



Kunnskap for ei betre verd

Quantitative Microbial Risk Assessment for Onsite Sanitation Systems

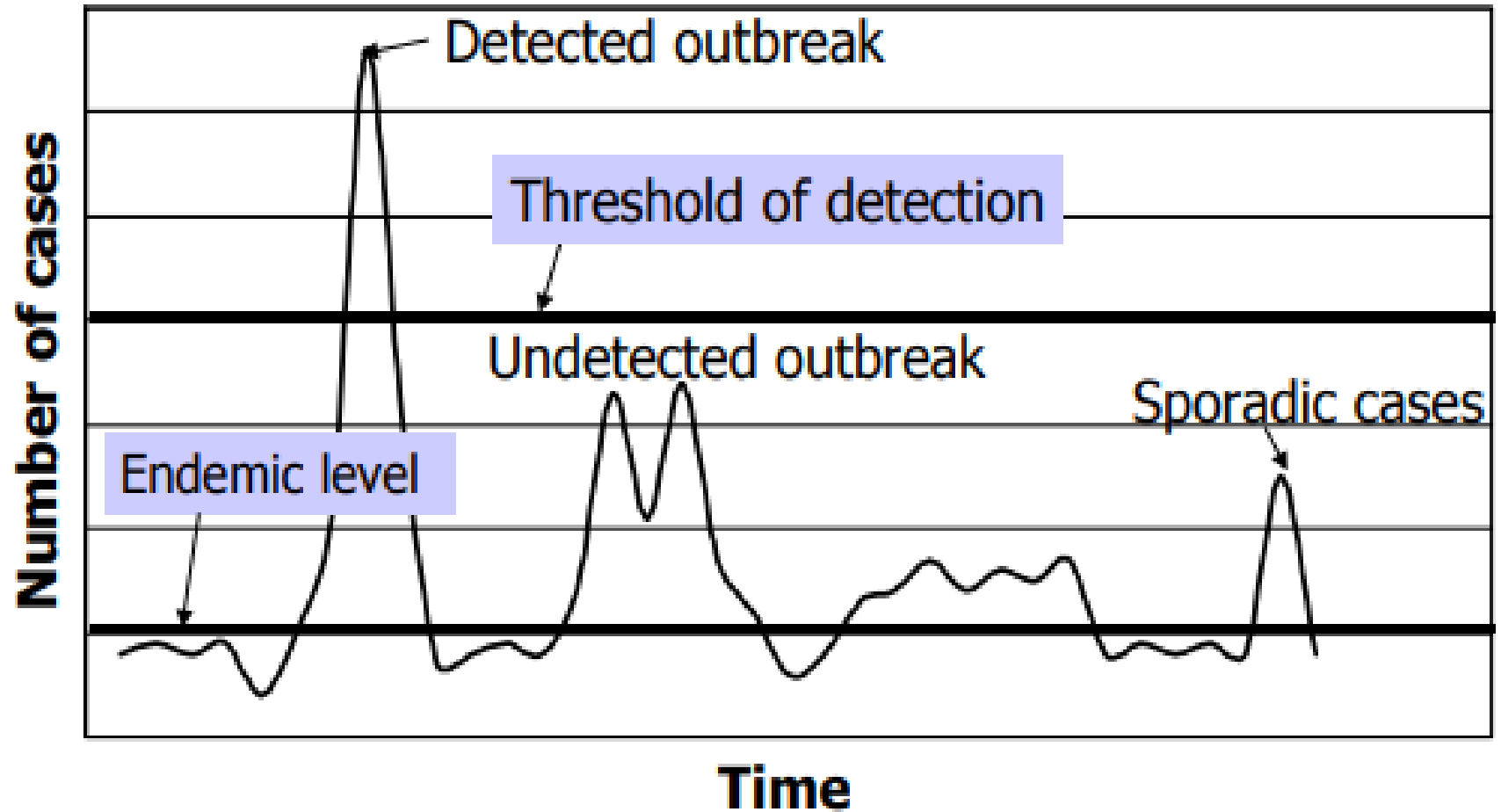
All4WASH Summer School 2024

Razak Seidu

Content

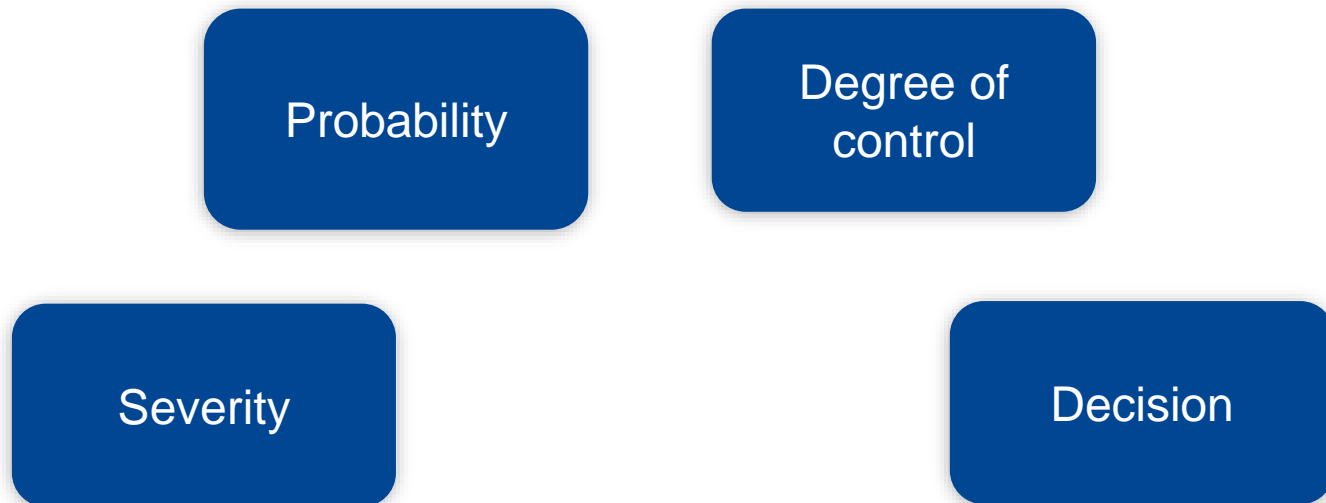
- Epidemiology and sanitation
- Sanitation Safety Planning Framework
- Quantitative Microbial Risk Assessment
- Exercise
- Data

Epidemiology....



What is Risk?

- In microbial risk assessment, risk is usually defined as having two dimensions:
 - the probability that something unpleasant happens: the probability of an adverse outcome, and
 - the severity of that outcome.



What is Risk?

Risk may be calculated as expected loss

For any possible outcome X_k there is a loss C_k while the probability that any outcome X_k occurs is p_k . Therefore, the risk R is

$$R = \sum_K C_k p_k$$

For a continuous outcome x with probability density $f(x)$ and loss function $c(x)$, the risk can be calculated as:

$$R = \int_x c(x) f(x) dx$$

Risk Analysis

Risk Analysis

Risk Assessment

Risk assessment is the qualitative or quantitative characterization and estimation of potential adverse health effects associated with exposure of individuals or populations to hazards (materials or situations, physical, chemical, and/or microbial agents).

Risk Management

The process for controlling risks, weighing alternatives, and selecting appropriate action, considering risk assessment, values, engineering, economics, and legal and political issues

Risk Communication

The communication of risks to managers, stakeholders, public officials, and the public, includes public perception and ability to exchange scientific information

Sanitation Safety Planning



QMRA Conceptual Framework

For onsite sanitation, risk assessment is an integral part of developing and implementing **Sanitation Safety Plans**.

The purpose of the risk assessment is

- to identify and evaluate the health risks associated with the sanitation service chain,
- to determine if the health hazards are adequately controlled,
- to inform operation and management of the sanitation system and
- to identify necessary improvements and upgrades to ensure the provision of safe sanitation.

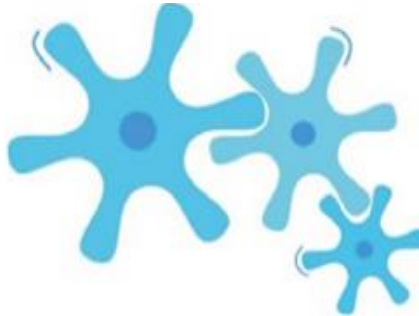
Quantitative Microbial Risk Assessment

Quantitative Scientific data

- Pathogen occurrence
- Pathogen persistence
- Barrier efficiency
- Exposure
- Infectivity
- Individual susceptibility
- Disease impact



