coming from developing countries. One QMRA indicated a range of 500-800 mL per day was a reasonable assumption for a slum area (Katukiza et al., 2013a) while the maximum drinking water value (2.9 L per day) was estimated from a community survey in Bangladesh. It should be noted that the Bangladesh data could be skewed high because it represents an extreme exposure scenario because the data was collected during very high ambient temperatures when there was little rainfall (Watanabe et al., 2004). Another QMRA assumed 100 mL of untreated water consumed per day based on observation in South Africa, but this data was never published (Chigor et al., 2014; Le Roux et al., 2012). One QMRA assumed a daily drinking water consumption volume of 1 L per day to not overestimate the risk (Grabow et al., 2004). Overall, there was limited country-specific data available for consumption of drinking water, which is preferable because consumption values vary widely worldwide (Mons et al., 2007).

For recreational water, the assumed volume ingested from incidental exposure ranged from 10 mL to 100 mL per day or event with the majority of QMRAs assuming 30 mL per day. These values are similar to recommended values for swimming in the United States ranging from 15 to 50 mL/day (Dufour et al., 2006; US Environmental Protection Agency, 2014). Incidental exposure from children playing next to surface water, adults working, or domestic exposures were assumed to range from 1 to 10 mL per day based on African data. In Accra, Ghana, field surveys estimated approximately 1 mL was incidentally ingested by children by water and 5 mL by workers (Labite et al., 2010). In South Africa, 10 mL was assumed as a best estimate of incidental ingestion during laundry or work (Genthe and Rodda, 1999; Steyn et al., 2004). In Cote d'Ivoire, 10 mL was assumed as a best estimate for washing plastic bags based on incidental ingestion from irrigation or laundry (Yapo et al., 2014). The amount of

produce consumed was assumed to range from 10 to 51 g per meal with the majority assuming 10 to 20 g per meal. The data on 10 to 20 g was collected from publications on salad consumption specific for Ghana (Fung et al., 2011; Obuobie et al., 2006) while the 10 to 51 g consumption was from a consumer survey done in Ghana (Antwi-Agyei et al., 2016). In many cases, best estimates were inferred in lieu of site-specific information. Again, country- or region-specific consumption data is preferable when available.

In conclusion, some QMRAs called for additional exposure assessment data including more pathogen concentration data (Barker et al., 2014; Enger et al., 2012), better recovery efficiency data (van Heerden et al., 2005c; Vivier et al., 2002), and better water consumption for this region (Genthe and Rodda, 1999; Rodda et al., 1993; van Heerden et al., 2005c; Vivier et al., 2002). Ultimately, whenever possible, site- or region-specific information or data from other developing countries should be used in QMRAs. Also, viral concentration data should be directly measured not extrapolated from indicators and coupled with recovery efficiencies. Finally, the assumed infectivity should be explicitly stated.

Dose-Response

The selection of a dose-response model is important and dose-response assessment is a key ingredient of QMRA as it links exposure of a hazardous agent to the health effect (Teunis and Havelaar, 2000). Overall, the majority of the QMRAs (Table 2) selected previously published, commonly used, and appropriate dose-response models for use. However, some QMRAs arbitrarily selected parameter values for the dose-response models even though published parameterisations were available. As a rule, QMRAs should use published and peer-reviewed models and document the associated dose-response parameters as well as any associated assumptions.

For EV QMRAs (n=3), one QMRA assessed the risk from echo 12, polio 1, and polio 3 using previously published dose-response models (Rose and Gerba, 1991). Another QMRA assumed a polio I dose-response model with $\alpha=0.097$ and $\beta=13020$; however, this parameterisation could not be verified (Genthe et al., 2013). Another QMRA assumed the EV was Coxsackievirus and used the exponential model with r=0.0145, which is a published parameterisation for both B4 and A21 strains (Haas et al., 2014; Haas and Eisenberg, 2001; McBride et al., 2002). For the CB-V QMRAs (n=2), both selected the exponential dose-response model ($r=7.75*10^{-3}$) fit to B4 strain data (Mena et al., 2003) and, as these QMRAs were specific to CB-V, the dose-response model selected was appropriate.

For RV (n=11), 9 of the QMRAs assumed the commonly used approximate beta-Poisson dose-response model ($\alpha=0.2531$, $\beta=0.4265$, $N_{50}=\sim6$) fit to human challenge data (Haas et al., 2014). However, one QMRA assumed the outdated exponential dose-response model (Howard et al., 2006) and another used the alternative ${}_1F_1$ model and parameterisation (Barker et al., 2014; Teunis and Havelaar, 2000). For HAdV (n=3), the QMRAs all assumed the exponential dose-response model (r =0.4172), which describes the inhalation pathway (Crabtree et al., 1997). A limitation of HAdV is the lack of an ingestion route of exposure instead relying on an inhalation exposure dose-response model. However, in the absence of

another dose-response model this model must be used (Lim et al., 2015; Teunis et al., 1999; US Environmental Protection Agency, 2010). For HAV (n=2), one QMRA assumed an exponential dose-response model (r=0.549), which is the parameterisation often recommended (Haas et al., 2014; Haas and Eisenberg, 2001; McBride et al., 2002). The other HAV QMRA acknowledged the exponential parameterisation, but assumed a different parameterisation of the approximate beta-Poisson (Chigor et al., 2014).

NoV dose-response is very complicated and for the 6 NoV QMRAs 6 dose-response models were used (approximate beta-Poisson, ${}_1F_1$ hypergeometric, ${}_2F_1$ hypergeometric, beta-binomial, ${}_2F_1$ hypergeometric with immunity, and fractional Poisson) (McBride et al., 2013; Messner et al., 2014; Schmidt, 2015; Teunis et al., 2008; Van Abel et al., 2017b). One QMRA used 4 NoV dose-response models (${}_1F_1$, ${}_2F_1$ with immunity, ${}_2F_1$, and fractional Poisson) to describe the uncertainty and variability associated with dose-response (Van Abel et al., 2017a). Two other QMRAs used the ${}_2F_1$ hypergeometric (Barker et al., 2014; Owusu-Ansah et al., 2017) while one used the ${}_1F_1$ hypergeometric (Fuhrimann et al., 2016). Two QMRAs assumed the approximate beta-Poisson with one assuming (α =0.022, β=50), which is a parameterisation from the exponential model combined with a randomly assumed β (Genthe et al., 2013; Le Roux et al., 2012). The other assumed parameters that could not be validated in the original source (Mohammadi, 2014). One QMRA used the beta-binomial because the QMRA generated discrete doses (Tsai, 2014). Two QMRAs cited the commonly used hypergeometric models; however, one did not specify which hypergeometric dose-response model (${}_1F_1$ or ${}_2F_1$) was used (Antwi-Agyei et al., 2015) and the other did not report the parameters for the ${}_2F_1$ (Owusu-Ansah et al., 2017). Overall, there are many unsettled questions about NoV dose-response and use of various NoV dose-response models has been recommended previously because no one model has been identified as best yet (Van Abel et al., 2017b, 2017c).

