Appendix D – Public Health Assessment

Wastewater overflow discharge consent - Queenstown Lakes District Council

Microbial risk assessment

Prepared for Queenstown Lakes District Council

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Final

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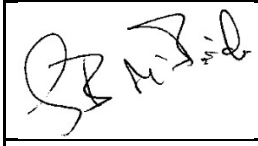	Reviewed by:	Graham McBride
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Contents

Executive summary **5**

1 Background **7**

2 Receiving environment **8**

2.1 Location of discharges 8

3 Assessing human health risks **9**

4 Methodology for conducting a QMRA **10**

4.1 Conventional QMRA modelling 10

4.2 Reverse QMRA approach 12

4.3 Reverse QMRA assumptions 12

5 Estimating public health risk **17**

6 Minimising adverse effects **21**

7 Conclusions **23**

8 References **24**

9 Acknowledgements **32**

10 Glossary of abbreviations and terms **33**

Appendix A Dose-response functions **34**

Appendix B Echovirus 12 clinical trial data analysis **38**

Appendix C Debate about norovirus infection dose-response **39**

Appendix D Recent microbiological water quality data **40**

Tables

Table 4-1: Distributions and inputs for the QMRA. 16

Table 5-1: Individual Illness Results for aggregated and disaggregated norovirus for five concentration orders, assuming typical illness patterns in the local community. 19

Table 5-2: Individual Infection Results for aggregated and disaggregated norovirus for five concentration orders, assuming typical illness patterns in the local community. 20

Figures

Figure 4-1: Process followed to relate human health risk to pathogen-contaminated surface waters. 10

Figure A-1: Conditional and unconditional infection dose-response curves. 35

Figure D-1: Microbial water quality for the Lake Wakatipu at Frankton Bay monitoring site. 40

Figure D-2: Microbial water quality for the Lake Wakatipu at Queenstown Bay monitoring site. 40

Figure D-3: Microbial water quality for the Lake Hayes at Mill Creek Shallows monitoring site. 41

Figure D-4: Microbial water quality for the Lake Wanaka at Roy Bay monitoring site. 41

Figure D-5: Microbial water quality for the Lake Hawea at Holiday Park monitoring site. 42

Executive summary

Sewer leaks, blockages and sewer pump failure or occurrence of sewage flows that exceed sewer pump capacities have the potential to cause occasional discharge of raw sewage into streams and lakes in the Queenstown Lakes district. These occasional, aberrational discharges create conditions where increased risk of illness to recreational water users exposed to contaminated water exist.

Queenstown Lakes District Council provides wastewater reticulation and treatment services across the district, and wishes to better understand the human health risks arising from these accidental exposure events as part of the resource consent process.

Currently much of the data required to undertake a Quantitative Microbial Risk Assessment (QMRA) do not exist. These data are required to estimate the dilution of raw sewage under various river flow and rainfall conditions. Dilution of the raw sewage determines the concentration of pathogens in the receiving environment, which in turn determines the number of pathogens likely to be ingested by recreational users.

Despite this limitation, it is possible to estimate Individual Illness Risk for scenarios determined by:

- a selected "model" pathogen (in this case Norovirus, generally the most widespread pathogen in wastewater from urban areas)
- typical virus concentrations in raw sewage (from 1000 to 10^7 infectious units/L)
- commonly accepted water ingestion rates for recreational water user (swimmers), adjusted for child receptors
- a range of relative sewage dilution rates (from 1x to 100,000x).

Incorporating these assumptions in a model that allows "Monte Carlo"-type random sampling from many recreational events (1000), it is possible to estimate the Individual Illness Risk (IIR) or Individual Infection Risk for a group of 100 recreational users exposed on any random occasion, expressed as a percentage. These results indicate:

- Illness risk is always lower than the infection risk –illness requires infection, whereas infection does not necessarily lead to illness.
- Aggregation of pathogens decreases infectivity and risk of illness considerably, especially at low doses.
- The risk of illness or infection decreases as dilution increases.

These results indicate a potential for significant health risk arising from the discharge of untreated sewage in the conditions assumed in each scenario. Improved estimates of the health risks created by these discharges requires estimates of sewer and sewer pump leakage volumes or flows, stream flows at the time of the overflow events, and use of a mixing model. For lakes, use of a calibrated hydrodynamic model, able to represent the mixing, dilution and advection of contaminants within the lake will be required. A model of this nature would also allow the IIR to be calculated at any location within the lake model domain.

Irrespective of the availability of a hydrodynamic model that may improve estimation of the human health risk potential, occasional discharges should be anticipated and appropriate response plans developed. These very infrequent discharges are likely to occur despite the best efforts and careful management of Council infrastructure. Having in place well-developed and comprehensive response plans will ensure that

when these very infrequent overflow events occur, public health risk is protected and the risk to the community is minimised.

Practical information and strategies that may be incorporated in response plans is provided in the Ministry of the Environment/Ministry of Health “Microbiological Water Quality Guidelines for Marine and Freshwater Recreational Areas" (2003). Among other things, these Guidelines recommend:

- public notification (signage, creation of exclusion zones and use of print and other modern media, such as social media) to encourage people to avoid certain areas and activities
- cross-agency cooperation and sharing of information and roles and responsibilities to maximise public health protection, and
- implementation of event-related monitoring activities, so that the extent of contamination (spatially and in time) is well known, and to ensure that the extent of the response plan is adequate.

The response plan included with the Assessment of Environmental Effects was reviewed as part of this health risk assessment, and we have concluded that it provides a suitable high-level indication of what would be done in response to a pollution event. Several recommendations were provided for consideration and possible inclusion to indicate more clearly how Queenstown Lakes District Council intends to respond to sewer overflows in an adaptive management approach.

If QLDC implements recommended response processes, then we consider that the risk to human health arising from occasional discharge of wastewater from the sewer network to be low to very low.

1 Background

Queenstown Lakes District Council (QLDC) provides wastewater reticulation services to the Queenstown Lakes District. Wastewater networks are subject to unpredictable factors which may lead to occasional accidental discharges of wastewater into the environment.

More specifically, wastewater may be discharged to land in circumstances where it may then enter a water catchment, or directly into a surface water catchment. The areas that may be subject to discharges may be utilised for several recreational purposes and may have broader amenity value to the community. The discharge of wastewater to areas with high amenity values (e.g., streams and rivers, or the lake foreshore) also creates the potential for measurable health risks.

NIWA was engaged by QLDC to undertake a public health risk assessment as part of the assessment of effects accompanying QLDC's wastewater network overflow discharges consent application. This report sets out the results of the Quantitative Microbial Risk Assessment (QMRA) process and makes recommendations for QLDC's management of public health risks. The Assessment of Environmental Effects[1] (referred to hereafter as the AEE) provides a fuller description of the project including the physical extent it covers within the Queenstown Lakes District. This is not repeated in this report.

[1] Prepared by Beca Ltd

2 Receiving environment

2.1 Location of discharges

The location of the current and future QLDC wastewater networks is explained in the AEE, with specific detail about the nature of the receiving waters set out in the ecological report prepared by Ryder Environmental Limited. In essence, discharges may occur to streams and rivers of varying size, as well as to lakes directly, and at a wide range of locations.

For the purpose of this assessment, key points to note include:

- Wastewater will be discharged into rivers and streams of varying sizes – this will determine the extent of immediate dilution available.
- The flow in all of the receiving streams will vary seasonally and in response to discrete rainfall events, which will also determine immediate dilution.
- Assuming the receiving streams are perennial, the duration of contamination at most stream and river discharge sites will probably be short (determined by the size and duration of the discharge and the flow in the stream).
- The contaminants of concern (pathogens) will always move in a downstream direction, and may be discharged to one of the lakes, or into a larger river.
- The duration of contamination in lakes is less easy to determine, and likely to depend on the persistence of contaminant plumes, and factors such as:
  - the temperature and density differences between diluted wastewater and lake waters
  - wind speed and direction, which will strongly influence localised wind mixing and determine the impact of larger-scale wind-driven currents, and
  - currents related to discharge of rivers from the lakes.

3 Assessing human health risks

Human health risks arising from exposure to microbial contaminants during recreational activities are generally assessed using recreational bathing monitoring programmes.

The Ministry for the Environment and Ministry of Health "*Microbiological Water Quality Guidelines for Marine and Freshwater Recreational Areas"* (MfE/MoH 2003) (MfE/MoH Guidelines) provide guidance regarding establishment and operation of recreational water quality monitoring programmes, and when interpreting the results derived from monitoring. Monitoring recreational water quality generally relies on use of faecal indicator bacteria (FIB) – *E. coli* in freshwaters. The MfE/MOH Guidelines are quite clear, however, that they should not be used during "exceptional circumstances". Exceptional circumstances relate to known periods of higher risk, such as during a "sewer rupture" or, in other words, any accidental discharge from the wastewater network. In these circumstances, alternate methods are required to assess human health risks arising from possible exposure to pathogens. These risks may be calculated using QMRA techniques, as explained hereafter.

This report explains the requirements for undertaking a QMRA, including data regarding receiving environment conditions and the choice of pathogens. In this study, several significant assumptions are required - justification of these choices is presented as well.