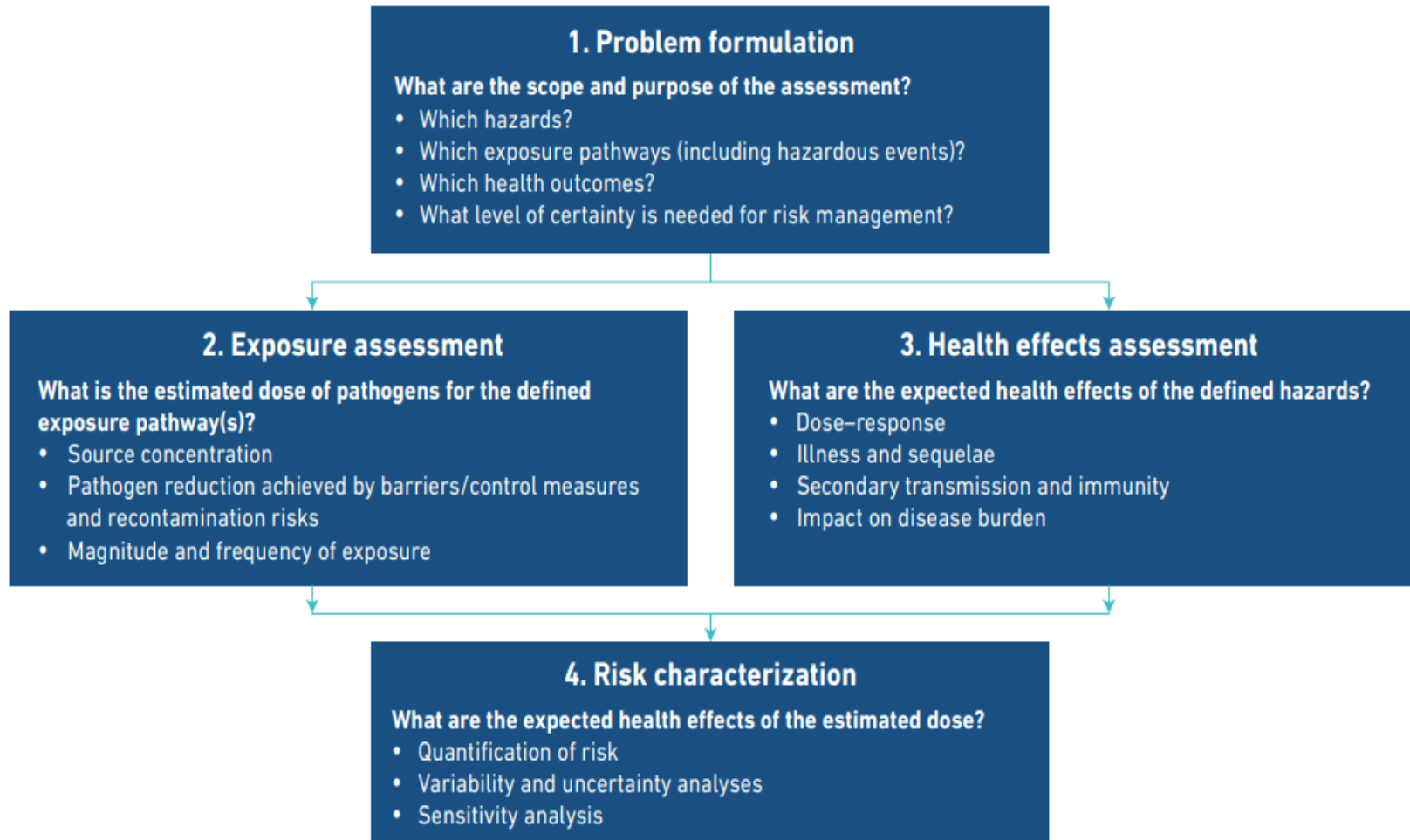
QMRA



Sanitation Safety Planning

- Improved system understanding
 - Risk drivers
 - Information needs
- Decision support
 - Critical limits
 - Prioritization
- Regulatory targets

QMRA Framework- Sanitation Systems



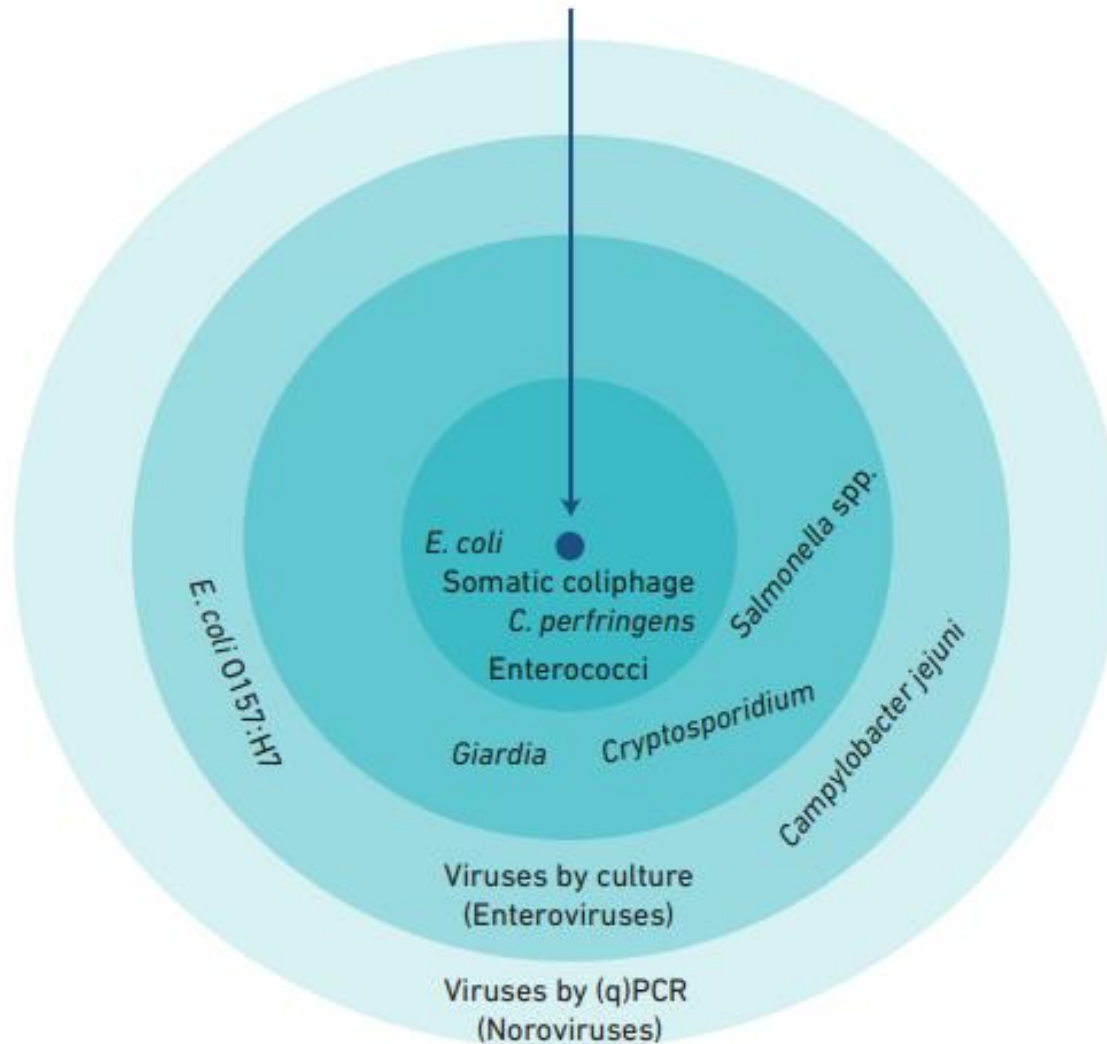
Problem Formulation- Which microbial hazard is of concern from the user interface to reuse/disposal?



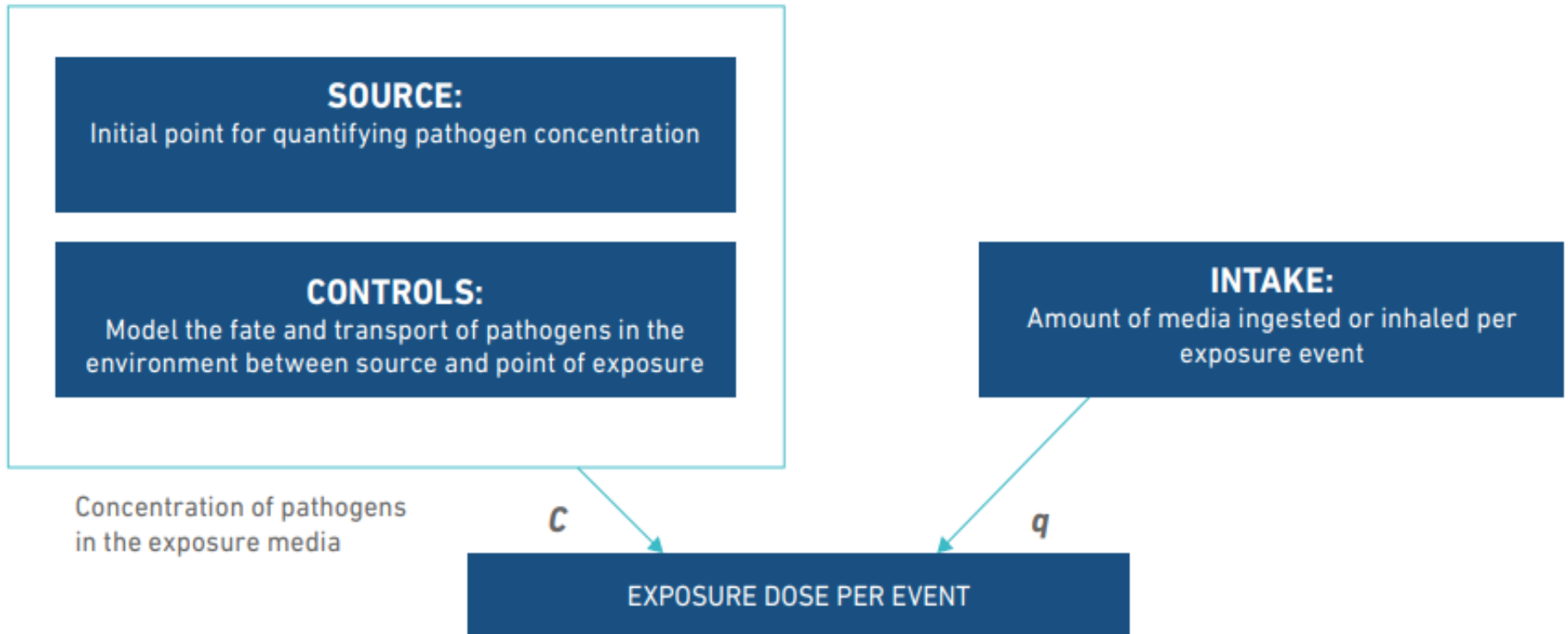
Group	Pathogen	Disease symptoms
Bacteria	<i>Aeromonas</i> spp.	Enteritis
	<i>Campylobacter jejuni/coli</i>	Campylobacteriosis – diarrhea, cramping, abdominal pain, fever, nausea, arthritis, Guillain-Barré syndrome
	<i>Escherichia coli</i> (EIEC, EPEC, ETEC, EHEC)	Enteritis. For EHEC there are also internal hemorrhages
	<i>Salmonella typhi/paratyphi</i>	Typhoid, headache, fever, malaise, anorexia, cough
	<i>Salmonella</i> spp	Salmonellosis – diarrhea, and abdominal cramps
	<i>Shigella</i> spp. <i>Vibrio cholera</i>	Shigellosis – dysentery (bloody diarrhea), vomiting, cramps, fever; Reiters syndrome Cholera – watery diarrhea
Virus	Adenovirus	Various; respiratory illness, here added due to enteric types
	Enteric adenovirus types 40 and 41	Enteritis
	Enterovirus types 68–71	Meningitis; encephalitis; paralysis
	Hepatitis A	Hepatitis – fever, malaise, anorexia, abdominal discomfort, jaundice
	Hepatitis E	Hepatitis
	Poliovirus	Poliomyelitis – fever, nausea, vomiting, headache, paralysis
	Rotavirus	Enteritis
Parasitic protozoa	<i>Cryptosporidium parvum</i>	Cryptosporidiosis – watery diarrhea, abdominal cramps and pain
	<i>Cyclospora histolytica</i>	Often asymptomatic; diarrhea; abdominal pain
	<i>Entamoeba histolytica</i>	Amoebiasis – often asymptomatic, dysentery, abdominal discomfort, fever, chills
	<i>Giardia intestinalis</i>	Giardiasis – diarrhea, abdominal cramps, malaise, weight loss
Helminths	<i>Ascaris lumbricoides</i>	No or few symptoms; wheezing; coughing; enteritis; pulmonary eosinophilia
	<i>Taenia solium/saginata</i>	Taeniasis
	<i>Trichuris trichura</i>	Trichuriasis – Unapparent through to vague digestive tract distress to emaciation with dry skin and diarrhea
	Hookworm <i>Schistosoma</i> spp. (blood fluke)	Itch; rash; cough; anemia; protein deficiency Schistosomiasis, bilharzias