Risk Characterisation

Uncertainty and variability was described in about half of the QMRAs that were probabilistic, while 10 were deterministic, and one was both deterministic and probabilistic. Probabilistic QMRAs require software, such as a MS Excel Add on such as @Risk (Palisade Corporation, Ithaca, NY) or R software (The R Foundation for Statistical Computing; Vienna, Austria), which can perform Monte Carlo simulations (or repeated sampling) to estimate variability and uncertainty. Many QMRAs (n=10) did not report what software was used. When reported, the most common software was any add-on for MS Excel (n=6), probably because of the ease of use, followed by R software (n=4). One QMRA used 4 different software platforms, including @Risk and R, as a check of model values and to assess consistency (Owusu-Ansah et al., 2017). It should be noted that a limitation for the adoption of probabilistic QMRA in the developing world was identified as the need for costly proprietary software (Howard et al., 2006). However, R software is freely downloadable although the learning curve is steeper than the more costly Excel Add-ons. Overall, all QMRAs should report which software was used for reproducibility.

The majority of the QMRAs (Table 2) concluded that the risk of infection, risk of illness, or Disability Adjusted Life Year (DALY) was exceptionally high and in excess of tolerable or acceptable risks for the various water sources. Seven QMRAs estimated DALYs (Figure 1) from the various exposure pathways. When comparing them to the WHO DALY threshold of 10^{-6} loss per person per year (pppy) or the less stringent 10^{-4} pppy (World Health Organization, 2011), then 86% and 60% exceeded, respectively. Fifteen QMRAs estimated daily or annual probabilities of infection and the data is plotted in Figure 2. All drinking water exposures exceeded the daily allowable risk of $<10^{-6}$ infections (Signor and Ashbolt, 2009) and all recreational exposures exceeded the annual illness benchmark of <3 illnesses per 100 events assuming that each infection led to illness (EU Directive, 2006; Soller et al., 2010). It should be noted that the probability of infection and illness benchmarks come from developed countries and may not be useful in a developing country context. Overall, a significant public health concern from exposure to viruses in a variety of water sources can be concluded with the majority of the risk estimates for the exposure pathways exceeding accepted benchmarks.

Insert Figure 1

Insert Figure 2

Risk characterisation is also the place to discuss vulnerable populations. A few QMRAs, highlighted how HIV/AIDS status could impact conclusions about the public health burden because a decreased immune system could result in a lower infectious dose leading to infection or illness in the population (Le Roux et al., 2012; Venter et al., 2007; Vivier et al., 2002). In Sub-Saharan Africa, HIV prevalence was estimated to be around 5% in 2012, but varied by country. For QMRA countries, lower prevalence was estimated in Ghana (1.5%) and the Democratic Republic of Congo (DRC) (1.1%); however, higher burdens were reported in Uganda (7.2%) and South Africa, which has a high estimated HIV prevalence at 18% (UNAIDS, 2013). Thus, this susceptible population must be considered in QMRA particularly for countries with extremely high HIV prevalence values.

CONCLUSIONS

Overall, the risk from viruses in contaminated water was identified as extremely high for almost all QMRA scenarios summarized in this review indicating the potential magnitude of the public health burden in Sub-Saharan Africa. In general, QMRA results can be used to develop local guidelines to protect public health, which may be warranted (Chigor et al., 2014; Seidu et al., 2008). In the very least, the QMRA results can be used as a call to begin more investigation into the pervasive water pollution problem in Sub-Saharan Africa including identifying appropriate interventions. While QMRA is useful for management of water supplies and for estimating potential adverse human health risks from exposure to contaminated water, as observed this paper QMRA is still not a well-developed or commonly used methodology in Sub-Saharan Africa. The advantage of QMRA is the ability to describe the public health burden as well as identifying water sources are polluted. Ultimately, this

review summarized the current use of QMRA in Sub-Saharan Africa and identified future steps that can expand the use of this methodology to this region of the world.

- There is a lack of data on the concentration of pathogens in water. Thus, more data must be collected on the presence of viruses in water as well as the concentration in the water. Also, infectivity of viruses measured by molecular methods must be addressed.
- There is a lack of data on water consumption volumes in Sub-Saharan Africa. Data should be collected on water consumption patterns for this region to improve QMRAs.
- In general, site- or region-specific information or data from developing countries should be used in QMRAs whenever possible.
- Dose-response models selected for use should be previously published and commonly used. When unpublished dose-response models are used to estimate the risk, an under- or over-estimate of the actual risk from water could occur. All models, parameters, and assumptions should also be clearly stated.
- Susceptible populations, those with HIV/AIDS, must be accounted for in countries with significant prevalence.
- Costly software does not need to be an impediment to adoption of QMRA in the developing world because freely downloadable software, such as R software, is available. Additionally, training and education in how to use this software should be provided.

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Table 1: Summary of Prevalence Data on Viruses in Water in Sub-Saharan

Country	Date	Water Category	Water Type	Virus	Method	Sample Volume	Prevalence Point Estimate or Range (%)	Concentration Min-Max (mean+/-SD)	Units	Reference
Benin	2003-2007	DW/GW	pump, well	HAdV	real-time PCR	10 L	2/247 (.8%) - 32/247 (13%)			(Verheyen et al., 2009)
		DW/GW	pump, well	RV	real-time RT-PCR		6/247 (2.4%)			
		DW/SW		HAdV	real-time PCR		3/247 (1.2%)			
Chad	Sept 2009	DW (SW & GW)	river, well, borehole, plant	HAV	nested RT-PCR	10 L	0			(Guerrero-Latorre et al., 2011)
				HAdV	real-time qPCR -> nested PCR		1/7 (14%) - 3/9 (33%)	$8.00*10^1$ - $4.10*10^2$ ($6.82*10^1±1.94*10^2$)	GC/L	
				HEV	semi- or nested RT-PCR		0			
Ghana	July 2010	IW	river & drain	HAdV	real-time qPCR	15-150 mL	11/20 (55%)	$(2.80±0.92)*10^2$ - $(6.50±0.60)*10^4$	GC/L	(Silverman et al., 2013)
				NoV GII	real-time RT-qPCR		16/20 (80%)	$(4.75±2.20)*10^2$ - $(1.58±0.28)*10^4$	GC/L	
Ghana	Jul 28 - Aug 2009	DW	treated	HuPyV	real-time PCR	100 L	1/6 (17%)			(Gibson et al., 2011)
		DW	treated	NoV GII	real-time RT-PCR		1/6 (17%)			
		GW		NoV GI			1/4 (25%)			
		SW		HAdV	real-time PCR		2/9 (22%)			
		SW		NoV GII	real-time RT-PCR		1/9 (11%)			
Ghana	NR	DW	stored; sachet	NoV GI	real-time RT-PCR + IC -> RT-qPCR	160 mL	0/114 (0%)		/100mL	(Tsai, 2014)
				NoV GII			0/122 (0%)		/100mL	
		IW		NoV GI		500 mL-20L	10/82 (12%)	$1.20*10^4$ - $3.10*10^5$ ($1.20*10^5±1.10*10^5$)	/100mL	
				NoV GII			13/82 (16%)	$2.40*10^3$ - $3.10*10^6$ ($2.90*10^5±8.30*10^5$)	/100mL	
		SW	flood/drain	NoV GI		500 mL	4/87 (4.6%)	$6.50*10^3$ - $1.10*10^5$ ($5.30*10^4±4.40*10^4$)	/100mL	