Risk assessment is applied to a diverse range of activities, including workplace health and safety, the design of structures, the planning and operation of space missions. Despite the diversity of these activities, several common factors need to be considered, and are provided here as definitions to guide the reader:

- **Hazard** - anything (e.g., work materials, equipment, methods, practices or activities) that has the potential to cause harm. In this case, the hazard is a wastewater discharge.
- **Risk** - the chance, high or low, that somebody may be harmed by the hazard. Risk is sometimes defined as **chance + hazard + exposure + consequence**, or "the likelihood of identified hazards causing harm in exposed populations in a specified time frame, including the severity of the consequences".[2] By its nature, risk is probabilistic and estimating risk requires the development of quantitative information.
- **Risk assessment** - the process of evaluating risks to individual safety and health arising from the hazards. It is a systematic examination of all aspects of an activity that considers:
  - what could cause injury or harm
  - whether the hazards could be eliminated, and if not
  - what preventive or protective measures are, or should be, in place to control the risks.

QMRA is a framework and approach that brings information and data together with mathematical models to address the spread of microbial agents through environmental exposures and to characterise the nature of the adverse outcomes. Although most microbes are harmless or beneficial, some are extremely dangerous – these are termed pathogens or Biological Agents of Concern (BAC). Although these have the potential to cause serious or fatal illness, they differ greatly in their physical characteristics, movement in the environment, and process of infection. These characteristics and the differences between potential pathogens are considered in the risk assessment process, to ensure that appropriate "model" pathogens are selected to assess human health risks.

[2] http://qmrawiki.canr.msu.edu/index.php/Quantitative_Microbial_Risk_Assessment

4 Methodology for conducting a QMRA

As indicated above, risk is probabilistic and estimating risk requires the development of quantitative information. We have generally followed two approaches, which we term conventional QMRA and reverse QMRA. The selection and use of one or both is principally determined by available data and information. We discuss both approaches briefly below.

4.1 Conventional QMRA modelling

QMRA consists of five basic steps:

A. Selection of the hazard(s), i.e., the pathogen(s) of concern—exposure to which can give rise to illness.

B. Assessment of exposure to the pathogen(s) at key sites (in terms of pathogen concentrations and duration of exposure).

C. Characterisation of human response to pathogen dose (creating suitable dose-response curves) – described in Appendix A.

D. Calculation of the health risk (in terms of infection and/or illness).

E. Communication of health risk, identifying appropriate response and mitigation actions.

Several components associated with or required for steps A-E are described in the schematic in Figure 4-1.

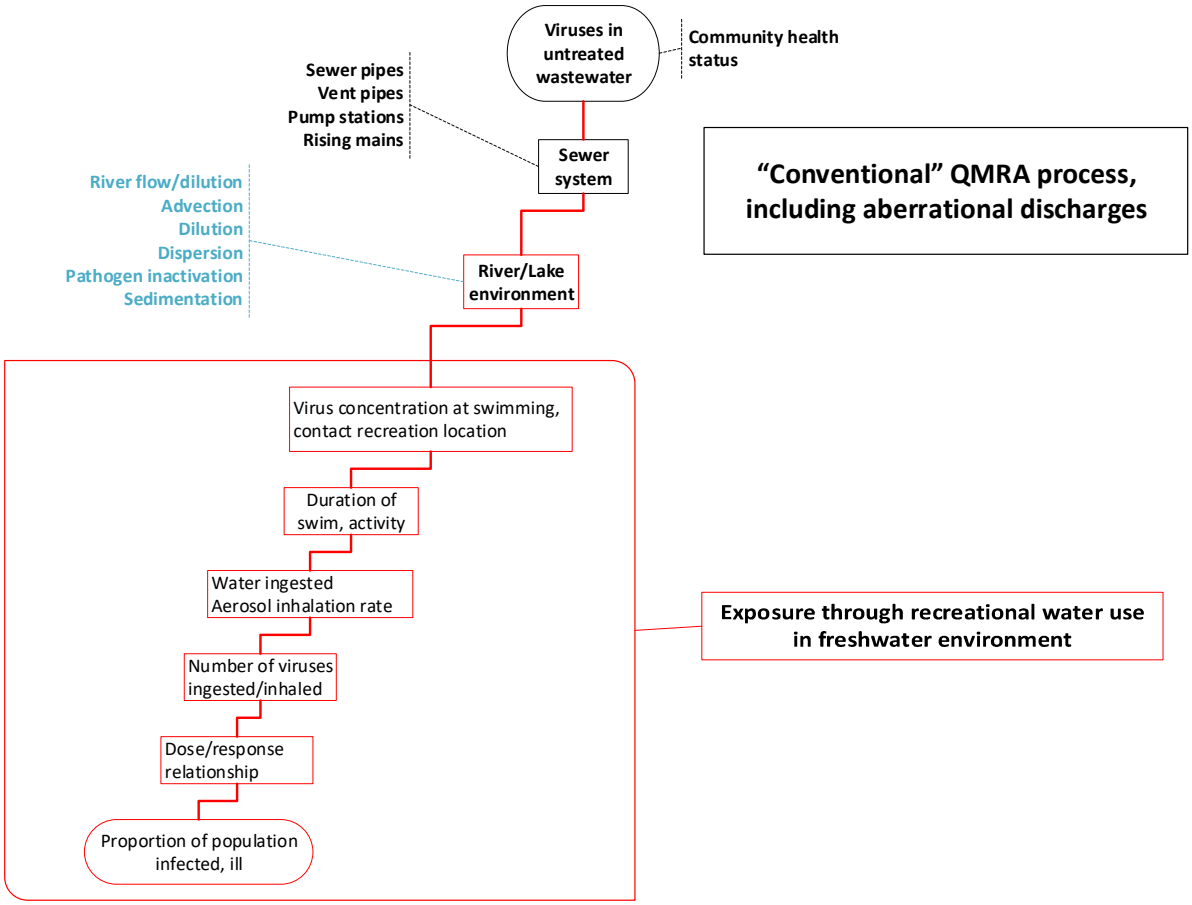

Figure 4-1: Process followed to relate human health risk to pathogen-contaminated surface waters. Items in blue are not available for this health risk assessment.

- The red lines and boxes in Figure 4-1 indicates the path followed from source ("Viruses in wastewater"), to the numbers of individuals likely to become infected or ill. Because a large representative "population" is used for the calculation, the results are generally expressed as a proportion.
- Callout boxes indicate the type of information or data required to make the process work.
- Data and information is required for the processes identified in the red boxes as well, but these data and information are less site-specific, and may be accessed from the literature, or values may be assumed (e.g., "Duration of swim or other activity").

In a full QMRA, these data and information are used as follows:

- Distributions of concentrations are created in response to a range of factors, such as river discharge/flow, tidal movements, tidal stage, rainfall, and wastewater treatment efficacy.
- Once these distributions are created, "recreational users" are exposed to a large number of likely concentrations, selected randomly using a Monte Carlo procedure.
- The pathogen concentration is also likely to vary widely according to the health status in the local community, the relative dilution of the wastewater, as well as in response to factors causing virus inactivation or attenuation. A likely range of concentrations based on measured values is used in this work – this is described in detail in Section 4.3.2.
- When calculating risks, use "Monte Carlo" statistical modelling, which calls for repetitive sampling from distributions and ranges of key variable concentrations, rather than just using single average concentration values. This approach is particularly important given that much of the risk is caused by combinations of inputs toward the extremes of their concentration ranges, the combined effects of which may not be detected when using average concentration values.
- The concentration of pathogens directly controls the size of infectious doses – the volume of water that needs to be ingested to be exposed to the number of organisms ('dose') required to cause infection.[3]
- This effectively allows the health risk to be estimated following exposure of a hypothetical large population size (typically 1,000 "individuals"), exposed on any particular "day".
- The output from this modelling process are estimates of **illness** risk (as opposed to just infection), attributable to the discharge of wastewater. These health risks were calculated for individuals engaged in **primary contact recreation** near the discharge. "Primary contact recreation" refers to activities such as swimming and paddling where full immersion is anticipated, i.e., ingestion of contaminated water is likely to be an outcome of recreation.

Items A), C) and D) above may be addressed using reported data, values from the scientific literature, or other information that is relatively easily available. Item B) however is more problematic. Review of existing information indicates that data are not currently available regarding the likely dispersion and dilution of materials discharged from the wastewater network to either streams or the lake. This makes

[3] Different individuals have different responses to a given dose, with some becoming infected, others not. Infectivity is therefore characterised by a dose-response curve ('function') and risk calculations need to be made for this range of sensitivity. Using averages to calculate a single risk value is highly inaccurate.

estimation of the concentrations of pathogens in receiving waters impossible, and as a consequence, estimation of the dose of pathogens to which human receptors may be exposed is also impossible.

Previously Palliser,Hudson (2018) attempted to estimate risks using information derived from expert opinion, information derived from other work (including advection, fate of sediment and likely impact of other contaminants on water), but the uncertainty arising from absence of key information makes such assessments overly conservative. While it is good to be cautious when considering risks arising from pathogen exposure (where the consequences can be severe), overly conservative risk estimation is likely to be unrealistic, alarmist and unhelpful when assessing human health risk.