Pathogen Concentration (C)- Precision

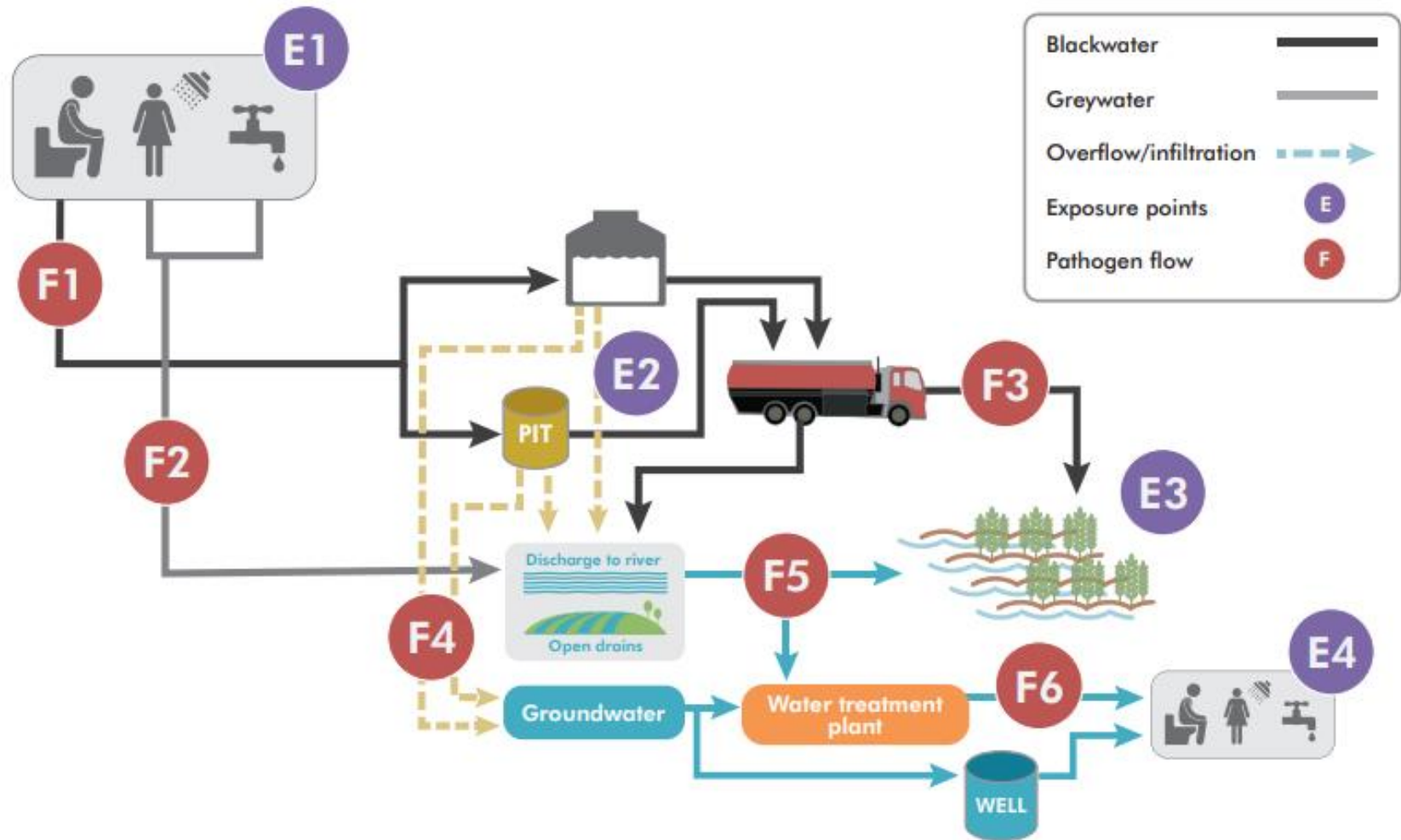
Target: What was the true concentration in the environmental sample?



Exposure Assessment Framework



Exposure Assessment (HAACP)



E1: Users and cleaners of toilet; **E2:** Ingestion of wastewater (workers); **E3:** Ingestion of sludge and consumption of crops (workers and consumers); **E4:** Consumption of contaminated surface and groundwater

Exposure Assessment

Exposure assessment simply involves **estimating the ingested dose of a pathogen resulting from exposure to water, vegetables, sludge, aerosol etc.** A generic model for exposure assessment may be formulated as:

$$D = C \times 1/Se \times Sp \times Z_{1,\dots,m} \times Y_{1,\dots,n} \times V$$

Where:

- D ingested dose of a pathogen.
- C concentration of the pathogen at the starting point of exposure.
- Se sensitivity of pathogen enumeration method.
- Sp specificity of the enumeration method.
- Z represents n consecutive treatments.
- Y represents pathogen regrowth
- V ingested amount of material containing pathogen (e.g. water, food, aerosol etc)

Pathogen dieoff kinetics

Log-linear decay model (Chick 1908):

$$\frac{C_t}{C_0} = e^{-kt}$$

$$C_t = C_0 e^{-kt}$$

Where:

C_t is the concentration at time t ,
 C_0 is the concentration at time zero
 k_1 and k_2 decay coefficients

The JM2 model is reported as the best model for pathogen survival in sewage, sludge, biosolids, and manure (Mitchell and Akram, 2019):

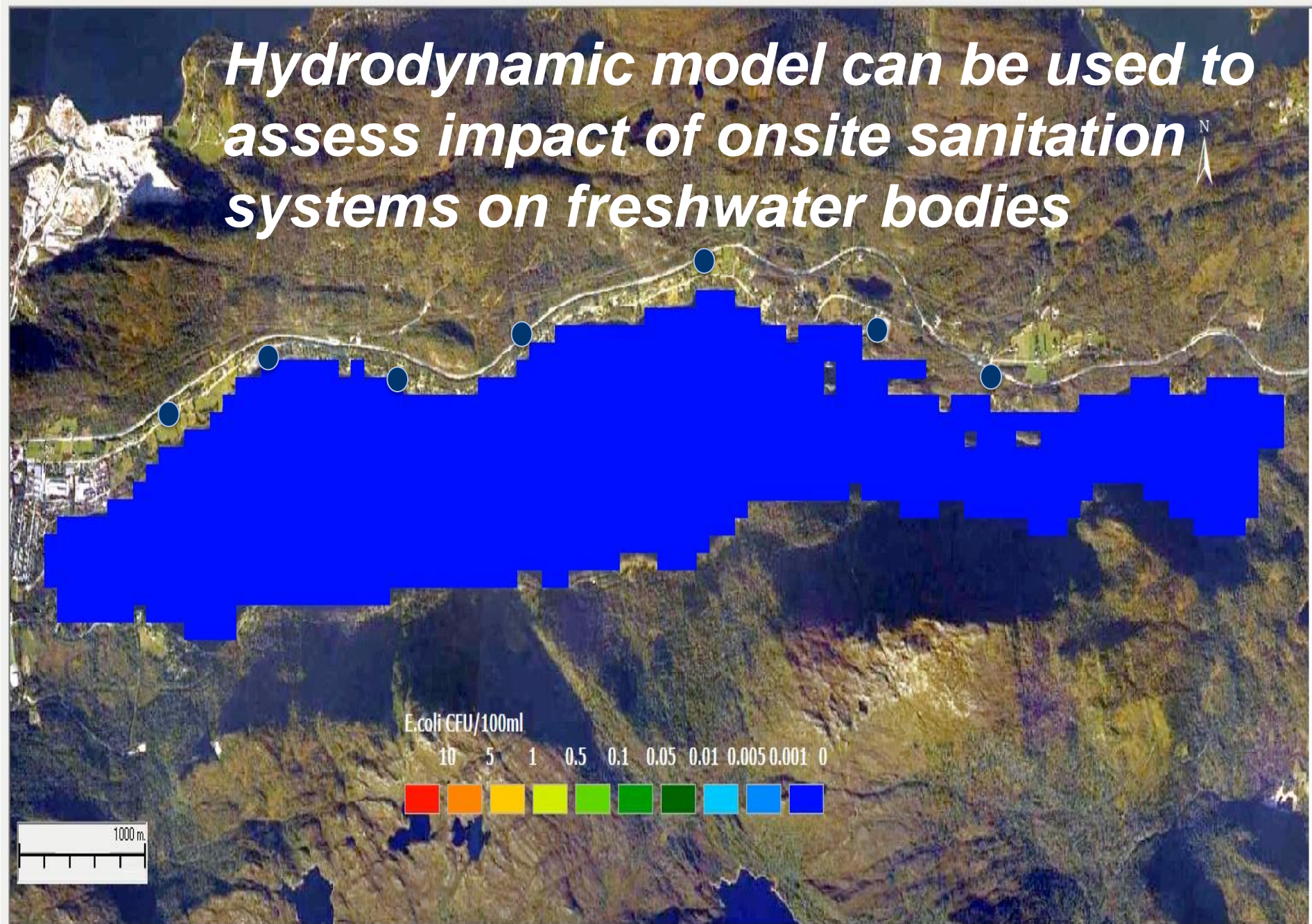
$$\frac{C_t}{C_0} = \frac{1}{1 + e^{k_1 + k_2 \ln(t)}}$$

$$C_t = C_0 \times \frac{1}{1 + e^{k_1 + k_2 \ln(t)}}$$

Where:

C_t is the concentration at time t ,
 C_0 is the concentration at time zero
 k_1 and k_2 decay coefficients