				NoV GII			0/58 (0%)		/100mL	
		septage	latrine	NoV GI		NR	15/40 (38%)	$7.80*10^4$ - $2.20*10^7$ ($3.80*10^6$±$6.70*10^6$)	/100mL	
				NoV GII			16/35 (46%)	$5.80*10^4$ - $8.20*10^6$ ($1.40*10^6$± $2.10*10^6$)	/100mL	
Kenya	Feb 2012 - Jan 2013	DW/GW	borehole & stored	NoV GI	real-time RT-PCR	10 L	0/4 (0%)			(Kiulia et al., 2014)
				NoV GII			1/4 (25%)			
		SW	river	NoV GI			0/12 (0%) - 1/12 (8.3%)			
		SW	river	NoV GII			3/12 (25%) - 9/12 (75%)			
Kenya	May 2007-Feb 2008	SW	river	EV	real-time RT-PCR	10 L	0/7 (0%) - 10/10 (100%)			(Kiulia et al., 2010)
				HAV	real-time RT-PCR		0/7 (0%) - 8/10 (80%)			
				HAdV	Conventional nested PCR		0/7 (0%) - 9/10 (90%)			
				HAstV	real-time RT-PCR		1/7 (14%) - 6/10 (60%)			
				RV	Conventional nested PCR		0/7 (0%) - 10/10 (100%)			
				NoV GI	real-time RT-PCR		0/7 (0%) - 9/10 (90%)			
				NoV GII	real-time RT-PCR		0/7 (0%) - 9/10 (90%)			
				SaV	real-time RT-PCR		0/12 (0%) - 9/10 (90%)			
		sewage	raw	EV	real-time RT-PCR		4/8 (50%) - 5/5 (100%)			
				HAV	real-time RT-PCR		0/5 (0%) - 1/8 (13%)			
				HAdV	Conventional nested PCR		7/8 (88%) - 5/5 (100%)			
				HAstV	real-time RT-PCR		0/5 (0%) - 7/8 (88%)			

				RV	Conventional nested PCR		1/5 (20%) - 8/8 (100%)			
				NoV GI	real-time RT-PCR		0/8 (0%) - 2/5 (40%)			
				NoV GII	real-time RT-PCR		2/5 (40%) - 4/8 (50%)			
				SaV	real-time RT-PCR		1/8 (13%) - 3/5 (60%)			
Nigeria	July - Sept 2010	SW	sewage-contam	EV	integrated cell culture-real-time RT-PCR	480 mL	5/15 (33%)			(Adeniji and Faleye, 2014)
		sewage	raw	EV			4/11 (36%)			
South Africa	Apr 2015-Mar 2016	WW	raw	NoV GI	real-time qRT-PCR	1 L	1/12 (8.3%) - 1/7 (14%)	$1.02*10^{2}$ - $3.41*10^{6}$	GC/L	(Mabasa et al., 2017)
				NoV GII			1/7 (14%) - 6/12 (50%)	$5.00*10^{3}$ - $1.31*10^{6}$	GC/L	
			effluent	NoV GI		10 L	2/12 (16.7%)	$1.02*10^{2}$ - $3.41*10^{6}$	GC/L	
				NoV GII			1/12 (8.3%) - 3/7 (43%)	$5.00*10^{3}$ - $1.31*10^{6}$	GC/L	
South Africa	Jan 2011 – Dec 2014	SW	river	NoV GI	real-time RT-PCR	10 L	All quantified samples were positive	$9.00*10^{1}$ – $1.90*10^{3}$	GC/L	(Van Abel et al., 2017a)
				NoV GII				$4.20*10^{2}$ – $9.76*10^{3}$	GC/L	
South Africa	Sept 2012-Aug 2013	WW	final effluent	HAV	real-time RT-qPCR	1 L	3/48 (6.3%)	<1	GC/L	(Adefisoye et al., 2016)
				HAdV	real-time qPCR		30/48 (63%)	$8.40*10^{1}$ - $1.30*10^{5}$	GC/L	
				RV	real-time RT-qPCR		0/48 (0%)			
South Africa	Sept 2012-Aug 2013	WW	final effluent	HAV	real-time RT-qPCR	1.25 L	5/12 (42%)	<=1	genomes/L	(Osuolale and Okoh, 2015)
				HAdV	real-time qPCR		5/12 (42%) - 11/12 (92%)	$1.00*10^{1}$ - $2.37*10^{5}$	genomes/L	
				RV	real-time RT-qPCR		1/11 (9.1%) - 5/12 (42%)	$1.60*10^{1}$ - $1.24*10^{5}$	GC/L	(Osuolale and Okoh, 2017)
				EV	real-time RT-qPCR		0%			
South	Jan and	SW	river, dam,	HAV	real-time RT-	10 L	7/8 (88%)			(Murray and