4.2 Reverse QMRA approach

A "reverse QMRA" may be undertaken in some circumstances. This process recognises at the outset that key data and information are missing, and that a full QMRA is therefore impossible. Using certain assumptions, the process then focuses on the human health risk and the amount of dilution required to achieve accepted health risk thresholds, such as those defined in the MfE/MoH Guidelines. This approach was previously followed by Hudson,McBride (2017). In the case of the QLDC wastewater network, information is lacking for:

- measured wastewater volumes from overflow discharges
- the dilution and fate of diluted wastewater in the receiving environment, and
- the dilution of wastewater by stormwater during discharge events (where these coincide with rainfall events).

A reverse QMRA approach delivers a table of risk estimates associated with varying levels of wastewater dilution. These results allow the community, health and regulatory agencies to understand and make decisions based on the relative health risks.

4.3 Reverse QMRA assumptions

We considered the locations of recorded sewer and pump station leaks or discharges in Section 2. This identified locations at which measurable risk may have previously existed.

The varying and unknown dilution arising from discharge of untreated wastewater overflow discharge into streams, rivers and lakes are accounted for by considering a range of dilution scenarios. For this study, $\log_{10}$ of dilution ranged from 1 (10× dilution) through to 5 (100,000× dilution). Rather than attempting to predict where risks exist (because the information required to do this just not exist), we consider what human health risk is likely to exist for a series of dilutions.

Although the approach is less sophisticated than that provided by the full QMRA process, it allows the relative risks of illness to be estimated for the conditions likely to be created by each dilution scenario. This is adequate for the purposes of this assessment. To undertake a full QMRA exercise, it would be necessary to predict the fate and dilution of pathogens arising from the wastewater discharge. This would require (among other things):

- development of a mixing and dilution model for each of the streams where wastewater contamination could occur
- a calibrated hydrodynamic model foreach lake to which untreated wastewater could be discharged, or to which a contaminated stream may discharge; a model of this nature would require substantial calibration data, including (but not limited to):

- measured stream discharge data
- lake water current, speed and direction etc.

NIWA has undertaken the human health risk assessment using:

1. Recently published scientific literature that has revisited previously accepted relative risk factors.
2. Estimates of wastewater pathogen concentrations.
3. Estimates of the range of dilution likely to occur in the receiving water – in this approach, the extent of dilution required to achieve specific risk thresholds were identified, rather than estimating the likely concentrations of pathogens.
4. Estimates of virus ingestion rates.
5. Available dose-response relationships for a representative virus.

We describe these selections below:

4.3.1 Selecting the pathogen(s) of concern

Several viruses may be used for risk assessment. In this study we use a model pathogen – Norovirus. This is appropriate because Norovirus is reported to be the most common aetiological agent in receiving waters (e.g., Sinclair et al. 2009), and the infection ID_{50} is in the order of 20 virions (among susceptible people). The dose-response curve indicates that ~20% of people may become infected after ingestion of just one virion.

4.3.2 Assessing exposure - predicting doses

To turn concentrations into doses we need:

1. Wastewater virus concentrations.
2. Ingestion or inhalation rates for recreational users exposed to wastewater contaminated waters.

Details on how these factors have been modelled and enumerated are given in Table 4-1.

Water ingestion rates by swimmers—a key component of dose-calculation—were studied using novel biochemical procedures in a pilot study (Dufour et al. 2006). These authors report a clinical trial observing 53 volunteers involved in recreational swimming in an outdoor community swimming pool. Using cyanuric acid (a decomposition product of chlorine-stabilising chloroisocyanurate) as a tracer, the volume of water ingested during active swimming events lasting at least 45 minutes was calculated for each swimmer. It has become standard practice to apply these ingestion rates to water recreation.[4]

The focus on "primary contact recreation" does not imply that exposure through other forms of recreation does not create risk. The health risks associate with paddle-boarding or canoeing are likely to be lower (there is little opportunity for ingestion), unless the individual capsizes or falls into the water. At such time, they are likely to have similar ingestion rates as a swimmer, but the duration of exposure is likely to be shorter. If the individual remains in the water for a longer period, then both ingestion rate and duration of exposure are likely to be similar to those of the swimmer. The swimming health risk is therefore a reasonable surrogate for other recreational users.

[4] Personal communication: Jeff Soller, Soller Environmental, California (http://www.sollerenvironmental.com/env/main/Home.html).

Individuals standing on the banks or shoreline of a contaminated water body are unlikely to face measurable health risk, because no direct ingestion route exists.

Where discharge occurs to land and then to water following runoff, an additional contaminant source will exist – fomites. These are (generally) inanimate objects that are contaminated and which subsequently enable transfer of a pathogen to a target via a secondary route of exposure (e.g., oral or direct contact). Examples of fomites include contaminated soil, plants, toys and tools. Inundation of footpaths, parklands and play grounds following a wastewater discharge would create the circumstances where fomites might have a role in disease transmission. Work undertaken in other studies (e.g., in the Heathcote and Avon River catchments, Christchurch, (Palliser et al. 2009)) suggests that the numbers of individuals likely to be exposed to pathogens via contact with contaminated objects is small, and the degree of exposure is low to very low. Overall, these risks were considered best-addressed through pollution response procedures.

4.3.3 Characterising dose-response

These relationships are mostly inferred from data reported by "volunteer studies" (i.e., clinical trials). These have been done for a restricted number of viruses. In these studies, healthy adult volunteers (typically between 50 and 100, in groups of 10 or so) are individually challenged with a pathogen dose and their infection and illness states are monitored for a few days thereafter. Such a study has been conducted for noroviruses (Teunis et al. 2008). Occasionally data from viral illness outbreaks become available, from which dose-response information can be inferred.[5] Note that to perform QMRA calculations, comparability between the definition of "dose" used in the clinical trial or outbreak study and the methods used in assessing virus concentrations in the wastewater of concern is required. For example, when assessing pathogens in treated wastewater, noroviruses cannot be cultured, so a quantitative molecular-based laboratory procedure (Reverse Transcription Polymerase Chain Reaction "RT-qPCR") is used to detect the norovirus genome. Since RT-qPCR detects genetic material, the method picks up both viable and non-viable viruses. Since there are variants of the qPCR procedure, some harmonisation between the methods used in the clinical trial and wastewater Norovirus enumeration methods may be required (and is so in this study).

4.3.4 Conducting the health risk assessment

To adequately reflect limits to knowledge on key features of the risk assessment, Monte Carlo statistical modelling is used (Haas et al. 1999, McBride 2005a). In simpler models, key inputs are described by a single number (e.g., wastewater treatment plant (WWTP) influent pathogen concentration). However, such inputs are known to be variable and some are uncertain. The way this variability and uncertainty has been addressed is shown in Table 4-1. The proprietary Excel plug-in product "@RISK" was used to perform the calculations, incorporating factors that reflect these distributions and inputs (Palisade Corp 2013).[6] The models were run for 1,000 iterations for the selected virus, for the proposed virus concentration distribution, and for each of five dilution scenarios. During each iteration, 100 individuals were 'exposed', by taking a random sample from statistical distributions covering the range of possible doses received by individuals ingesting water possibly containing pathogen.

It can be appropriate to report the results in terms of infection (which is the approach taken for the freshwater component of the MfE/MoH Guidelines), rather than illness. For the present study where Norovirus is the model pathogen, we take standard values of the probability of illness, given that infection has occurred. The output metric is an individual's illness risks, to facilitate comparison with relevant

[5] An example is a study by Thebault et al. (2013) of norovirus illness outbreaks among consumers of oysters in southern France.
[6] The @RISK models use named cells as much as possible, to facilitate checking and readability.

guidelines. [7,8] We do however account for "aggregation" – clumping together of viral particles to form a single infectious mass, rather than existing as several or many discrete particles. The extent and likelihood of aggregation is determined by the presence and amount of organic matter able to facilitate attraction between and binding of these infectious agents.

[7] There is insufficient time and information to also compute DALY metrics (Disability-Adjusted Life Years) as often used when assessing health risks associated with drinking-water (WHO 2011, chapter 7).

[8] The individual's illness risk (IIR) is calculated as the total number of predicted illness cases divided by the total number of exposures to potentially contaminated water or shellfish flesh. It represents the risk to an individual swimmer or shellfish consumer on any day, having no prior knowledge of any contamination from the outfall. It is calculated via the Monte Carlo modelling, for which 100 individuals are exposed on each of 1,000 separate days, i.e., 10^5 exposures. The total number of cases is 1,000m where m is the mean infection case rate over 100 people (readily calculated by the Monte Carlo software—@RISK, Palisade Corp. 2013). So the individual's infection risk, expressed as a proportion, is 1,000m/10^5 = m/100. When expressed as a percentage, IIR = m%.