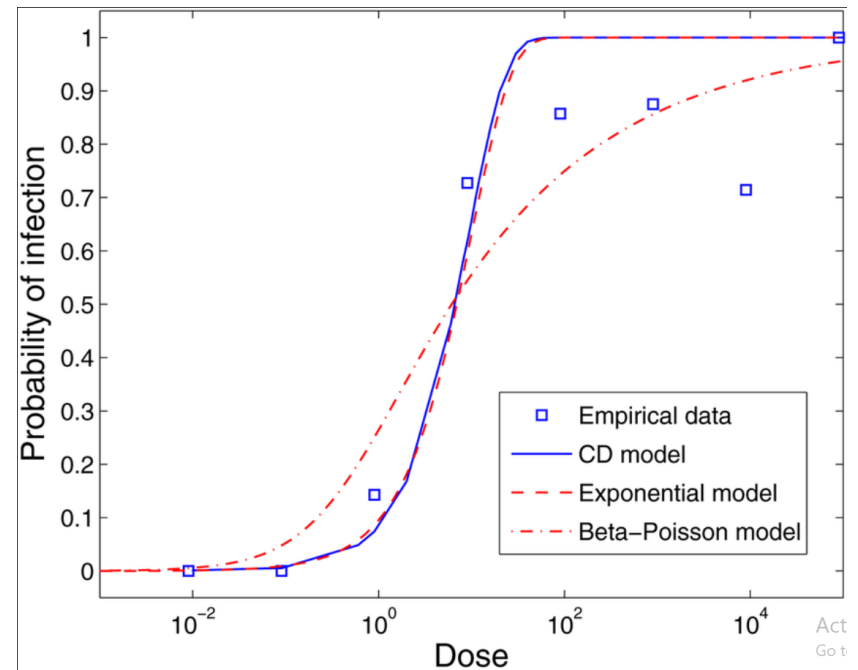
Hydrodynamic model can be used to assess impact of onsite sanitation systems on freshwater bodies



Dose-Response Assessment

A dose-response model is a mathematical function of the relationship between the dose of an agent (pathogen) to which a person is exposed, and the probability of an adverse health effect (infection or illness) bounded by zero (no effect) and one (absolute adverse effect)

The most applied models within QMRA are based on **the single-hit theory**: where every ingested pathogen particle is assumed to act independently and has an individual probability of causing infection.



Single Hit Dose- Response Models

Exponential Model

If the single-hit probability of infection P_I (probability that any single ingested pathogen succeeds in infecting the host) is r , then the dose– response model for infection associated with ingesting a dose (d) of pathogens is

$$P_I(d) = 1 - \exp(-rd)$$

Exact Beta-Poisson Model

If r is variable and its variability is described by a beta distribution with parameters (α, β) , then: Provided that $\alpha \ll \beta$ and $\beta \gg 1$. ${}_1F_1$ is a confluent hypergeometric function (Kummer function).

$$P_I(d) = 1 - F_1(\alpha, \alpha + \beta; -d)$$

Beta-Poisson Model -Approximation

The approximation becomes poorer at small values of β or large values of d (and risk). N_{50} is the ID50, the 50% infectious dose.

$$P_I(d) = 1 - \left[1 + \frac{d}{N_{50}} (2^{1/\alpha} - 1) \right]^{-\alpha}$$

$$P_I(d) = 1 - \left(1 + \frac{d}{\beta} \right)^{-\alpha}$$

Dose-Response Studies

	Parameters	Comments	Reference
<i>Campylobacter jejuni</i>	$a = 0.145, \beta = 7.59$	H.f.t. ^a by Black <i>et al.</i> 1988	Medema <i>et al.</i> , 1996
<i>Salmonella spp.</i> (non-typhi)	$a = 0.3126,$ $N_{50}^b = 23\ 600$	H.f.t. by McCullough and Wesley Eisele 1951a, 1951b, 1951c. Several strains	Haas <i>et al.</i> , 1999
<i>E. coli</i> O157:H7	$a = 0.2099,$ $N_{50}^b = 1\ 120$	Based on h.f.t. on <i>Shigella</i> by DuPont <i>et al.</i> (1969, 1972) and Levine <i>et al.</i> (1973)	Crockett <i>et al.</i> , 1996
Hepatitis A	$a = 0.49,$ $N_{50}^b = 5.96 \times 10^5$ $a = 0.2, N_{50}^b = 30$	Rabbit study by Pai <i>et al.</i> 1986 Assumption by Shuval <i>et al.</i> , 1997	Haas <i>et al.</i> , 2000 Shuval <i>et al.</i> , 1997
Rotavirus	$a = 0.253, \beta = 0.422$	H.f.t. by Ward <i>et al.</i> 1986	Teunis <i>et al.</i> 1996
Adenovirus 4	$k^c = 2.397$	H.f.t. by Couch <i>et al.</i> 1966	Haas <i>et al.</i> , 1999
<i>Giardia lamblia</i>	$r = 0.0199$	H.f.t. by Rendtorff 1954	Teunis <i>et al.</i> 1996
<i>Cryptosporidium parvum</i>	$k^c = 238.6$	H.f.t. by DuPont <i>et al.</i> 1995	Haas <i>et al.</i> , 1996
<i>Ascaris</i>	$r = 1$	None available	Assumption

^a H.f.t. = human feeding trials. ^b $\beta = N_{50}(2^{1/\alpha}-1)$. ^c $r = 1/k$.

For *Ascaris*, $N_{50} = 859$ and $r = 0.104$ respectively (Navarro *et al.*, 2009).

Probability of Infection: Multiple Exposure Events

- Dose–response models typically estimate the probability of infection (or, in some cases, illness) associated with a single-exposure event.
- To consider multiple events over a longer time frame, it is necessary to combine the individual probability using the following equation:

$$P_{inf} = 1 - (1 - P_{inf/single})^N$$

$P_{inf/combined}$ = probability of one or more infections over N events

$P_{inf/single}$ = single-event probability of infection

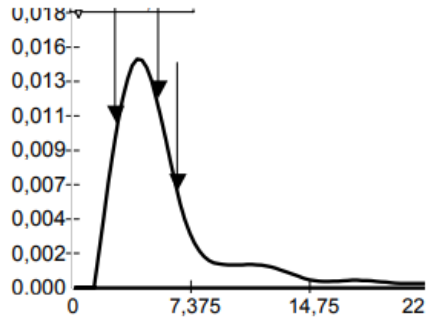
Probability of Infection: Multiple Exposure Events

- When combining infection probabilities associated with different event conditions, the following equation is used:

$$P_{inf/combined} = 1 - \prod_{i=1}^m (1 - P_{inf/i})^{N_i}$$

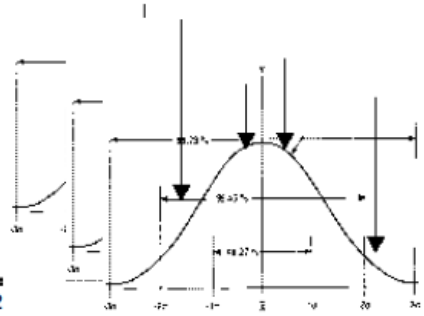
where $P_{inf/i}$ is the probability of infection associated with event i (of a total of m events to be considered in the analysis), which occurs N_i times over the period for which the combined risk $P_{inf/combined}$ is calculated.

Point (Deterministic) vs stochastic estimates

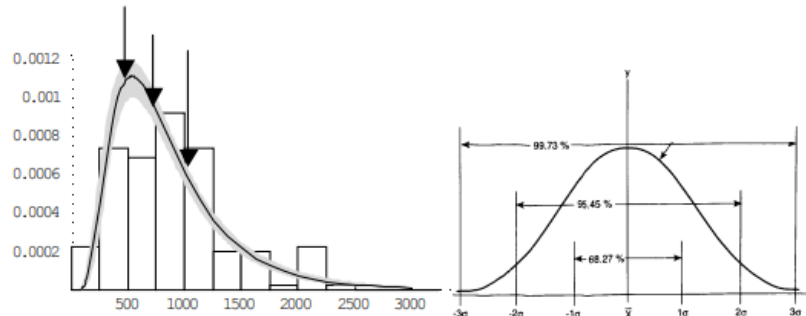


Source water

x



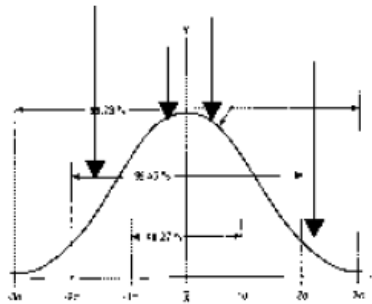
Reduction during treatment



Exposure

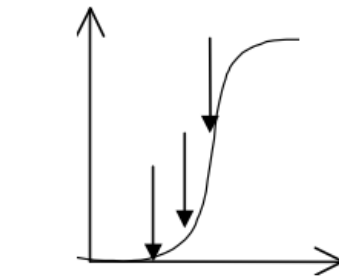
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Dose



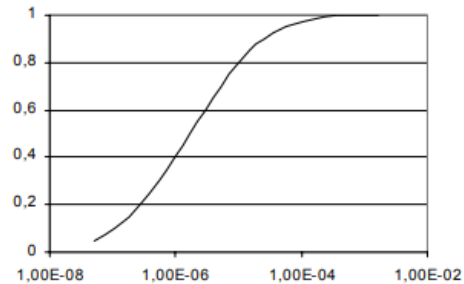
Dose

x



Dose-response equation

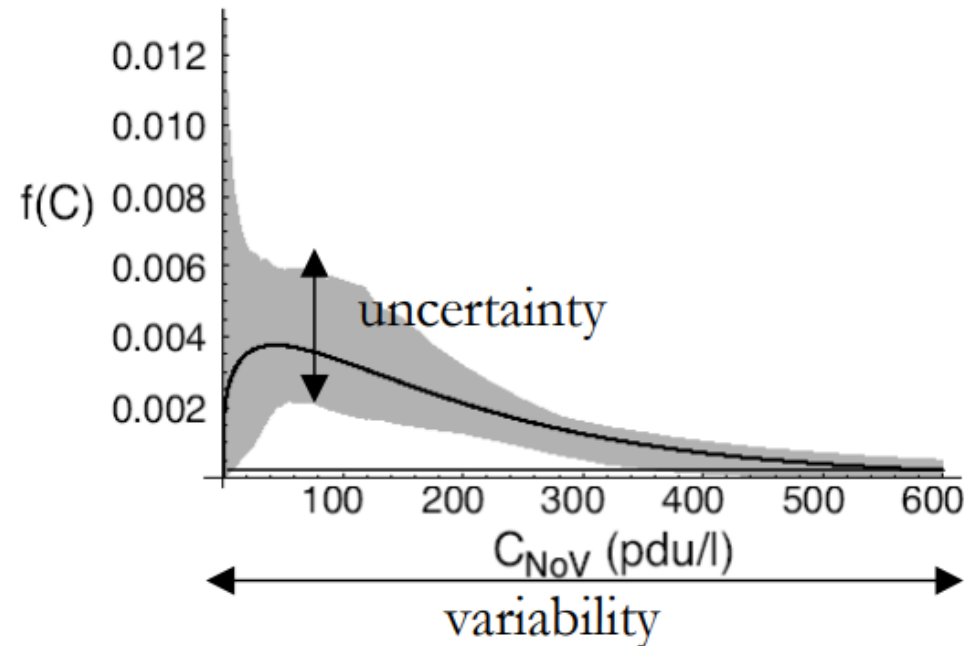
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Risk of infection