Africa	Mar 2012		discharge	NoV GI & GII	PCR		7/8 (88%)			Taylor, 2015)
				SaV	real-time RT-qPCR		8/10 (80%)	$1.11*10^{5}$ - $1.62*10^{7}$ (median = $2.54*10^{6}$)	copies/L	
South Africa	Jan 2012-Aug 2012	IW	river, dam	HAV	real-time RT-PCR	10 L	16/21 (76%)			(Said et al., 2014)
South Africa	Aug 2010-Dec 2011	WW	outflow	HAV		75-100 mL	19/51 (37%)			(Murray et al., 2013a, 2013c)
				NoV GI	real-time RT-PCR		15/51 (29%)			
				NoV GII	real-time RT-PCR		32/51 (63%)			
				NoV GIV	modified 2-step real-time RT-PCR		0/51 (0%)			
				SaV	real-time RT-qPCR		37/51 (73%)	$4.24*10^{3}$ - $1.31*10^{6}$ (monthly avg=$4.24*10^{3}$)	copies/mL	
South Africa	Aug 2010-Jul 2011	SW	river	HAdV	real-time qPCR	1 L	22/72 (31%)	$1.00*10^{0}$ - $8.49*10^{4}$	GC/L	(Sibanda and Okoh, 2012)
South Africa	Aug 2010-Jul 2011	SW	river, dam	HAdV	real-time qPCR	1 L	25/72 (35%)	$1.20*10^{1}$ - $4.71*10^{3}$	GC/L	(Chigor and Okoh, 2012a)
				HAV	real-time RT-qPCR		31/72 (43%)	$1.50*10^{1}$ - $1.90*10^{5}$ ($2.50*10^{4}$)	GC/L	(Chigor and Okoh, 2012b)
				RV			10/72 (14%)	$2.50*10^{1}$ - $2.10*10^{3}$ ($6.20*10^{2}$)	GC/L	
				EV			7/72 (9.7%)	$1.30*10^{1}$ - $8.60*10^{1}$ ($4.00*10^{1}$)	GC/L	
South Africa	Apr, Jul, Oct2011; Jan 2012	SW	river	HAdV	nested PCR & integrated cell culture nested PCR -> real-time qPCR	20 L	17/20 (85%)	$1.46*10^{4}$ - $8.95*10^{6}$	copies/L	(Lin and Singh, 2015; Singh, 2012)
				EV	nested RT-PCR & integrated		20/20 (100%)	$1.10*10^{3}$ - $2.00*10^{6}$	copies/L	
				RV			20/20 (100%)	$2.54*10^{6}$ - $3.72*10^{11}$	copies/L	

					cell culture nested RT-PCR -> real-time RT-qPCR					
				HBV	Conventional nested PCR ->real-time qPCR		np (100%)			
South Africa	Jan 2009-Dec 2010	SW	river	SaV	real-time RT-PCR	10 L	3/17 (18%) - 16/18 (89%)			(Murray et al., 2013b)
South Africa	Jan 2008-Dec 2010	SW	river	NoV GI NoV GII	real-time RT-PCR	10 L	0/8 (0%) - 13/38 (34%) 0/12 (0%) - 18/42 (43%)			(Mans et al., 2013)
South Africa	Jan 2003-Feb 2005	DW	final treated	RV	conventional RT-PCR-> nested PCR	10 L	7/416 (1.7%)			(van Zyl et al., 2006)
		DW	partially treated	RV			2/17 (12%)			
		GW	borehole; empound	RV			0/163 (0%)			
		IW	river, borehole	RV			9/102 (8.8%)			
South Africa	June 2002-July 2003	DW	treated	HAdV	conventional nested PCR->real-time qPCR	200 L	10/188 (5.3%)	<1	copy/L	(van Heerden et al., 2005b)
		SW	river	HAdV		25 L	10/45 (22%)	<1	copy/L	
South Africa	Jan 2002 - Mar 2003	pool		HAdV	conventional nested PCR	1 L	3/28 (11%) - 8/38 (21%)	$1.23*10^{-1}$ - $2.36*10^{-1}$	viruses/L	(van Heerden et al., 2005a)
South Africa	Jul 2000 - Jun 2002	DW	treated	RV	conventional RT-PCR-> nested PCR	10 L	2/41 (4.9%) - 5/77 (6.5%)			(van Zyl et al., 2004)
		DW/GW	borehole	RV			0/15 (0%)			
		SW & partially treated	dam	RV			1/13 (7.7%) - 4/26 (15%)			
		sewage		RV		100 mL	1/9 (11%) - 3/27 (11%)			

		DW	treated	EV		> 100 L	159/850 (19%)			
		GW	borehole	EV			27/108 (25%)			
South Africa	Jul 2000-Jun 2002	SW	dam & spring	EV	conventional RT-PCR-> nested PCR	10-20 L	53/197 (27%)			(Ehlers et al., 2005)
		SW	river	EV			17/60 (28%)			
		sewage		EV			42/100 (42%)			
		DW	treated	HAdV	integrated cell - culture- conventional nested PCR	100-1000 L	59/198 (30%)			
South Africa	Jul 2001-Jun 2002	SW	dam	HAdV		25 L	8/50 (16%)			(van Heerden et al., 2004)
		SW	river	HAdV		25 L	22/50 (44%)			
South Africa	Jul 2000-Jun 2001	DW	treated	HAdV	conventional nested PCR	100-1000 L	0/204 (0%) - 61/204 (30%)	$1.4*10^{-4}$ - $2.45*10^{-4}$	viruses/L	(van Heerden et al., 2003, 2005c)
		SW	river	HAdV		NR	NR (20-60%)	$5.46*10^{-3}$	viruses/L	
		SW	dam	HAdV		NR	NR (0-60%)	$9.97*10^{-4}$	viruses/L	
South Africa	Apr 1999-Mar 2000	DW	treated	EV	integrated cell-culture conventional RT-PCR->nested PCR	100-1000 L	10/88 (11%) - 14/84 (17%)	$4.67*10^{4}$ - $8.90*10^{4}$	viruses/L	(Vivier et al., 2002, 2004)
		SW	dam	HAV	integrated cell-culture conventional RT-PCR hybridisation assay	± 190 L	23/154 (15%)	($2.13*10^{-3}$)	HAV/L	
South Africa	Jun 1997-Jun 2000	SW	river	HAV		± 25 L	27/154 (18%)	($1.99*10^{-2}$)	HAV/L	(Venter et al., 2007)
			river	HAV	integrated cell-culture conventional RT-PCR hybridisation assay	20 L	18/51 (35%)			
				HAstV			11/51 (22%)			
South Africa	June 1997-May 1998	SW	dam	HAV		200 L	19/51 (37%)			(Taylor et al., 2000)
				HAstV			3/51 (5.9%)			