Table 4-1: Distributions and inputs for the QMRA. Plain numbers in the Statistics column are for typical health conditions in the local community; italicised numbers are for the rare case where there is a norovirus illness outbreak in that community. [a]

Component	Statistics		Distributions/comments
Influent virus concentrations			Bounded "hockey stick" distribution (McBride 2005a), strongly right-skewed with a hinge at the 95%ile.
Sewage norovirus concentration, genome copies per litre	Minimum = 10^3 *10^3* Median = 10^4 *10^6* Maximum = 10^6 *10^7*		Typical ranges found for New Zealand cities (e.g., Napier, New Plymouth—McBride 2011, 2012, 2016).
Duration of swim (hours)	Minimum = 0.1 Median = 0.5 Maximum = 4		Child receptor.
Swimmers water ingestion rate, mL per hour	Minimum = 20 Median = 53 Std. Dev. = 75 Maximum = 270		Lognormal distribution, for a child (adult rate is half this rate). For a review on this see Wood et al. (2015, sec. 6.2.1).
Dose-response equations and parameters	–		Appendix A Norovirus, disaggregated: beta-binomial [eq. (5)]: $\alpha = 0.04$, $\beta = 0.055$ (so $ID_{50,infection} = 26$); Pr(susceptible) = $P = 0.72$ (Teunis et al. 2008); Pr(ill | Infection) = 0.60 (Soller et al. 2010). Also Messner et al. (2014) exponential equation for aggregated norovirus: $Pr_{inf} = [1 - P\, e^{d/\mu}]$, where d = dose and μ = mean aggregate size (taken as $\mu = 1106$).

[a] Those high values, persisting for over a month, have not been seen in subsequent Mangere influent virus assays. Yet were they to recur during an undetected outbreak in the contributing community, one could expect elevated illness risk.

5 Estimating public health risk

Table 5-1 summarises the predicted number of illness cases (out of 100 people exposed on any random occasion) and the Individual Illness Risk (IIR). The Individual Infection Risk (IInR) is summarised in Table 5-2. The values in these tables indicate the proportion of exposed individuals who are predicted to either become ill or infected respectively. In both cases the proportion is expressed as a percentage.

- Comparison of the model output in Table 5-1 and Table 5-2 indicates that illness risk is always lower than the infection risk – illness requires infection, whereas infection does not necessarily lead to illness.
- In both Table 5-1 and Table 5-2, aggregation of pathogens decreases infectivity and risk of illness considerably.
- In both Table 5-1 and Table 5-2, the risk of illness and infection decreases as dilution increases.

It is informative to relate these illness or infection risks to those implicit in and defined in the MfE/MoH Guidelines (e.g., Hudson and McBride 2017; Hudson and Wadhwa 2017). For example, following grading of a recreational beach using FIB concentration derived from a weekly surveillance monitoring programme, it is possible to relate a B grade (where 95th percentile enterococci concentrations fall in a range from 41 to 200 enterococci/100 mL)[9] to a 1-5% gastrointestinal illness risk, or a 0.3 – 1.9% acute febrile respiratory illness risk. These risks can be restated as an average probability of illness of one case of gastroenteritis in 20 exposures, or approximately one case of acute febrile respiratory illness in 50 exposures.[10]

In the case of the Waimea Inlet (Hudson and McBride 2017), the illness risks derived from the QMRA modelling (similar to those summarised in Table 5-1 and Table 5-2 below) could be related directly to the MfE/MoH Guideline risks because the latter were derived from viral pathogens and a QMRA.

In the case of freshwaters, the MfE/MoH Guideline risk values relate to *E. coli* concentrations as an indicator of *Campylobacter* infection and illness risk. We do not currently have equivalent illness risks defined for viral pathogens in freshwater. For freshwaters therefore, we must identify and discuss human health risks entirely in terms of the risks of illness and infection for the model pathogens selected for the QMRA. In this assessment, we selected Norovirus.

These results indicate a potential for health risk arising from the discharge of untreated wastewater in the conditions assumed in each scenario. All other factors remaining constant, the health risk is strongly determined by dilution of the pathogen load discharged to water. Although absence of dilution and advection information limits the usefulness of these health risk estimates (for example, we cannot predict where any particular level of risk will apply, or the time period during which a level of risk will apply), the risks implicit in the MfE/MoH Guidelines may be used as examples of other levels of risk with which communities, regulatory agencies and community health protection professionals are familiar.

[9] Refer to Table D1 of the MfE/MoH Guidelines (p D6)
[10] Refer to Table H1 of the MfE/MoH Guidelines (p H25)

As discussed previously, swimming at a B grade recreational beach (where 95th percentile enterococci concentrations fall in a range from 41-200 enterococci/100 mL) has an inferred risk of one case of gastroenteritis in 20 exposures, or approximately one case of acute febrile respiratory illness in 50 exposures. An individual may consider this risk of illness to be high or low, but generally regulatory agencies and public health protection agencies would regard this beach grading as indicating "good to high" microbiological quality. Warning signs would not be present, and the public would not be discouraged from swimming at a beach with this grading, i.e., where this level of health risk applied. When considered using a traffic light approach, this grading would generally be regarded as green.

Currently we do not have actual or predicted dilution data, which makes it difficult to decide what level of risk exist, and whether as individuals or public health protection specialists the risk is "acceptable". As discussed in Section 4.1, more accurate estimation of the health impact of sewer discharges therefore requires use of a mixing dilution model at each discharge point, and use of a calibrated hydrodynamic model, able to represent the mixing, dilution and advection of contaminants within various lake receiving environments. A calibrated hydrodynamic model would also allow the IIR to be calculated at any location within the model domain.

Irrespective of whether a hydrodynamic model is available or not, or whether we have perfect knowledge regarding the actual concentrations at any point in the environment following an untreated wastewater discharge event, some form of response would be required to mitigate human health risk. This is discussed in Section 6.

Table 5-1: Individual Illness Results for aggregated and disaggregated norovirus for five concentration orders, assuming typical illness patterns in the local community. Norovirus concentrations were assumed to range from 1,000 (min), 100,000 (median) and $1E^7$ (maximum) (virus particles/L).

Statistic	Norovirus, disaggregated, for five log_{10} dilution orders					Norovirus, aggregated, for five log_{10} dilution orders				
	1	2	3	4	5	1	2	3	4	5
Min	16	3	0	0	0	0	0	0	0	0
5%ile	22	14	2	0	0	0	0	0	0	0
10%ile	23	17	3	0	0	0	0	0	0	0
15%ile	25	19	4	0	0	0	0	0	0	0
20%ile	26	20	4	0	0	0	0	0	0	0
25%ile	27	21	5	0	0	0	0	0	0	0
30%ile	27	22	6	0	0	0	0	0	0	0
35%ile	28	22	6	0	0	1	0	0	0	0
40%ile	29	23	7	0	0	1	0	0	0	0
45%ile	29	24	7	1	0	1	0	0	0	0
50%ile	**30**	**25**	**8**	**1**	**0**	**1**	**0**	**0**	**0**	**0**
55%ile	30	25	8	1	0	1	0	0	0	0
60%ile	31	26	9	1	0	1	0	0	0	0
65%ile	32	27	9	1	0	2	0	0	0	0
70%ile	32	27	10	1	0	2	0	0	0	0
75%ile	33	28	11	2	0	2	0	0	0	0
80%ile	34	29	11	2	0	2	0	0	0	0
85%ile	35	30	13	2	0	3	1	0	0	0
90%ile	36	31	14	3	1	4	1	0	0	0
95%ile	37	33	20	4	1	6	1	0	0	0
96%ile	38	34	23	8	2	11	2	0	0	0
97%ile	39	35	26	15	2	18	3	1	0	0
98%ile	40	36	29	20	3	25	4	1	0	0
99%ile	41	38	31	25	5	29	7	1	0	0
99.5%ile	42	39	33	27	7	32	9	2	0	0
99.9%ile	43	43	37	31	8	34	11	3	0	0
Max	46	43	38	31	9	35	12	3	1	0
Mean (= IIR)	**29.752**	**24.411**	**8.633**	**1.701**	**0.252**	**2.236**	**0.327**	**0.043**	**0.001**	**0**

Table 5-2: Individual Infection Results for aggregated and disaggregated norovirus for five concentration orders, assuming typical illness patterns in the local community. Norovirus concentrations were assumed to range from 1,000 (min), 100,000 (median) and 1.10^7 (maximum) (virus particles/L).