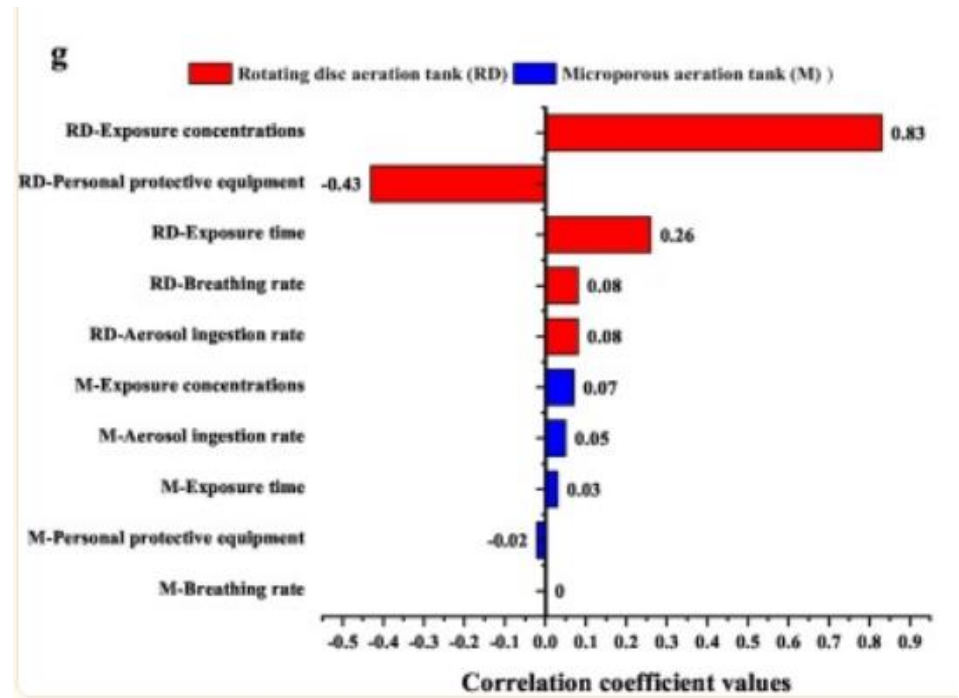
Variability and Uncertainty

- Variability is the inherent variation in the data, which cannot be reduced.
 - Spatial
 - Temporal variability
 - Inter-individual variability
- The uncertainty reflects flaws in the data collection and can accordingly be reduced by increased investigations.



Sensitivity Analysis

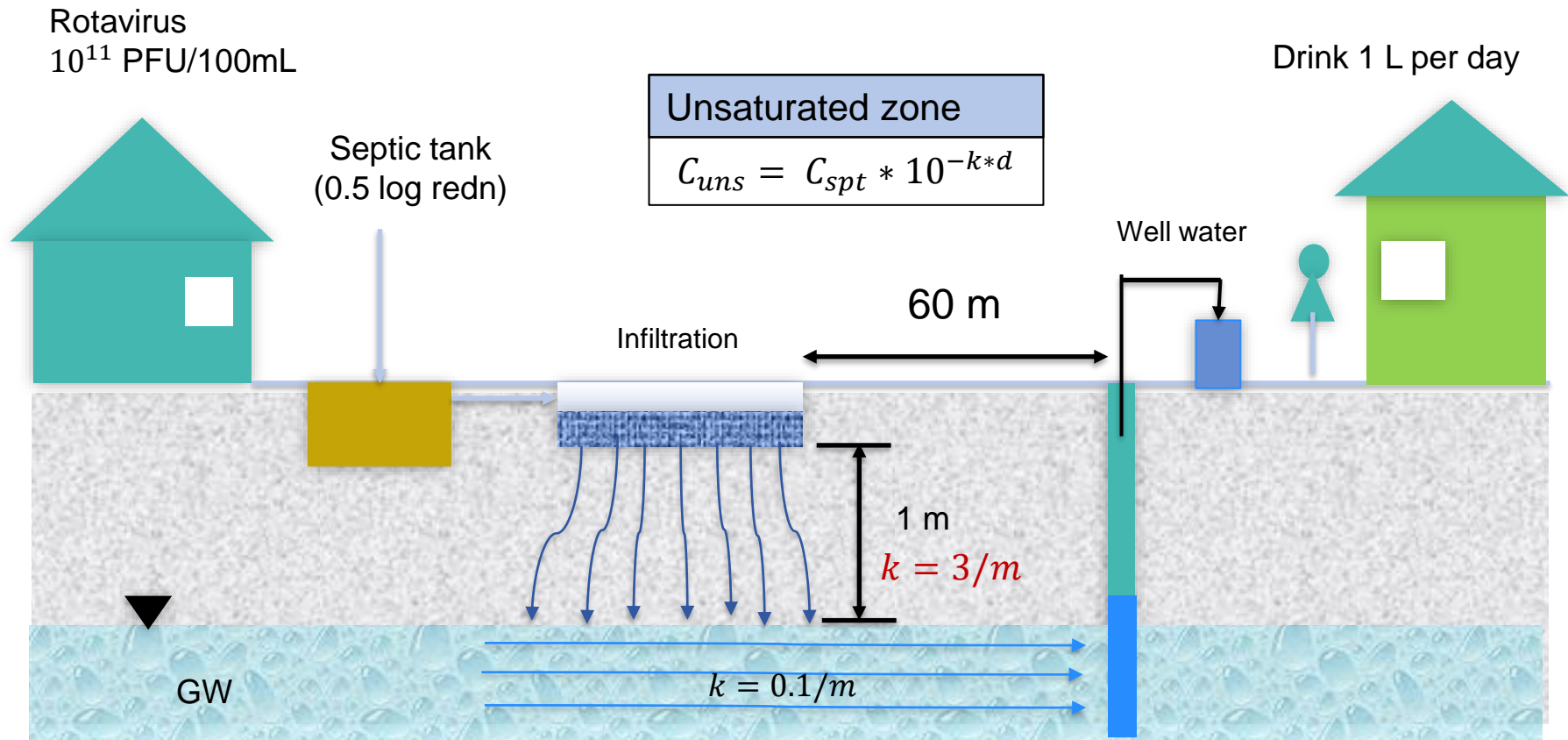
- The input variables in a QMRA have large statistical variability and uncertainties, while the quantitative effect of various phenomena is unknown.
- With sensitivity analysis the effects of the input variables on the output risk can be assessed.



Tolerable Infection Risks

- In order to decide a baseline for the infection risks that can be tolerated from a societal point of view there is a need to set up an '**acceptable**' or '**tolerable risk**' level.
- In the U.S. the Environmental Protection Agency (USEPA) have accepted a yearly risk of 1 infected person in 10 000 from drinking water, which is often expressed as **10^{-4} per person per year**.
- This is approximately equivalent to a lifetime excess cancer risk of 10^{-5} (i.e. one excess case of cancer per 100 000 of the population ingesting drinking water containing the substance at the guideline value over a lifespan).

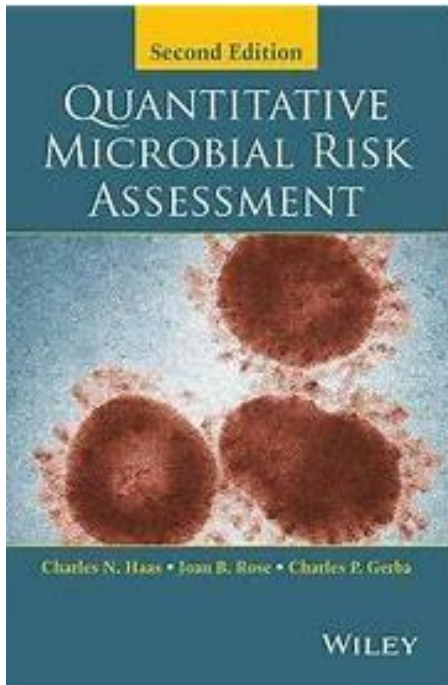
Exercise: is the distance between the infiltration unit and well OK for rotavirus infection ?