Tanzania	Mar-May 2010	DW & cooking	stored	EV	real-time RT-PCR	1.63 L	0/216 (0%)			(Mattioli et al., 2014)
				HAdV			2/216 (0.9%)			
				RV			4/216 (1.9%)			
Uganda	Jan 11 - Feb 3 2011	SW		HAV	real-time RT-qPCR	10 L	4/26 (15%)			(Katukiza, 2013; Katukiza et al., 2013b)
				HAdV	real-time qPCR		26/26 (100%)	$2.64*10^{-1}$ - $3.27*10^{1}$	GC/mL	
				HEV	real-time RT-qPCR		0			
				RV			20/26 (77%)	$2.96*10^{-1}$ - $1.87*10^{2}$	GC/mL	
		Grey		HAV	real-time RT-qPCR		3/11 (27%)	$6.87*10^{-1}$ - $7.40*10^{-1}$	GC/mL	
				HAdV	real-time qPCR		5/11 (45%)	$0-2.13*10^{0}$	GC/mL	
				RV	real-time RT-qPCR		4/11 (36%)	$3.32*10^{1}$ - $4.70*10^{1}$	GC/mL	
		GW	spring	HAV	real-time RT-qPCR		0%			
				HAdV	real-time qPCR		1/3 (33%)			
				RV	real-time RT-qPCR		0%			
Uganda	Nov 10, 2014-May 27, 2015	DW	tap	EV	conventional qPCR	2 L	NR (6.0%)			(Sadik, 2016)
		SW	spring	EV			NR (12%)			
		SW	drainage	EV		0.5 L	NR (8.0%)			
		SW	lake	EV			NR (29%)			

NR=not reported, GC=genome copies

DW=drinking water, GW=groundwater, IW=irrigation water, SW=surface water, WW=wastewater

EV=enterovirus, HAdV=human adenovirus, HAstV=human astrovirus, HAV=hepatitis A virus, NoV=norovirus, RV=rotavirus, SaV=sapovirus

Table 2: All QMRAs done in Sub-Saharan Africa

			HAZARD ID		EXPOSURE ASSESSMENT						DR ASSESSMENT			
Ctry[a]	RA type[b]	Soft-ware	Virus[c]	Water type[d]	Exposure pathway	Conc Mean/Pt (min-max)	Conc Units	Recovery Eff (%)[e]	Deg of Inf (%)[f]	Vol, Amt, & # cons	DR model[g]	DR params	Ref	Comments/ Critiques[h]

DRC	prob	Octave 3.2/ MATLAB 7.11	RV	DW (w/ & w/o filter)	drinking	(0-0.18)	virions/L	predicted data		1.178 L/day	app BP	α=0.253, N_{50}=6.17	(Enger et al., 2012)	Conc predicted from epi data; Some variables deterministic (i.e. water ingestion); Published RV DR model params
GHA	prob	@Risk	RV	IW (watering)	incidental	(0 - 0.98)	RV/ 100 mL	extrap[i]	NR[i], but no reduction of virus	1-5 mL[j]; 75 day/yr	app BP	α=0.253, N_{50}=6.17	(Seidu et al., 2008)	Conc extrapolated from surrogate (FC); Assumed worst case scenario so no reduction/inactivation of virus; Published RV DR model params
				IW	ingestion raw produce	(0.03-0.19)	RV/ 100 g wgt			10-12 g[j]; 208 day/yr				
GHA	determ	NR[i]	RV	SW	recreation(s)	NR[i]		extrap		100 mL/swim; 7 #/yr	app BP	α=0.253, N_{50}=6.17	(Lulani et al., 2008)	Conc data not presented, but stated as extrapolated from surrogate (EC); Assumed high vol consumed value for DW (dev country Asia) & swimming (Australia); Published RV DR model params
				SW (flood)	incidental ingestion					1 mL, 1 #/yr				
					Immersion[k]					30 mL,1 #/yr				
					kids playing					1 mL, 1 #/yr				
				SW (open drainage)	kids playing					5 mL, 4 #/yr				
				DW (contam water)	drinking					2.9 L/day				
GHA	determ	NR[i]	RV	DW (trted)	drinking	($4.1*10^{-9}$ - $2.31*10^{-6}$)	viruses/ L	extrap		2.9 L/day; 1-365 #/yr	app BP	α=0.253, N_{50}=6.17	(Labite et al., 2010)	Conc extrapolated from surrogate (EC); Assumed high vol consumed value for DW (dev country Asia) & swimming (no ref); Published RV DR model params
				SW (river)	recreation	$1.70*10^{-2}$[m]				75 mL/day; 2 #/yr				
				SW (lagoon)	immersion[k]	$1.20*10^{1}$[m]				30 mL/day; 1 #/yr				
				SW (drainage)	incidental from play	$1.20*10^{3}$[m]				5 mL/day; 2-4 #/yr				
				SW (flood)	incidental	$1.20*10^{1}$[m]				1 mL/day; 1#/yr				
GHA	determ	NR[i]	RV	DW (adults)	drinking	($1.84*10^{-1}$- $1.04*10^{1}$)	viruses/ L	extrap		2.9 L/day	app BP	α=0.253, N_{50}=6.17	(Machdar et al., 2013)	Conc extrapolated from surrogate (EC); Assumed high vol consumed value for DW (adults: USA male guidance
				DW (children)		$3.04*10^{1}$[n]				1 L/day				

				DW (sachets, irreg use)		8.00*10^{-2}[n]				0.5 L/day				value for rehydration); Published RV DR model params
GHA	prob	'R' vers 2.12.2	RV NoV	IW (WW)	ingestion of raw produce	(3*10^{-3}-2*10^{1}) (9*10^{-1}-9*10^{2})	#/g #/g	NA[o] 11.1-50	NR[i]	20 g/meal; 1-7 days/wk	1F1 2F1	α=0.167, β=0.191 α=0.04, β=0.055, a=0.9997	(Barker et al., 2014)	RV conc extrapolated from surrogate (FC or TC); NV conc extrapolated from surrogate (EC or FC); Published RV DR model params; Assumed NoV DR model with aggregation
GHA	prob	@Risk 6.2	NoV	DW	drinking	EC:NV (1:2.2*10^{-4} to 1.7*10^{-1})[p]	#/mL	11.1-50	NR	1.2-2.9 L	app BP[x]	α=0.1109, N_{50}=16963	(Mohammadi, 2014)	NoV conc extrapolated from surrogate (EC or FC) with corresponding recovery data like Barker et al 2014; Units not reported for vol consumed; Assumed alt NV DR model and params
GHA	prob	R software	NoV GI NoV GII	IW (farm water)	incidental	(1.2*10^{4}-3.1*10^{5}) (2.4*10^{3}-3.1*10^{6})	gc/100 mL	NR[i]	Constant; DR & exposure used PCR	1.0-5.0 mL/day; 7 days	Beta-Binomial	α = 0.04, β = 0.055	(Tsai, 2014)	NoV GI and GII conc measured; Recovery efficiency not reported; Infectivitiy assumed constant bc PCR used for DR and exposure; Discrete doses so used beta-binomial DR
GHA	prob	NR[i]	NoV	IW (ww)	ingestion (ww on produce) ingestion of raw produce	EC:NV (0.1-1:10^{5})[p] EC:NV (0.1-1:10^{5})[p]	/100 mL /100 g	extrap		NRi 10-51 g lettuce/day; 2-4 days/wk	Not specified (Teunis et al 2008 cited)		(Antwi-Agyei et al., 2015)	NoV conc extrapolated from surrogate (EC); Vol of ww on lettuce not provided; Did not specify DR model or params, but did cite Teunis et al 2008
GHA	prob	R, MatLab, Mathematica, @Risk	NoV	IW (ww)	Ingestion of raw produce	EC:NV (0.1-1:10^{5})[p] 1.21*10^{2}-3.4*10^{4} (gamma distribution)	gc/mL	11.1-50	50	10-20 g/meal; 0.00775-0.108 mL/g	2F1 or 1F1 if large doses	NR (Teunis et al 2008 cited)	(Owusu-Ansah et al., 2017)	Conc extrapolated from surrogate (EC) and pooled from published literature then fit to gamma distribution; Conc data linked to published recovery efficiencies; Assumed 50% infectivity; Also, factored in pathogen decay constant, irrigation cessation period before harvest, and virus