Statistic	Norovirus, disaggregated, for five $\log_{10}$ dilution orders					Norovirus, aggregated, for five $\log_{10}$ dilution orders				
	1	2	3	4	5	1	2	3	4	5
Min	29	4	0	0	0	0	0	0	0	0
5%ile	41	27	3	0	0	0	0	0	0	0
10%ile	43	31	5	0	0	0	0	0	0	0
15%ile	44	34	7	0	0	0	0	0	0	0
20%ile	45	35	8	0	0	1	0	0	0	0
25%ile	46	36	9	0	0	1	0	0	0	0
30%ile	47	37	10	1	0	1	0	0	0	0
35%ile	47	38	11	1	0	1	0	0	0	0
40%ile	48	39	12	1	0	2	0	0	0	0
45%ile	49	40	12	1	0	2	0	0	0	0
50%ile	**50**	**41**	**13**	**1**	**0**	**2**	**0**	**0**	**0**	**0**
55%ile	50	42	14	2	0	2	0	0	0	0
60%ile	51	43	15	2	0	2	0	0	0	0
65%ile	52	43	16	2	0	3	0	0	0	0
70%ile	52	44	17	2	0	3	0	0	0	0
75%ile	53	46	18	2	0	3	0	0	0	0
80%ile	54	47	19	3	0	4	1	0	0	0
85%ile	55	48	20	3	1	4	1	0	0	0
90%ile	57	50	22	4	1	5	1	0	0	0
95%ile	59	52	29	6	2	9	2	0	0	0
96%ile	60	52	39	17	2	17	2	1	0	0
97%ile	60	53	43	25	3	31	5	1	0	0
98%ile	61	55	48	31	6	43	7	1	0	0
99%ile	63	58	51	38	10	49	13	2	0	0
99.5%ile	64	60	53	41	11	51	14	2	0	0
99.9%ile	66	63	55	44	13	52	17	4	1	0
Max	67	69	56	47	13	55	18	4	1	1
Mean (=IInR)	**49.653**	**40.567**	**14.435**	**2.788**	**0.405**	**3.762**	**0.556**	**0.07**	**0.004**	**0.001**

6 Minimising adverse effects

From a public health protection perspective, the development of a suitable overflow response management plan is an essential requirement for minimising the potential adverse effects of wastewater discharges. Irrespective of whether results from a full QMRA are available or not, actions such as the following are essential:

- repair of the fault(s) in the sewer network and cessation of further discharge
- where possible, bunding and recovery of wastewater before it enters a stream or lake
- identification of areas where contamination is likely, taking measures to alert the public to this effect
- Notifying Otago Regional Council and public health authorities
- Signage to inform the public.

Once immediate response to a contamination event is underway, actions such as microbial water quality monitoring would be appropriate. Collection of samples would provide a direct measure of the concentration of faecal indicator organisms (specifically *E. coli*). The pollution response monitoring should have both a temporal and spatial perspective:

- water quality samples should be collected upstream of the discharge site, and at downstream sites, particularly at locations where recreational exposure is likely
- in the event of discharge of pathogens to lake waters, samples should be collected along the shoreline either side of the discharge point
- these spatial samples would help define the likely area where elevated risks may prevail, and further signage or public notification could be undertaken.
- The period over which samples should be taken would be determined by the results of the monitoring:
  - as long as elevated FIB concentrations are observed, monitoring should persist
  - once FIB concentrations have returned to typical or before discharge values, cessation of event-related monitoring should be considered
  - the decision to discontinue monitoring should probably involve stakeholder groups and be part of the overall response plan.

The signage and public notification will minimise risk to recreational users, and the sampling will help determine when waters may be considered safe again for recreational use. Several agencies are generally involved in these response activities, guided in part by the MfE/MoH recreational water quality guidelines, as well as other emergency response plans.

The discussion regarding the location and frequency of discharge events (Section 2.1) indicated that these events should probably be considered as being of extremely low probability.

The definition of hazards, risk and risk assessment provided in Section 3 indicates the requirement to also consider mitigation measures.

If a robust plan exists to respond adequately to discharge event such as these, it would be appropriate to consider these events to have extremely low probability and moderate risk, and the overall health risk to local communities will be low to very low. We have reviewed the incident response plan of QLDC, and we consider that:

1. It is suitable as a high-level strategy document, but that considerable additional detail should be provided before it can be considered sufficiently robust.

2. It should incorporate the term "adaptive management" as a descriptor, and

    i. the more detailed plan that should be associated with it should be implemented in an adaptive management framework

    ii. the plan should also be described as a "living document", and should be revised or modified to allow QLDC to better respond to future events.

3. The plan should also explicitly make reference to Section H of the MfE/MoH Guidelines, where the principles underlying a pollution-response strategy are described, and detailed information and practical guidance is provided. In part this will allow these response plans to be customised to meet local conditions and community expectations. One area where immediate further attention could be given is with regard to post-discharge monitoring.

    i. A suitable event-related microbiological water quality monitoring programme should be developed in association with other agencies, addressing aspects such as the sampling locations, frequency of sampling, and specific laboratory tests will be agreed.

    ii. In some circumstances it may be possible to utilise continuous water quality measurements of FIB surrogates, or other water quality variables to provide supporting information.

    iii. The water should be tested at the agreed frequency and locations until the water quality is back to acceptable standards.

    iv. Although limited water quality data exist for most of the Queenstown Lakes District, data derived from the recreational water quality monitoring programme operated by ORC provides approximately 25 water quality results annually for several sites in the region.

    v. These data are accessible from the LAWA website[11] and may be suitable for defining "typical water quality" for some parts of the Queenstown Lakes District. Graphical summaries of recent recreational monitoring derived from the LAWA website are included in Appendix D.

[11] https://www.lawa.org.nz/explore-data/swimming/

7 Conclusions

We have described why absence of key data makes conduct of a conventional QMRA impossible. However, by using a model pathogen, a series of assumptions and applying ranges and distributions of several input variables, we were able to relate health risk to the level of wastewater dilution. The results provided in Section 5 indicate a potential for health risk arising from the discharge of untreated wastewater in the conditions assumed in each scenario. All other factors remaining constant, the health risk is strongly determined by dilution of the pathogen load discharged to water.

These estimates of risk were compared with the levels of risk associated with recreational water quality grading to illustrate that approximately 3500× dilution would be required to reduce the illness risk for sewage discharged from the sewer network to less than 5% gastrointestinal illness risk, which is present in B grade waters (defined by the MfE/MoH (2003) recreational water quality guidelines).

In Section 2, risk was defined as "chance + hazard + exposure + consequence". Routine maintenance and management of the sewer network by QLDC will reduce the **chance** of discharge of untreated wastewater. The **hazard** is in part determined by the health status in the community, and is out of the control of QLDC. The development and implementation of a suitable overflow response management plan is an essential requirement for minimising human health risk associated with wastewater discharges. The response plan should allow for adaptive management, and should incorporate a suitable microbiological water quality monitoring plan. An inter-agency response, erection of physical barriers, signage, and other means of notifying the public will contribute to reducing the opportunities for the public to be exposed to potentially contaminated waters.

If QLDC implements the recommended response processes identified in Section 6 above then I consider the risk to human health arising from occasional discharge of wastewater from the sewer network to surface waters to be low to very low.

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10 Glossary of abbreviations and terms

Aetiological agent	Microorganisms and microbial toxins that cause disease in humans.
Beta-Binomial dose-response curve	A mathematically-derived infection dose-response curve for variable infectivity, in which individual doses are known.
Beta-Poisson dose-response curve	A mathematically-derived infection dose-response curve for variable infectivity, in which only mean doses are known.
Conditional illness probability	The probability of illness at a given dose given that infection has already occurred.
Conditional infection dose-response models	The (simpler) mathematical form of a dose-response equation that results when individual doses are known. (More complicated mathematical functions arise when individual doses are not known).
Hypergeometric functions	Mathematical equations that defy simple calculation, yet are important in the analysis of clinical trial data and outbreak data for the infection response of a population exposed to a pathogen, and where individual doses are randomly distributed about a known mean value.
Illness ID_{50}	The dose required to cause illness in 50% of an exposed population, who are already infected.
Infection ID_{50}	The dose required to cause infection in 50% of an exposed population.
PCR	Polymerase Chain Reaction, a molecular technique for virus enumeration using DNA segment matching.
QMRA	Quantitative Microbial Risk Assessment.
RT-qPCR	Reverse-transcription quantitative PCR, used for RNA viruses.
Sequelae	An illness that is the result of a previous disease.
Simple binomial dose-response curve	A mathematically-derived infection dose-response curve for constant infectivity, in which individual doses are known.
Simple exponential dose-response curve	A mathematically-derived infection dose-response curve for constant infectivity, in which only mean doses are known.
$TCID_{50}$	Median Tissue Culture Infectious Dose: A laboratory culture technique measuring the amount of virus that produces a cytopathic effect in 50% of cell cultures inoculated.
Virion	Shorthand for “virus particle”.

Appendix A Dose-response functions

For infection

Standard clinical trial procedures involve challenging groups of volunteers with aliquots taken from serially-diluted preparations whose well-mixed concentrations are measured. Doses in individuals' challenges are not measured. Consequently only the average dose given to each member of a group is known. Nevertheless, by making two simple assumptions the mathematical form of the infection dose-response equation can be obtained (Haas et al. 1999, McBride 2005a):

1. The "single-hit" hypothesis: That a single pathogen, surviving the body's barriers (e.g., acidic digestion system) and reaching a potential infection site, is sufficient to cause infection.
2. Poisson distribution of pathogens in the preparation—as is appropriate for a random well-mixed population.

The mathematical result, after averaging across each group's individual Poisson-distributed doses, is the single-parameter "simple exponential" equation:

$$\Pr_{inf}(d) = 1 - e^{-rd} \quad (1)$$

where d is the average doses given to each group, "e" is the standard exponential number (the base of natural logarithms, e = 2.7183…), and r is the probability that a pathogen survives the body's defences and reaches an infection site.