Unsaturated zone
$C_{uns} = C_{spt} * 10^{-k*d}$

Saturated zone
$C_{GW} = C_{uns} * 10^{-k*d}$

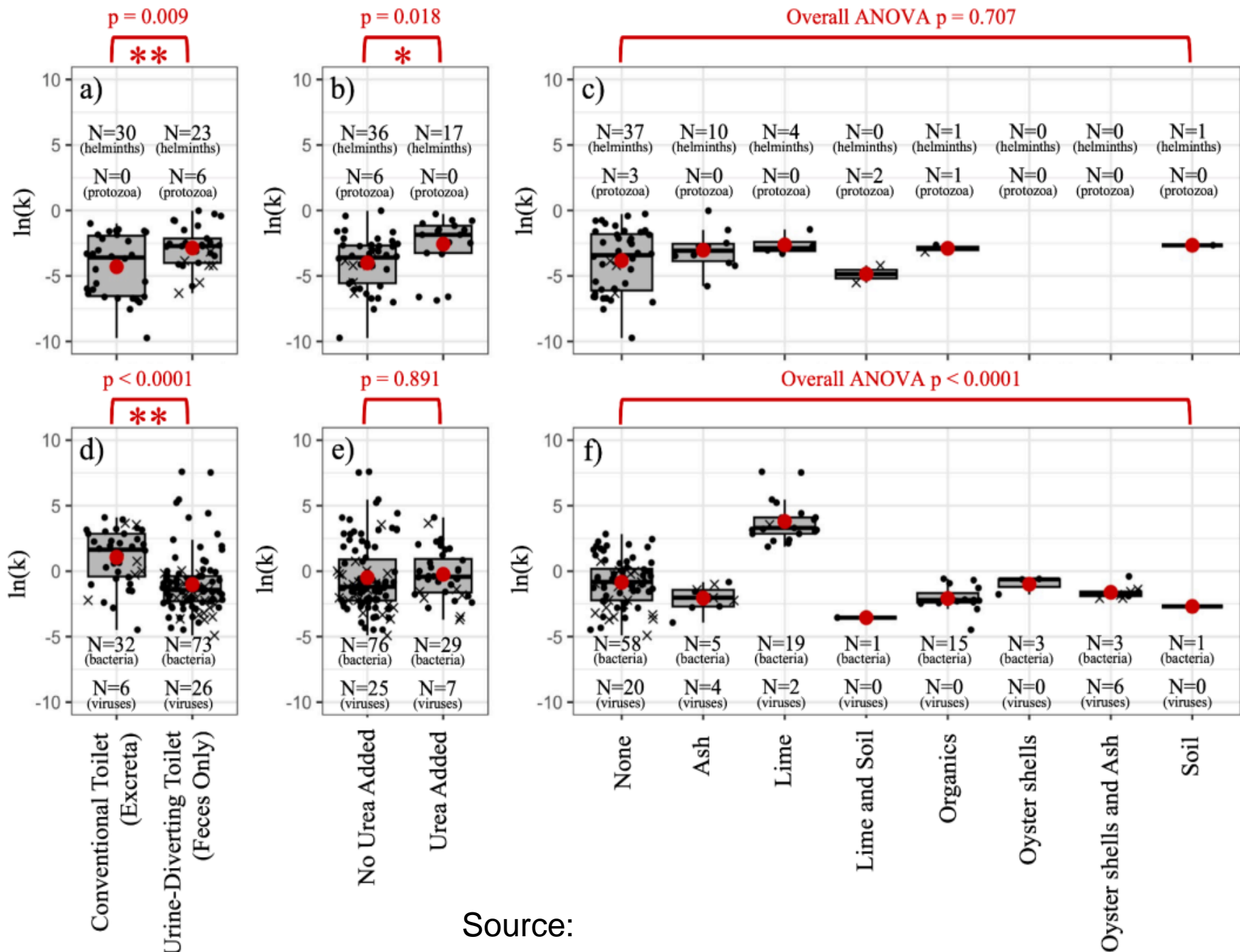
Resources



 **SANITATION, WASTEWATER MANAGEMENT AND SUSTAINABILITY**
FROM WASTE DISPOSAL TO RESOURCE RECOVERY

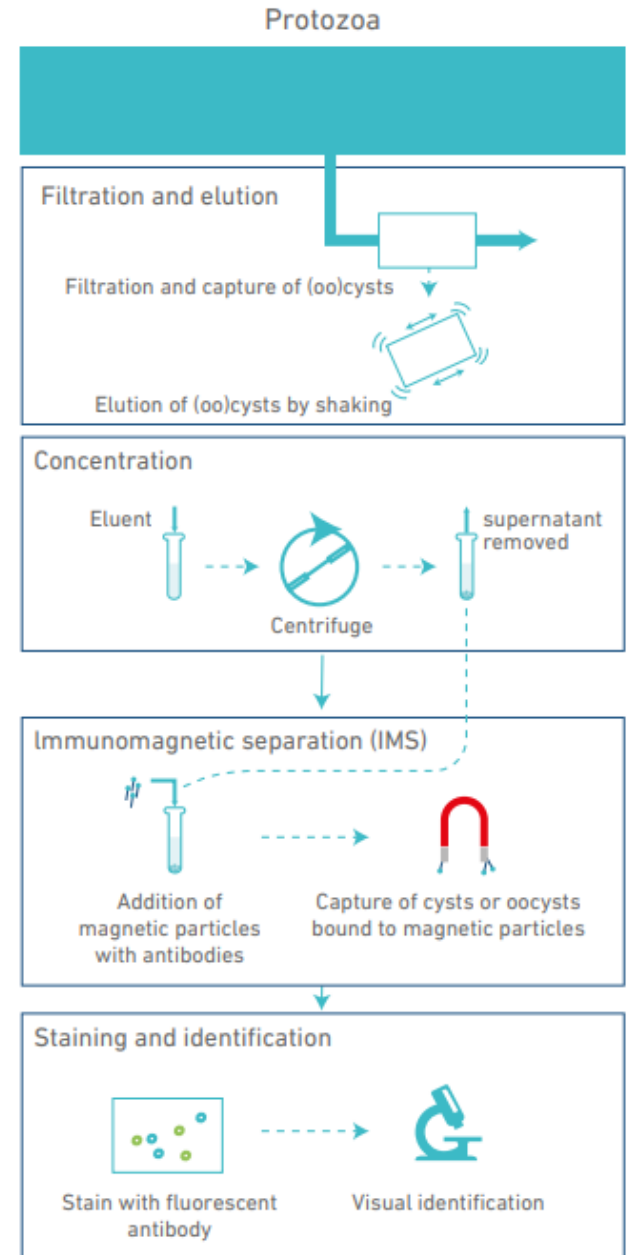
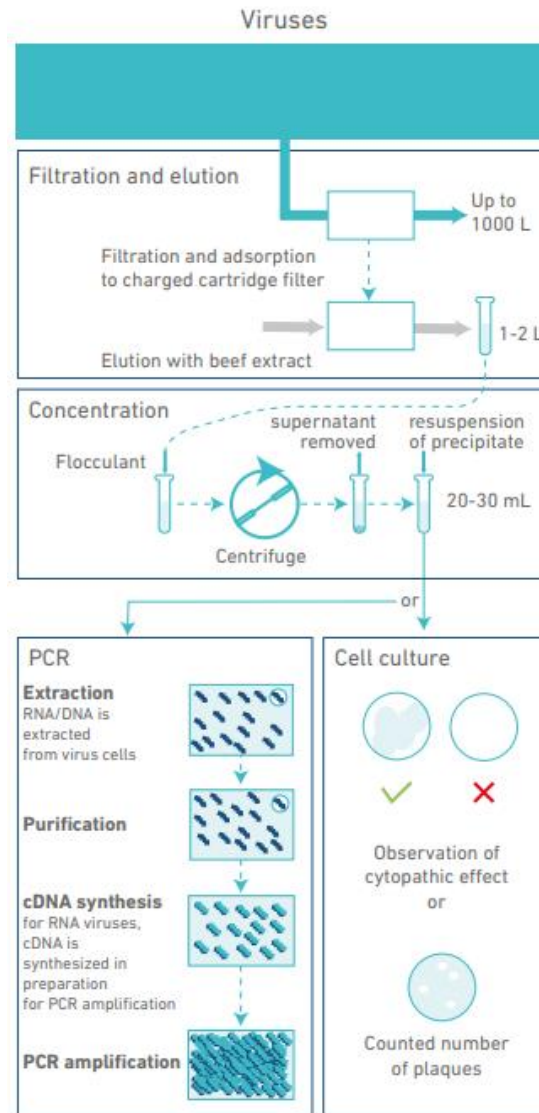
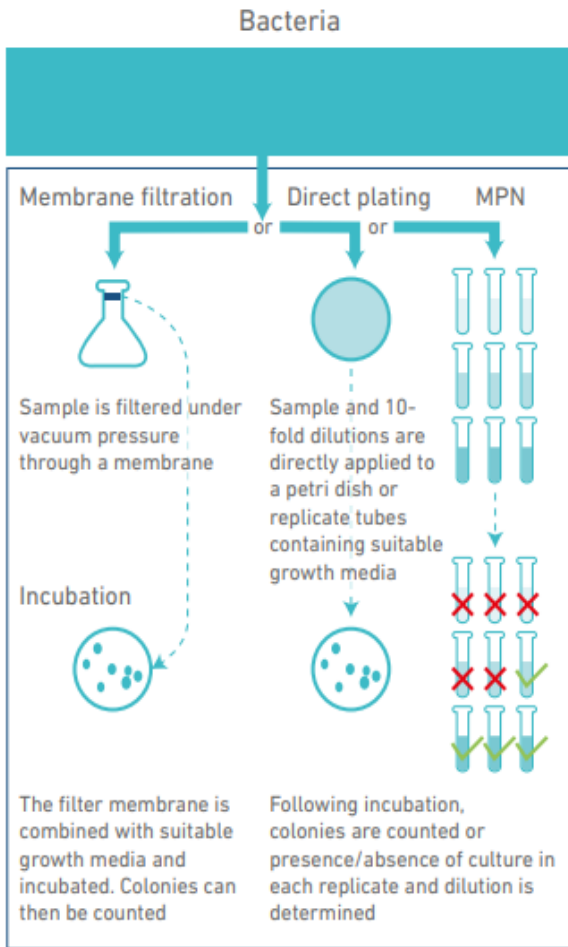


Data



Pathogen Concentration (C)- Microbial Enumeration Methods

Multiple steps for concentration and purification



Pathogen Concentration (C)- Quantitative or Qualitative

Although results are often reported as a concentration, because microorganisms are discrete units, the concentration cannot be measured directly and is an inference based on one of three types of observations:

1. Quantitative

Quantitative (counted number of organisms, colonies or plaques), where the precision for predicting the original concentration depends on the number of organisms counted and the volume (including dilution) of the sample (e.g. a count of 2 in 100 mL is less precise than a count of 200 in 10 L)

2. Qualitative

Qualitative (presence or absence of an observed response in a sample volume). Quantitative concentrations are frequently reported from a series of qualitative results as the MPN.

3. Semiquantitative

Semiquantitative. Quantitative PCR (qPCR) uses the rate of amplification of the target genetic material in the sample to predict the original concentration of genomes or genome equivalents in the environmental sample. In QMRA, This is considered semi-quantitative as it is uncertain. the translation of the qPCR result back to an absolute concentration of infectious pathogenic units in the original sample is uncertain

Pathogen Concentration (C)- Statistical Inference

Often implicit and not directly considered in quantifying pathogen concentration

Sample processing

What volume of original sample is represented?
What opportunities exist for microbial loss during processing and enumeration?

Observations

What was observed?
How many observations per sample?

STATISTICAL INFERENCE

Reported concentrations

e.g. MPN, CFU, PFU, PDU, MPNCU, Genome copies, etc.
The uncertainty in the reported concentrations depends upon the sample processing and the observations (number and nature)

STATISTICAL INFERENCE

Quantified concentration of reference pathogen for QMRA

What is the uncertainty in the quantified concentration for QMRA given the observations?