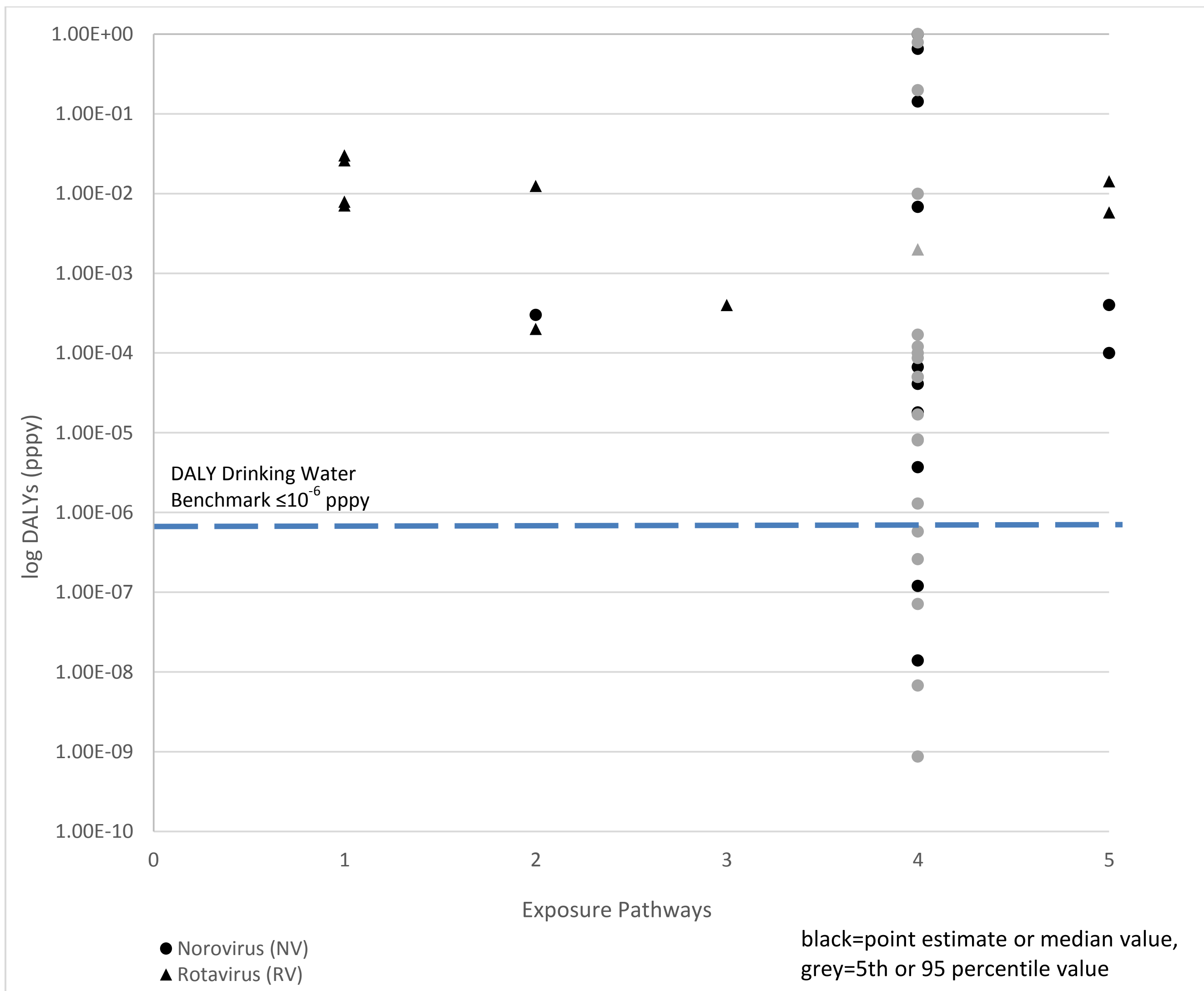
														reduction post-harvest washing; Assumed aggregated form of NoV DR (2F1)
RSA	determ & prob	@Risk	echo 12 polio 1 polio 3	SW (marine)	recreation	(0.315/ND[q]-4.00)	MPN/ $TCID_{50}$ per 100 mL	n/a cell culture method used		100 & 10 mL/day; 7 & 250 #/yr	app BP app BP app BP	α=1.3, β=75 α=15, β=1000 α=0.5, β=1.14	(Genthe and Rodda, 1999; Rodda et al., 1993)	Not all conc data available; Assumed high vol consumed value for rec (Australia rec water guidelines) and low ("more realistic" value); Published echo 12, polio 1& 3 DR model params
			echo 12 polio 1 polio 3	DW (raw & trted)	drinking	NR[i] (graph only)	MPN/ $TCID_{50}$ per 10 L			2 L/day; daily	same as above			
RSA	prob	@Risk	CB-V	DW (trt A) DW (trt B)	drinking	($2.00*10^{-4}$-$1.00*10^{-3}$)[r] ($7.94*10^{-4}$-$9.99*10^{-4}$)[r]	viruses/ L	40	100	1.13 L/day	exp	r= $7.75*10^{-3}$	(Vivier et al., 2002)	Some variables deterministic (i.e. rec eff & infectivity); Conc estimated from +/- data; Pub & unpub data for efficiency of recovery assumption; Test samples (+) in cell culture led to 100% deg of inf assumption; Published CB-V DR params (B4)
RSA	determ	NR[i]	CB-V	DW (trted) DW (raw)	drinking	($4.37*10^{-4}$-$2.62*10^{-2}$) ($2.84*10^{-2}$-$1.28*10^{-2}$)	avg count/L	40 (low turb) 30 (high turb)	75	1 L/day	exp	r= $7.75*10^{-3}$	(Grabow et al., 2004)	Conc estimated from +/- data; Pub & unpub data for efficiency of recovery assumption; Test samples (+) in cell culture led to 75% deg of inf assumption (no overest); Assumed less than average vol consumed for DW (no overest); Published CB-V DR params (B4)
RSA	determ	NR[i]	HAdV	DW (trt A) DW (trt B) SW (river)	drinking recreation	$1.40*10^{-4}$[n] $2.45*10^{-4}$[n] $5.46*10^{-3}$[n]	viruses/ L	40	100	2 L/day 30 ml/day	exp	r=0.4172[s]	(van Heerden et al., 2005c)	Conc estimated from +/- data; Prev pub data for efficiency of recovery assumption; Test samples (+) in cell culture led