Sometimes host-pathogen interactions are such that a constant value of r is implausible (e.g., because of differential immunity, or varying pathogen virulence, as indicated by lack of fit to the single-parameter model). In that case r is replaced by a standard two-parameter beta distribution with shape parameter α and location parameter β. The mathematical result is the much-more-difficult-to-evaluate[13] Kummer hypergeometric function (denoted as ${}_1F_1$):

$$\Pr_{inf}(d) = 1 - {}_1F_1(\alpha, \alpha + \beta, -d) \quad (2)$$

For obvious reasons this can be called the "beta-Poisson" equation.[14] Fortunately in many cases we find that β >> 1 and α << β, in which case this equation can be well-approximated by the following equation (confusingly, also called "beta-Poisson"):

$$\Pr_{inf} = 1 - \left(1 + \frac{d}{\beta}\right)^{-\alpha} \quad (3)$$

However this approximation is inadequate for noroviruses because the fitted parameter doublet (α = 0.04 and β = 0.055, Teunis et al. 2008) constitute a serious breach of the approximation-validity criteria (α << β, β>>1). Analysis of clinical trial data for noroviruses therefore calls for specialist software that can evaluate (2), as reported by Teunis et al. 2008, Thebault et al. (2013).

[13] Equation (2) can't be evaluated in Excel.

[14] Because a two-parameter (α and β) beta distribution is used instead of the single parameter r and the doses are assumed random, i.e., Poisson-distributed. Strictly, β is not properly a location parameter for equation (2), but it is for its approximation equation (3) (because d is simply divided by β in that equation: increasing the value of β shifts the curve to the right).

Simplifying the infection dose-response calculations for QMRA

Good QMRA practice, especially for virulent pathogens, is to "expose" *multiple* people on each exposure occasion.[15] In that case the individual doses are known (i.e., are calculated and assigned to individuals by the model) so that there is no need for Poisson-averaging. This somewhat simplifies the mathematical development of the infection dose-response formulae such that for constant r the simple one-parameter exponential model is replaced by the simple binomial model:

$$\mathrm{Pr}_{\mathrm{inf}} = 1-(1-r)^{i} \tag{4}$$

where *i* is the individual's dose.

Also, the two-parameter beta-Poisson model (the ${}_1F_1$ functional form) is replaced by the "beta-binomial" model:

$$\mathrm{Pr}_{\mathrm{inf}} = 1-\frac{\mathrm{B}(\alpha,\beta+i)}{\mathrm{B}(\alpha,\beta)} \tag{5}$$

where B is the standard beta function (Abramowitz & Stegun 1972) and α and β are as defined previously. This equation can be simply evaluated in Excel.[16]

These two equations have been described by Haas (2002) as conditional infection dose-response models, the condition being that individual doses are known.

The following figures (Figure A-1a&b) give examples of these functions for adenovirus 4 and for Norwalk virus, for both conditional and unconditional infection dose-response models.

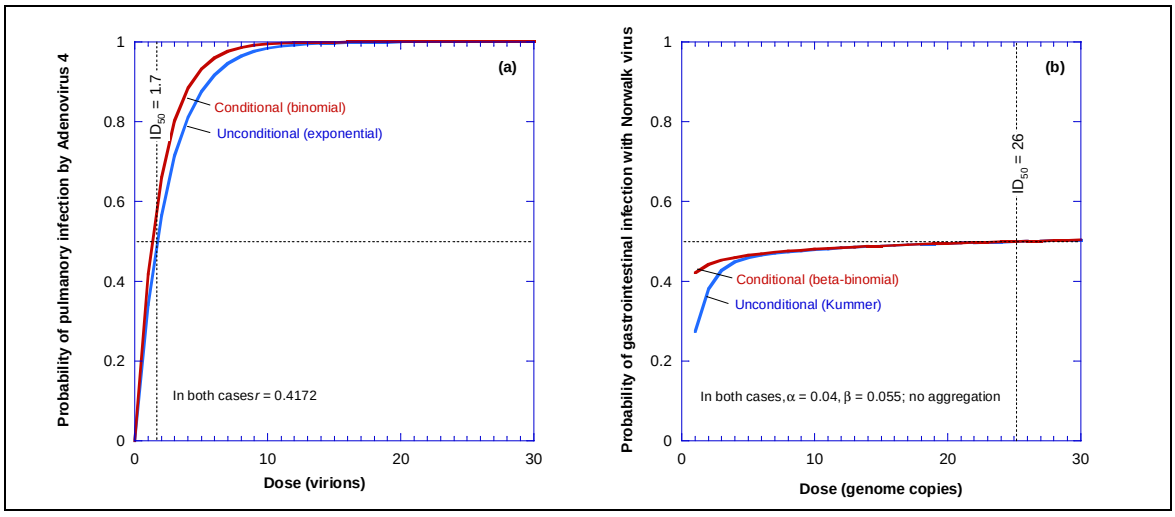

Figure A-1: Conditional and unconditional infection dose-response curves. (a) single-parameter models for adenovirus 4, and (b) double-parameter models for Norwalk virus (only for susceptible individuals).

[15] To not do so gives rise to implausible risk profiles. For example if only one individual is exposed per exposure occasion—as a representative of a group visiting a contaminated beach—and if the probability of infection given ingestion of one pathogen is high (say, 20%), then probabilities of infection *between* 0% and 20% are impossible. The resulting risk profile becomes extremely jagged (McBride 2005b). In such cases exposing a group of people per exposure occasion (say, 100), each with different doses (some swim for a few minutes, others for an hour or so), allows many values between 0 and 20% to be calculated.

[16] To do so we note that B(α,β) = Γ(α)Γ(α)/Γ(α+β), where Γ is the standard Gamma function (Abramowitz & Stegun 1972). Standard Excel includes the natural logarithm of the gamma function (as the function 'GAMMALN'), so that we can derive : Pr = 1 – EXP{GAMMALN(β+*i*) + GAMMALN(α+β) – [GAMMALN(α+β+*i*) + GAMMLN(β)]}.

These graphs highlight some important features of infection dose-response curves:

- The single-parameter models (e.g., Figure B-1a) rise inexorably to unit probability, precisely because their common parameter (r) is constant.
- The double-parameter models (e.g., Figure B-1b) "flatten out" well before reaching unit probability.[17]
- Whilst the relatively high infection ID_{50} for Norwalk virus (26 genome copies among susceptible individuals) occurs on the flattened top of its dose-response curve, infection probabilities are still appreciable at much lower doses.[18]
- The unconditional curves have a jagged profile around the conditional forms, yet deploying the latter in a QMRA gives rise to the same averaged risk.[19]
- Whilst the adenovirus 4 infection dose-response curve is in all respects more severe than that for Norwalk virus, for two reasons that doesn't mean that it is the most severe pathogen:
    i. adenoviruses that can cause respiratory ailments are a minor part of the total adenovirus population in sewage,[20] with most causing gastro-intestinal illness
    ii. exposure to respiratory adenoviruses (via inhalation, e.g., whilst surfing) tends to be lower than ingestion of water whilst swimming.[21]

However, having double-stranded DNA, adenoviruses are more resistant to disinfection processes.

For illness

Some individuals who become infected (e.g., as measured by serological response, or by evidence of pathogen shedding) may not go on to exhibit symptoms, i.e., they are asymptomatic. In that case, to obtain the unconditional probability of illness (given dose) we first need to calculate the conditional probability of illness given infection for each dose, denoted as $Pr_{ill|inf}$. The probability of illness is calculated as:

$$Pr_{ill} = Pr_{ill|inf} Pr_{inf} \quad (6)$$

Two common approaches are used for the conditional illness function:

[17] In fact these models approach unit probability only for enormous doses.
[18] The "flat top" is caused by the variable host-pathogen interactions, including a proportion of exposed population who high (but incomplete) immune. There is also another group who are completely immune.
[19] That's because applying the unconditional form to a single individual representing a group of people, as is common practice, doesn't capture the fact that, by good luck, some people at a beach will avoid exposure whilst the averaged dose is above zero (McBride 2005b).
[20] Typically respiratory serotypes are detected less frequently than adenovirus F serotypes and so the gastro-intestinal (GI) disease-causing serotypes tend to predominate in sewage studies (Osuolale & Okoh 2015). However, a proportion of respiratory versus GI serotypes detected will depend on the cell line used for culture assays and the target primers for molecular methods. For example, Hewitt et al. (2011) used cell line 594 and reported that culturable adenoviruses were mainly A-E types (which are respiratory and conjunctivitis serotypes) and there was still around 3 log presence in effluents.
[21] Water-contact-related respiratory illness is an area worthy of further research, particularly in the light of the respiratory illness rates reported in the one New Zealand epidemiological study on this matter—McBride et al. (1998). In that study (at seven New Zealand beaches) those rates were generally more prominent than gastrointestinal rates, a phenomenon that is not fully understood.

Hazards model

Teunis et al. (1999) developed hazard models for the illness given infection, with two forms:

Decreasing hazard
$$\Pr_{ill|inf}(d) = 1 - \left(1 + \frac{\eta}{d}\right)^{-r} \quad (7)$$

and

Increasing hazard
$$\Pr_{ill|inf}(d) = 1 - (1 + \eta d)^{-r} \quad (8)$$

where η is a location parameter, and *r* is a shape parameter.[22]

Dose independence

Existing models of the conditional probabilities of illness (the condition being that infection has already occurred) are held in some doubt internationally. For example, the norovirus model (Teunis et al. 2008) predicts substantial infection probabilities at very low doses, but predicts substantial illness probabilities (among the infected) only at very high doses. A large body of work has taken the view that the conditional probability of illness-given-infection should be independent of dose—Schoen & Ashbolt (2010), Soller et al. (2010, 2015), Viau et al. (2011) and Boehm et al. (2015). Indeed, that approach is endorsed by WHO (2011), with the result that for the pathogens considered here the conditional illness probabilities are on the order of ½.