Approach 1: Quantified concentration based on reported concentrations

Sample processing

What volume of original sample is represented?
What opportunities exist for microbial loss during processing and enumeration?

Observations

What was observed?
How many observations per sample?

STATISTICAL INFERENCE

Quantified concentration of reference pathogen for QMRA

What is the uncertainty in the quantified concentration for QMRA given the observations?

Approach 2: Quantified concentration based on the sample processing and observations

Pathogen concentration in faeces

	Incidence ^a (per 100 000)	Under-reporting	Morbidity (%)	Excretion ^b (g ⁻¹ faeces)	Duration ^b (days)	ID ₅₀ ^c
<i>Salmonella</i>	42-58	3.2 ^d	6-80 ^e	10 ⁴⁻⁸	26-51	23 600
<i>Campylobacter</i>	78-97	7.6 ^d	25 ^f	10 ⁶⁻⁹ g	1-77 ^h	900
EHEC	0.8-1.4	4.5-8.3 ⁱ	76-89 ⁱ	10 ²⁻³	5-12	1 120
Hepatitis A	0.8-7.8	3 ^k	70 ^m	10 ⁴⁻⁶	13-30	30
Rotavirus	21 ^d	35 ^d	50 ⁿ	10 ⁷⁻¹¹	1-39	6
Norovirus	1.2 ^d	1562 ^d	70 ^o	10 ⁵⁻⁹ p	5-22 ^q	10?
Adenovirus	300 ^d	-	54 ^r	10 ¹¹ s	1-14 ^r	1.7
<i>Cryptosporidium</i>	0.3-1.6	4-19 ^t	39 ^e	10 ⁷⁻⁸	2-30	165
<i>Giardia</i>	15-26	20 ^k	20-40 ^u	10 ⁵⁻⁸	28-284	35
<i>Ascaris</i>	15-25 ^v	-	15 ^g	10 ⁴	107-557	0.7

Concentration of pathogens found in sludge

Type	Organism	Density in primary sludges (/g of dry wt)	Density in secondary sludges (/g of dry wt)
Virus	Various enteric viruses	$10^2 - 10^4$	3×10^2
	Bacteriophages	10^5	-
Bacteria	Total coliforms	$10^8 - 10^9$	7×10^8
	Faecal coliforms	$10^7 - 10^8$	8×10^6
	<i>Enterococci</i>	$10^6 - 10^7$	2×10^2
	<i>Salmonella</i> spp	$10^2 - 10^3$	9×10^2
	<i>Clostridium</i> spp	10^6	-
	<i>Mycobacterium Tuberculosis</i>	10^6	-
Protozoa	<i>Giardia</i> spp	$10^2 - 10^3$	$10^2 - 10^3$
Helminths	<i>Ascaris</i> spp	$10^2 - 10^3$	10^3
	<i>Trichuris vulpis</i>	10^2	$< 10^2$
	<i>Toxocara</i> spp	$10 - 10^2$	3×10^2

Concentration of pathogens found in wastewater

	Mean	Range	Pos. ^a	Place	Reference
<i>Campylobacter</i>	160 000	500-4 400 000	100%	Germany	Höller, 1988
<i>Salmonella</i>	Det. in 1 mL	n.a.	41%	Sweden	Carlander and Stenström, 2001
EHEC	22 000 Det.	930-110 000 n.a.	100% 93% ^b	Finland Germany	Koivunen <i>et al.</i> , 2003 Höller <i>et al.</i> , 1999
	Det. in 25 g	n.a.	53% ^b	France	Vernozy-Rozand <i>et al.</i> , 2002
Enterovirus	28 000	4 200-720 000	100%	Sweden	Ottoson <i>et al.</i> , <i>submitted</i>
Hepatitis A	Det in 50 µL	n.a.	23%	France	Schvoerer <i>et al.</i> , 2000
Rotavirus	215	40-510	100%	USA	Rao <i>et al.</i> , 1987
Norovirus	1 900	<800-4 500	78% ^c	Sweden	Ottoson <i>et al.</i> , <i>submitted</i>
Adenovirus	7 600 ^d	250-25 000	100%	Sweden	Bofill-Mas <i>et al.</i> , 2000
<i>Giardia</i>	2 000	260-13 000	100%	Sweden	Ottoson <i>et al.</i> , <i>submitted</i>
<i>Cryptosporidium</i>	20	<8-160	28%	Sweden	Ottoson <i>et al.</i> , <i>submitted</i>
<i>Ascaris</i>	30	n.r.	n.r.	USA	AWWA, 1999

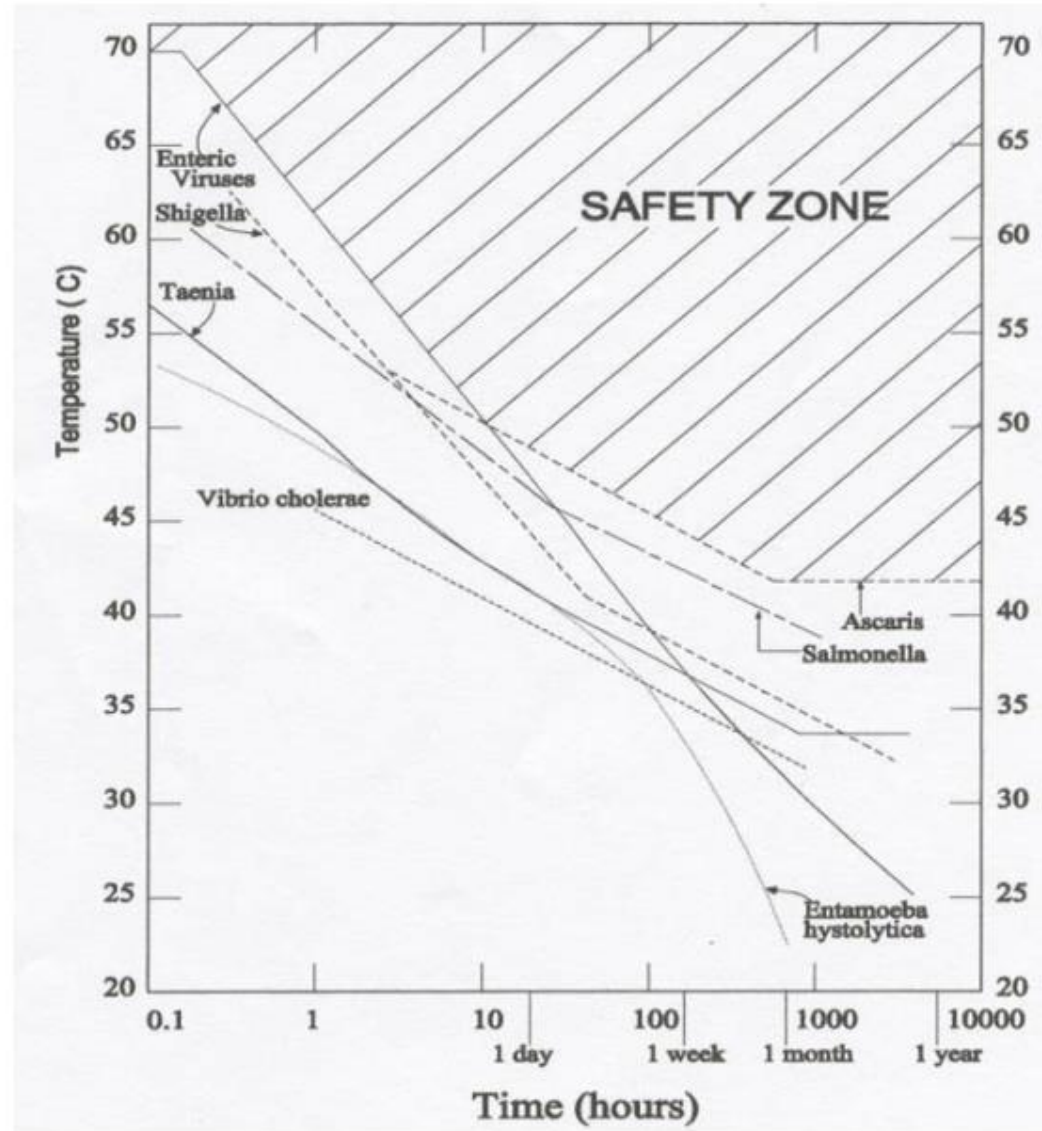
^a Percent positive samples. ^b Positive for stx genes by PCR, *E. coli* O157:H7 one of the serotypes detected. ^c 78% positive in winter time, otherwise <100 L⁻¹. ^d Mean calculated from only four samples. Det. = detected, n.a. = not applicable, n.r. = not reported

Log-linear and JM2 model decay rate coefficients in faecal sludge

Microbial group	Calculated pseudo-first order decay rate coefficient, k (days ⁻¹)			Calculated JM2 model coefficients					
	Median	5%	95%	Coefficient k_1			Coefficient k_2		
				Median	5%	95%	Median	5%	95%
Viruses	0.19	0.025	16.8	-7.2	-31.1	17.7	4.9	1.5	17.9
Bacteria	0.93	0.012	243	0.88	-78.3	216	3.6	0.45	216
Protozoa	0.0095	0.0016	0.035	-13.4	-13.6	-6.5	3.1	2.1	3.31
Helminths ^b	0.0042	0.000008	0.52	-8.7	-58.1	-1.8	3.8	1.4	17.6