				SW (dam)		$9.97*10^{-4}$[n]								to 100% deg of inf assumption; Published HadV DR model is for inhalation pathway data
RSA	determ	NR[i]	HAV	SW (river) SW (dam) SW (river) SW (dam)	recreation drinking	$7.94*10^{-3}$[n] $8.53*10^{-4}$[n] $7.94*10^{-3}$[n] $8.53*10^{-4}$[n]	viruses/ L	40	100	100 mL/day 2 L/day	exp	r=0.549	(Venter et al., 2007)	Conc estimated from +/- data; Unpub data for efficiency of recovery assumption; Test samples (+) in cell culture led to 100% deg of inf assumption; Assumed rec water vol was stated in orig pub as lacking validated data; Published HAV DR model params
RSA	determ	NR[i]	HAdV HAV RV EV	SW (river/ dam)	drinking recreation drinking recreation drinking recreation drinking recreation	(ND[q]-$2.70*10^{3}$)[t] ($2.25*10^{3}$-$9.68*10^{4}$)[t] (ND[q]-$2.79*10^{3}$)[t] (ND[q]-$1.25*10^{2}$)[t]	gc/L	56	50 2 10 1	100 mL/day; 30 ml/day 100 mL/day; 30 ml/day 100 mL/day; 30 ml/day 100 mL/day; 30 ml/day	exp app BP app BP exp	r=0.4172[s] α=0.200, N_{50}=1000[u] α=0.2531, β=0.4265 r=0.0145[v]	(Chigor et al., 2014)	Conc measured; Prev pub data for efficiency of recovery and deg of inf assumptions; Assumed lower vol consumed value for DW based on SA data that was observed but not provided in cited pub; Published HAdV DR model is for inhalation pathway; Assumed alt HAV DR model and params; Published RV DR model; EV DR model is for Coxsackie virus (B4, A21)
RSA	determ	NR[i]	EV NoV	SW (river)	drinking	(ND[q]-2760) (ND[q]-828)	counts/ 100 mL & avg virions /10 L[w]	NR[i]		100 mL & 1.2 L/day[w]	app BP	α=0.097, β=13020[x] α=0.022, N_{50}=50[y]	(Genthe et al., 2013; Le Roux et al., 2012)	Contrasting conc units and water intake volumes reported in 2 citations; Lower vol consumed value in 1 citation for DW based on observed (not provided) SA data; EV DR model is for Polio 1 but cited params not verified; Outdated and unused NoV DR model, also cited α is for exp DR model
RSA	prob	R	NoV	SW (river)	drinking	GI: μ=360;	gc/L	Low:	100	μ=1.3 L/day	1F1,	**1F1**	(Van	Conc was measured, site-

		software	GI & GII		domestic	GII: μ=1780		0.01-3.80; High: 7-60		1-10 mL/day;	2F1i, 2F1, FP	α = 0.04, β = 0.055; **2F1i** α = 2.91, β = 2734, φ=0.2754; **2F1** α = 0.04, β = 0.055, a=0.9997; **FP** P=0.72, μ_a=1106	Abel et al., 2017a)	specific, and fit to lognormal distribution; Published recovery efficiency data for low and high recoveries; Assumed 100% infectivity; Volume and amounts consumed were based on distributions: drinking (lognormal), domestic (uniform), swimming (lognormal & negative binomial), boating (triangle and uniform), playing (triangle & lognormal); 4 NoV DR models were used to account for variability and uncertainty
					recreation (swimming)					μ=55 mL/event; 24 events/yr				
					recreation (boating)					μ=1.9 mL/hr; μ=2.1 hr/day; 1-108 days/yr				
					incidental (playing)					μ=2.1 mL/hr; μ=82 min/day; μ=15 days/yr				
UGA	determ	NR[i]	RV	DW (trted)	drinking	1000[m]	organisms/L	extrap		1 L/day	exp	r= $2.70*10^{-1}$	(Howard et al., 2006)	Concentration extrapolated from surrogate (somatic coliphage); Lower vol consumed value; Prev published DR model now updated
UGA	determ	MS Excel	RV	DW (trted) DW (Raw)	drinking	0.0095[m] 950[m]	pathogens/ L	extrap		1 L/day	app BP	α=0.253, β=0.422	(Hunter et al., 2009)	Concentration extrapolated from surrogate (somatic coliphage); Lower vol consumed value; Published RV DR model params
UGA	prob	XL Sim software 3	RV	GW (drainage) StW (drainage)	incidental ingestion	(0.344 - 8.85) (1.66-$2.98*10^{1}$)	gc/ mL	NR[i]	100	5-10 mL; 6-8 #/yr	app BP	α=0.2531, N_{50}=6.17	(Katukiza et al., 2013a)	Conc measured; Assumed measured conc in QMRA (deg of inf 100%); Lower DW vol consumed value bc was assumed to be reasonable for a slum area; Published RV DR model params; Published HAdV DR model is for inhalation pathway
			HAdV (F&G)	SW (protected spring)	drinking	7.62E-03 (±1E-02)				500 mL; 365 #/yr	exp	r=0.4172		
				GW (drainage) StW (drainage)	incidental ingestion	($1.35*10^{-1}$-$7.80*10^{-1}$) ($3.27*10^{-1}$-$2.65*10^{1}$)				5-10 mL; 6-8 #/yr				