[22] The decreasing hazards model has only been reported for a clinical trial on adults exposed to *Campylobacter* (Teunis et al. 1999): All other conditional illness models that I am aware of infer an increasing hazards model, including a *Campylobacter* outbreak study for children (Teunis et al. 2005).

Appendix B Echovirus 12 clinical trial data analysis

Echovirus is a member of the enterovirus family. Haas et al. (1999) reported fitting a one-parameter simple exponential model to clinical trial data for an echovirus 12 study (Akin 1981),[23] with an estimated infection ID_{50} = 54 virions, corresponding to their calibrated *r* value of 0.0128.[24] Haas (1983) had earlier fitted a slightly different value to the Akin data, with $r = 0.012$ (giving infection ID_{50} = 58) and also a two-parameter beta-Poisson curve (with $\alpha = 1.3$ and $\beta = 75$), so that the infection ID_{50} [$= \beta(2^{1/\alpha} - 1)$] = 53. Clearly, these approaches give consistent results with an infection ID_{50} about 50.

The beta-Poisson result was used in the QMRA performed for the Mangere wastewater treatment upgrade (DRG 2002, Simpson et al. 2003), this choice being particularly influenced by the observation that enterovirus illness can give rise to more serious consequences (i.e., sequelae) relative to other virus groups.

Akin's data were in fact preliminary results from an ongoing clinical trial, full results of which were reported three years later in Schiff et al. (1984a&b). Their 1984a paper is the proceedings of a conference held two years earlier in Herzliya, Israel. It contains the Akin data. But the 1984b document (a peer-reviewed journal paper) multiplied all the doses, including those reported by Akin, by a factor of 33, to account for the re-analysis of the stock dose suspension using a more sensitive cell line[25]. These published data were analysed by Teunis et al. (1996) giving rise to a two-parameter "beta-Poisson" model ($\alpha = 0.401$, $\beta = 227.2$, as reported by Teunis et al. 1996) and a higher infection ID_{50} = 1052 virions.[26]

We propose to use the beta-Poisson model ($\alpha = 1.3$ and $\beta = 75$, with infection ID_{50} = 53 virions). Note that this conflicts with the approach taken in the increasingly-influential CAMRA website[27] ($\alpha = 1.06$ and $\beta = 171.3$), giving rise to an infection ID_{50} = 922. This has implications for the enterovirus concentrations to be presented to this dose-response function in the QMRA calculations.[28]

[23] This widely-quoted paper (Akin 1981) seems to have been read by only a few, given its appearance only in the "grey literature", decades past. The author of this report has a copy, courtesy of Professor Haas (Drexel University), which is available on request.

[24] For the simple exponential model, algebraic manipulation shows that $ID_{50} = -\ell n(½)/r \approx 0.693/r$.

[25] At page 864 of Schiff et al. (1984b): "The original plaque assay used for determination of the titre of the echovirus-12 pool and of the various challenge doses administered to volunteers was based on the use of LLC-MK_2 cells and an agar overlay procedure; in the present study this assay was shown to be significantly less sensitive than the plaque neutralization assay involving RD cells and a soft agar overlay procedure. The latter system increased the plaquing efficiency of the challenge virus by 33-fold."

[26] For the approximate beta-Poisson model, algebraic manipulation shows that $ID_{50} = \beta(2^{1/\alpha} - 1)$.

[27] Center for Advancing Microbial Risk Assessment Not http://qmrawiki.canr.msu.edu/index.php/Dose_Response

[28] The adopted dose-response function refers to echovirus 12 data gathered using the "LLC-MK_2" cell line (Schiff et al. 1984a). The CAMRA dose-response function refers to data re-analysed using "RD" cell line. Comparison of dose-response functions for other members of the enterovirus group (e.g., polio virus, hepatitis A, coxsackie) indicates that ID_{50} of the order of 50 is more tenable than of the order of 1000.

Appendix C Debate about norovirus infection dose-response

We have taken a form of norovirus infection dose-response that has become an "industry standard" in the last five years. It is based on a clinical trial, and is broadly supported by an outbreak study on French oysters (Thebault et al. 2013). That choice reflects a reasonable precautionary stance. Two recent contributors to the journal *Risk Analysis* have presented findings that norovirus may be even more infectious (Messner et al. 2014), or less infectious (Schmidt 2014) than the industry standard dose response, depending largely on the assumed degree of virus aggregation. There is currently much debate about all that. For example, another writer used data from a new clinical trial to claim that norovirus is much less infectious than the industry standard (Atmar et al. 2011, 2014) (this analysis appears to be flawed, as it ignored the role of aggregation, see McBride 2014a).

The role of noroviruses in QMRA will continue to be contentious, not least because a recently published procedure for their enumeration by culture (Jones et al. 2014) supplanted an earlier unsuccessful claim to such a procedure (Straub et al. 2007). This reflects the fact the QMRA is still an emerging discipline, with a number of issues that will take years to resolve. Nonetheless, experience indicates that QMRA is a more informative approach to human health risk assessment relative to that provided by levels of indicator bacteria derived from epidemiological studies at sites generally far-removed from the effects of discharges from large wastewater treatment plants.

Appendix D Recent microbiological water quality data

Results derived from recent recreational water quality monitoring. Note that the y-axis scale is variable.

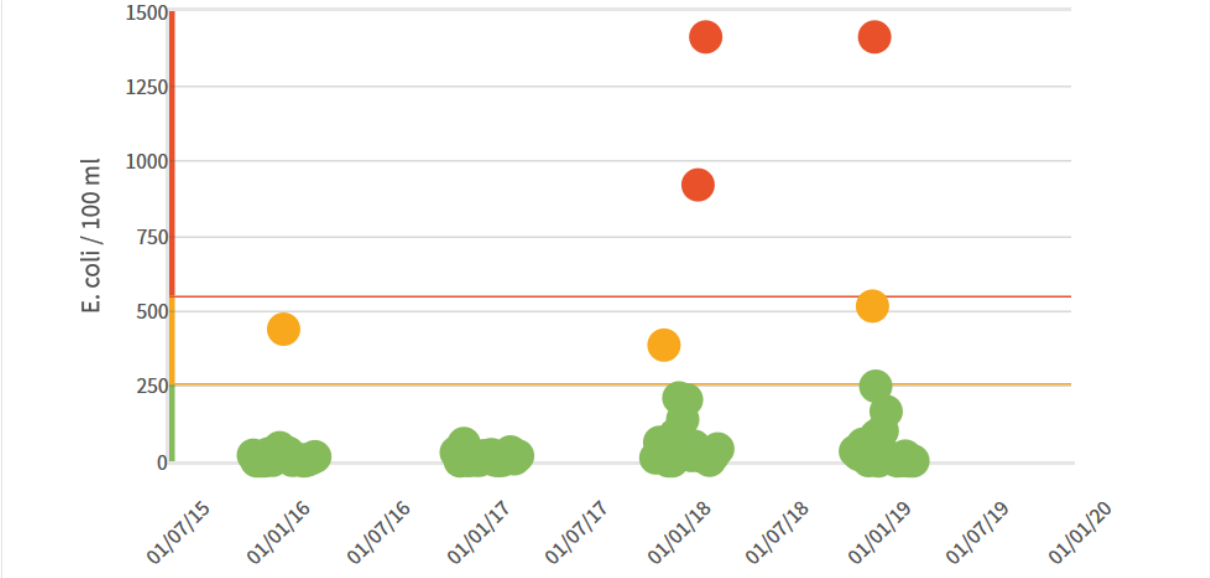

Figure D-1: Microbial water quality for the Lake Wakatipu at Frankton Bay monitoring site. The red and yellow lines define the thresholds between "not suitable for swimming" and "caution advised" (550 *E. coli*/100 mL) and "caution advised" and "suitable for swimming" (260 *E. coli*/100 mL), which are defined according to the 95th percentile value for each bathing season (this figure presents each data point). This figure was copied from the LAWA website https://www.lawa.org.nz/explore-data/otago-region/swimming/lake-wakatipu-at-frankton-bay/swimsite.

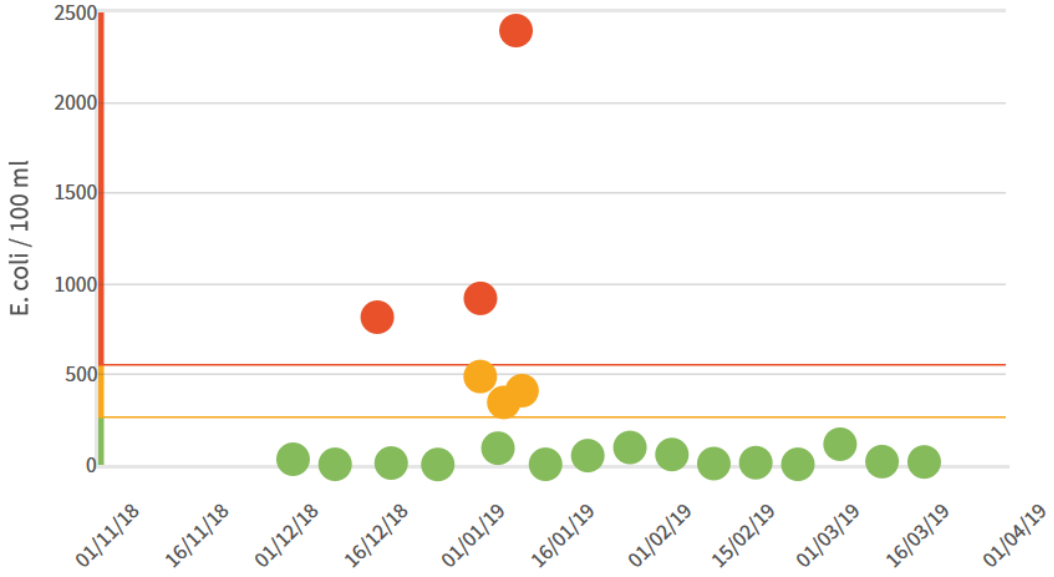

Figure D-2: Microbial water quality for the Lake Wakatipu at Queenstown Bay monitoring site. The red and yellow lines define the thresholds between "not suitable for swimming" and "caution advised" (550 *E. coli*/100 mL) and "caution advised" and "suitable for swimming" (260 *E. coli*/100 mL), which are defined according to the 95th percentile value for each bathing season (this figure presents each data point). This figure was copied from the LAWA website https://www.lawa.org.nz/explore-data/otago-region/swimming/lake-wakatipu-at-queenstown-bay-1/swimsite.

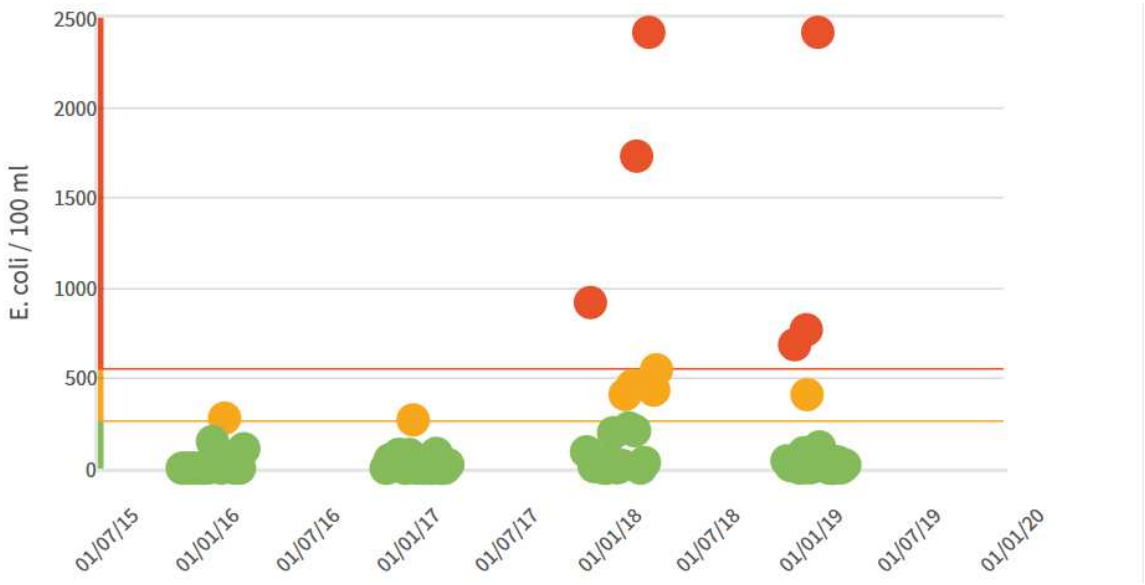

Figure D-3: Microbial water quality for the Lake Hayes at Mill Creek Shallows monitoring site. The red and yellow lines define the thresholds between "not suitable for swimming" and "caution advised" (550 *E. coli*/100 mL) and "caution advised" and "suitable for swimming" (260 *E. coli*/100 mL), which are defined according to the 95th percentile value for each bathing season (this figure presents each data point). This figure was copied from the LAWA website https://www.lawa.org.nz/explore-data/otago-region/swimming/lake-hayes-at-mill-creek-shallows/swimsite.

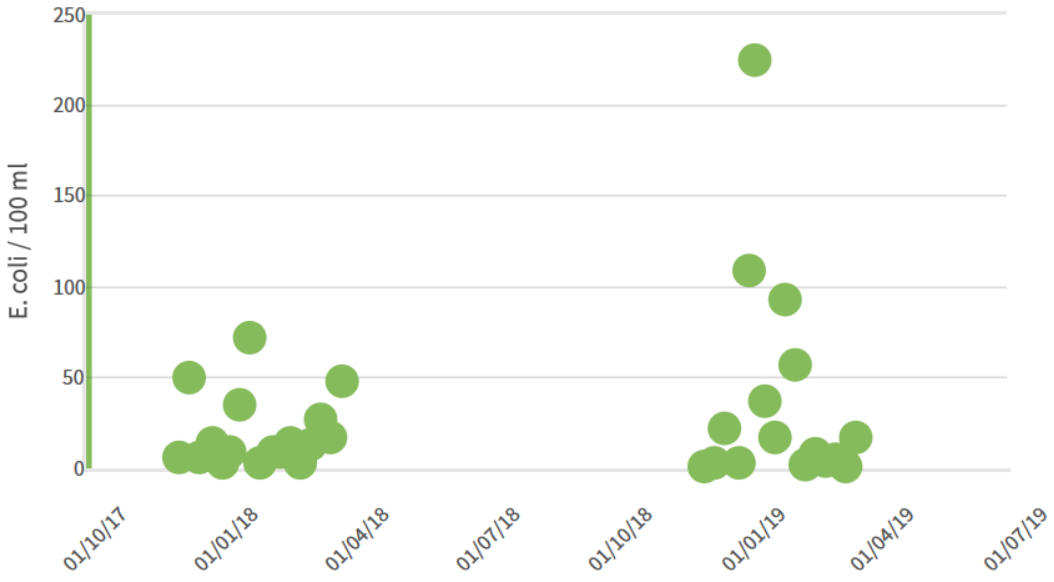

Figure D-4: Microbial water quality for the Lake Wanaka at Roy Bay monitoring site. All results indicate "suitable for swimming" (260 *E. coli*/100 mL) status, defined according to the 95th percentile value for each bathing season (this figure presents each data point). This figure was copied from the LAWA website https://www.lawa.org.nz/explore-data/otago-region/swimming/lake-wanaka-at-roys-bay-shore/swimsite.

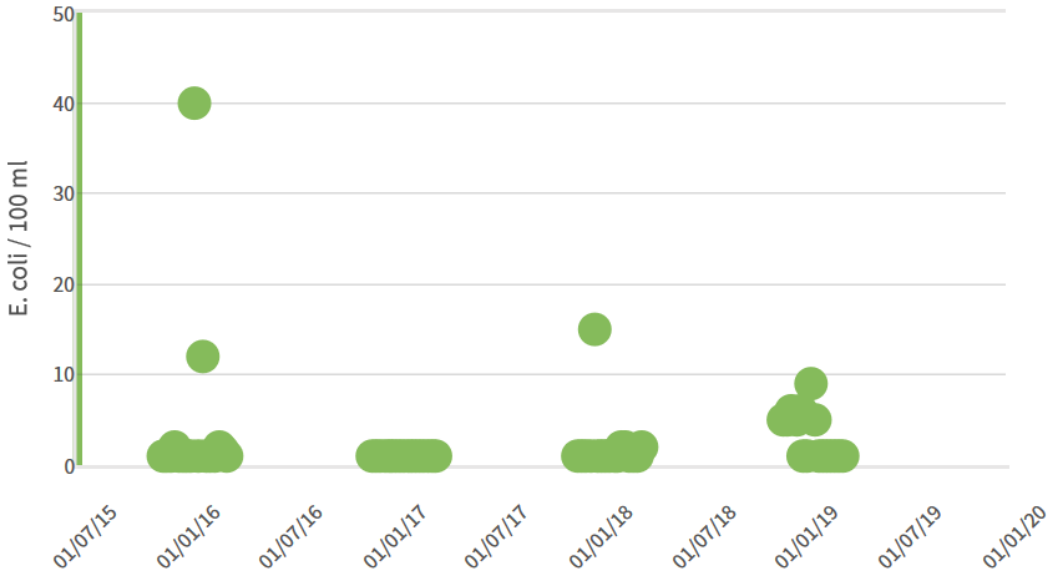

Figure D-5: Microbial water quality for the Lake Hawea at Holiday Park monitoring site. All results indicate "suitable for swimming" (260 *E. coli*/100 mL) status, defined according to the 95th percentile value for each bathing season (this figure presents each data point). This figure was copied from the LAWA website https://www.lawa.org.nz/explore-data/otago-region/swimming/lake-hawea-at-holiday-park/swimsite.