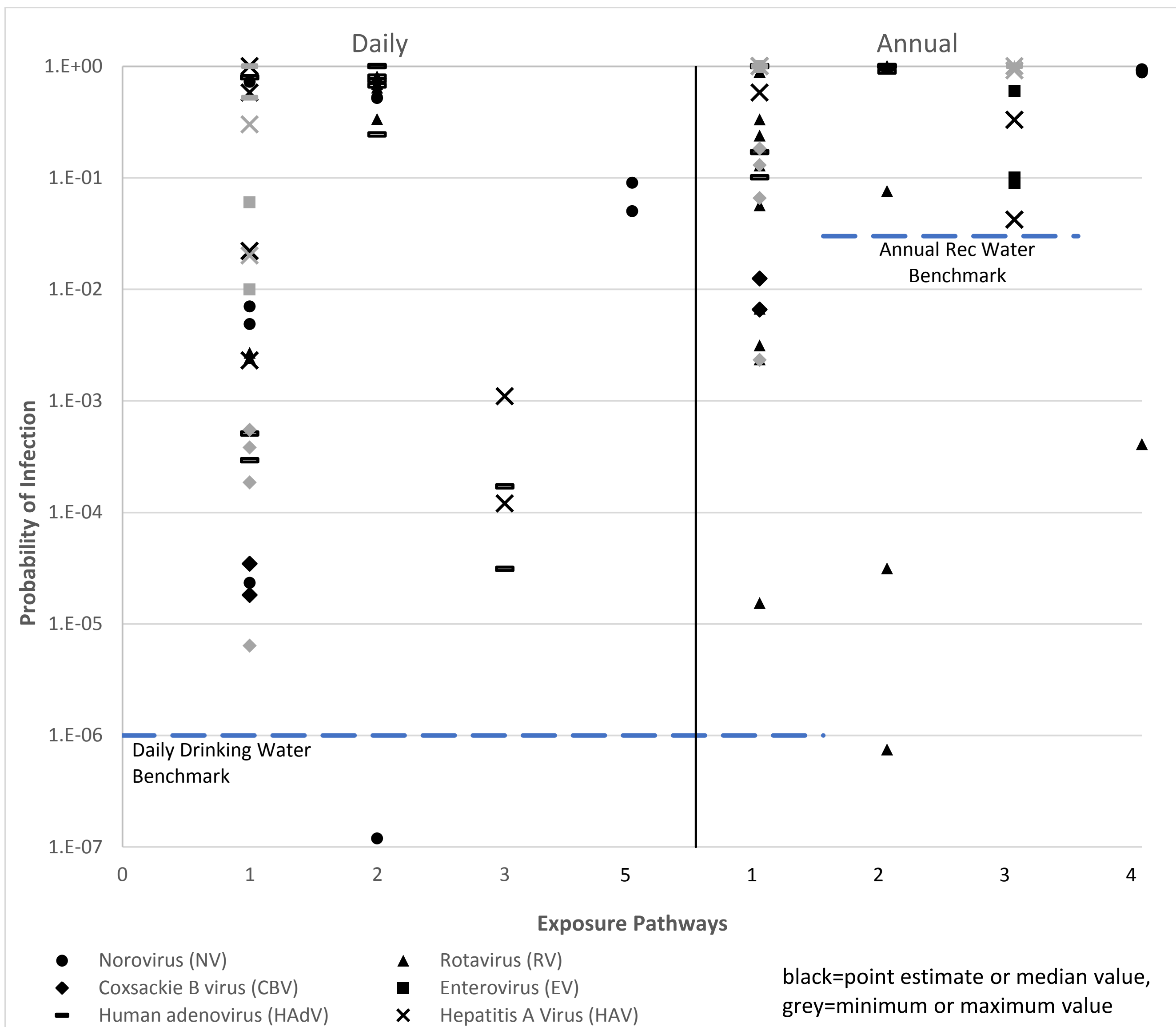
UGA	prob	@Risk version 6	NoV	SW (flood)	immersion[k]	NoV PERT (0.1, 0.55,1) /100 mL per 10^5 EC[p]	extrap	10-30 mL/evt ;6 events/yr	1F1	α = 0.04, β = 0.055	(Fuhrimann et al., 2016)	NoV and RV conc extrapolated from surrogate (EC); Discrepancy in listed # of exposure events (4 vs 6); NV DR described as 1F1 and beta-binomial was assumed for uncertainty; RV DR model params
				WW	incidental (work)			1-5 mL/day; 312 days/yr				
				SW (flood+ww)	incidental (work)			10-50 mL/day ;297 days/yr				
				SW (flood+ww)	incidental (play)			1-5 mL/day; 365 days/yr				
				SW	recreation			20-50 mL/evt; 6 #/yr				
			RV	SW (flood)	immersion[k]	RV PERT (0.1, 0.55,1) /100 mL per 10^5 EC[p]		10-30 mL/evt ;6 events/yr	app BP	α =0.253, N_{50}=6		
				WW	incidental (work)			1-5 mL/day; 312 days/yr				
				SW (flood+ww)	incidental (work)			10-50 mL/day 297 days/yr				
				SW (flood+ww)	incidental (play)			1-5 mL/day; 365 days/yr				
				SW (flood)	recreation			20-50 mL/evt; 6 #/yr				

[a]Country DRC=Democratic Republic of the Congo, GHA=Ghana, RSA=Republic South Africa, UGA=Uganda, [b]Approach used: Probabilistic (prob) or Deterministic (determ), [c]Virus type CB-V=coxsackie B virus, EV=enterovirus, HAdV=human adenovirus, HAV=hepatitis A virus, NoV=norovirus RV=rotavirus,
[d]Water type DW=drinking water, GW=ground water, IW=irrigation water, StW=storm water, SW=surface water, WW=wastewater, [e]Recovery Efficiency from water concentration method
[f]Percentage of estimated viruses that are viable and infectious, [g]DR Model 1F1=$_1F_1$ hypergeometric, 2F1=$_2F_1$ hypergeometric, app BP=approximate beta-Poisson, exp=exponential, 2F1i=$_2F_1$ hypergeometric with immunity, and FP=fractional Poisson, [h]Surrogates EC=*E. coli*, FC=faecal coliforms, TC=total coliforms, [i]extrap=extrapolated from other data or NR = Not reported in article, [j]Uniform distribution assumed, [k]incidental from immersion, [m]Point estimate value, [n]Mean value, [o]NA=Data not available, [p]ratio of surrogate to virus provided and not viral concentration, [q]ND=Not detected, [r]95% Confidence Interval, [s]For inhalation pathway, [t]Corrected values reported in the Table, [u]These values were not obtained from data, were a guess, [v]Data for Coxsackie virus, [w]Disparate units and values were published in the two papers for same QMRA (i.e. same results reported in each paper), [x]Data not in original cited publication, [y]Published α was actually for an exponential DR model, N_{50} is a guess assuming 50% infectious dose for NoV is 10-100 gc



1=ingestion of drinking water, 2=incidental ingestion from playing/recreating by water, 3=incidental ingestion from contact with water (swimming), 4=ingestion of raw produce, 5=incidental ingestion while working

Figure 1: DALYs for Different Exposure Pathways



1=ingestion of drinking water, 2=incidental ingestion from playing/recreating by water, 3=incidental ingestion from contact with water (swimming), 4=ingestion of raw produce. 5=incidental ingestion while working

Figure 2: Daily and Annual Probability of Infection Risk Results for Different Exposure Pathways