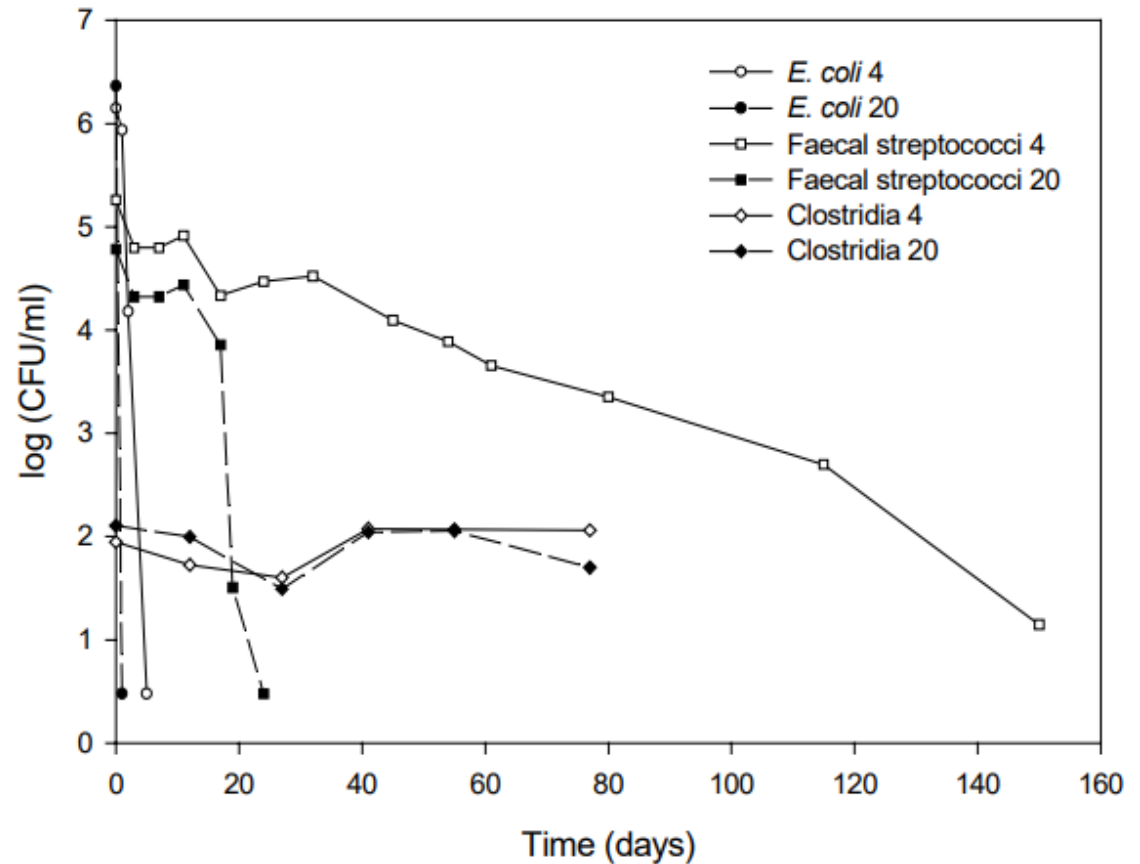
Exposure Assessment: Pathogen Reduction and persistence

Pathogen decay with respect to time and temperature



Exposure Assessment: Pathogen reduction and persistence

Inactivation of *E. coli*, faecal streptococci and *C. perfringens* spores (clostridia) in source-separated human urine (pH 9) at 4°C and 20°C.



Exposure Assessment pathogen reduction & persistence

Temperature	Timeframe	Pathogen Reduction
2-20°C	1.5-2 years	<ul style="list-style-type: none">• Will eliminate bacteria, although some might be dormant and could be reactivated• Will reduce viruses and protozoa below risk levels• Some helminth eggs may persist
20-35°C	At least 1 year	<ul style="list-style-type: none">• Substantial to complete inactivation of viruses, bacteria, and protozoa• Inactivation of helminth eggs within a few months, apart from <i>Ascaris</i> eggs that can take longer

(WHO, 2006)

pH	Time	Pathogen Reduction
Above pH 9	At least 6 months	<ul style="list-style-type: none">• Inactivation of all pathogens will take longer if the fecal sludge is wet and/or the pH is lower

(WHO, 2006)

Exposure Assessment: Amount ingested & frequency

Risk assessment study	Location	Exposure pathway	Input to QMRA	Reference for input
Asano et al. (1992)	California, USA	Swimming	100 mL	Haas (1983)
Mena et al. (2003)	Not specific	Swimming	100 mL: 1, 5 and 10 days of exposure	NG
Craig, Fallowfield & Cromar (2003)	Australia, coastal	Swimming	Uniform 20–50 mL	Ashbolt, Reidy & Haas (1997)
Steyn, Jagals & Genthe (2004)	South Africa	Full immersion	100 mL	Genthe & Rodda (1999); Haas, Rose & Gerba (1999)
Steyn, Jagals & Genthe (2004)	South Africa	Intermediate	50 mL	Medema et al. (2001)
Steyn, Jagals & Genthe (2004)	South Africa	Other – accidental gulping	10 mL	Genthe & Rodda (1999); Medema et al. (2001)
Westrell et al. (2004)	Sweden	Swimming	50 mL 50 times per year	NG
van Heerden et al. (2005)	South Africa	Swimming	30 mL	Crabtree et al. (1997)
Diallo et al. (2008)	Thailand	Swimming	100 (50) mL	NG

NG: not given

Exposure Assessment: pathogen reduction & persistence

Type of exposure	Volume ingested (mL or g)	Frequency (times * year ⁻¹)
1. WWTP worker at pre-aeration	1	52
2. WWTP worker at belt press	5	208
3. (Un)intentional immersion at wetland inlet	30	1
4. Child playing at wetland inlet	1	2
5. Recreational swimming	50	10
6. Child playing at sludge storage	5	1
7. Entrepreneur spreading sludge	2	30
8. Consumption of raw vegetables	1	2

Treatment Barrier: Reuse and hygiene practices

Control measure	Pathogen reduction (log units)	Notes
Wastewater treatment	1-6	The required pathogen reduction to be achieved by wastewater depends on the combination of health protection measures selected
Localized drip irrigation (low growing crops)	2	Root crops and crops such as lettuce that grow just above, but partially in contact with the soil
Localized drip irrigation (high growing crops)	4	Crops, such as tomatoes, the harvested parts of which are not in contact with the soil
Spray rift control (spray irrigation)	1	Use of micro-sprinklers, anemometer-controlled direction switching sprinkler, inward-throwing sprinkler etc
Spray buffer zone(spray irrigation)	1	Protections of residents near spray or sprinkler irrigation. The buffer zone should be 50-100m
Pathogen die-off	0.5 -2 per day	Die-off on crop surfaces that occur between last irrigation and consumption. The log unit reduction achieved depends on climate (temperature, sunlight intensity, humidity), time, crop type, etc.
Produce washing with water	1	Washing salad crops, vegetables and fruit with clean water
Produce disinfection	2	Washing salad crops, vegetables and fruit with weak disinfectant solution and rinsing with clean water
Produce peeling	2	Fruits, root crops
Produce cooking	6-7	Immersion in boiling or close to boiling water until the food is cooked ensures pathogen reduction

Source: WHO, 2006

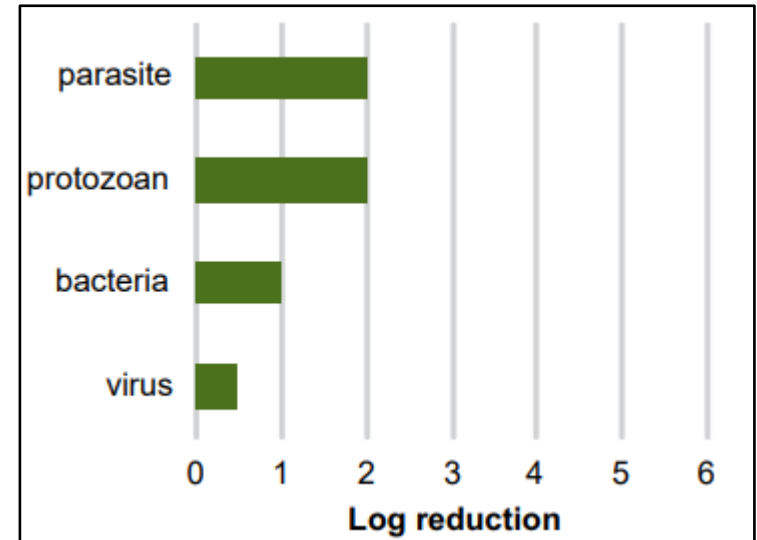
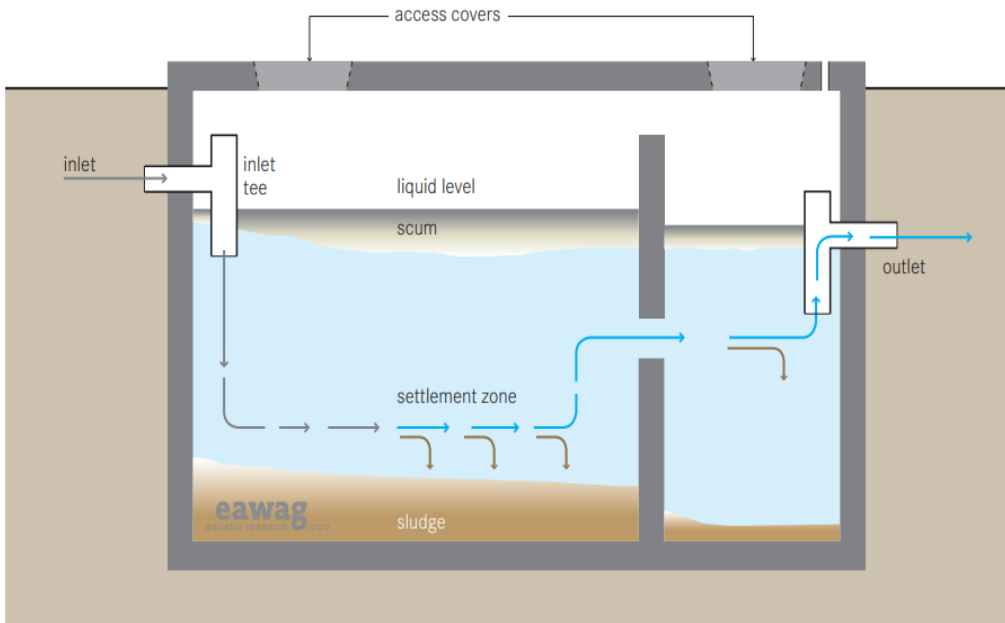
Table A5.1 Intended uses and associated exposures for recycled water

Activity	Route of exposure	Volume (mL)	Frequency/ person/ year	Comments
Garden irrigation	Ingestion of sprays	0.1	90	Garden watering estimated to typically occur every second day during dry months (half year). Exposure to aerosols occurs during watering.
Garden irrigation	Routine ingestion	1	90	Routine exposure results from indirect ingestion via contact with plants, lawns, etc.
	Accidental ingestion	100	1	Infrequent event.
Municipal irrigation	Ingestion	1	50	Frequencies moderate, as most people use municipal areas sparingly (estimate 1/2–3 weeks). People are unlikely to be directly exposed to large amounts of spray, and therefore exposure is from indirect ingestion via contact with lawns, etc. Likely to be higher when used to irrigate facilities such as sports grounds and golf courses (estimate 1/week).
Food crop consumption (home grown)	Ingestion	5 (lettuce)	7	100 g of lettuce leaves hold 10.8 mL water and cucumbers 0.4 mL at worst case (immediately post-watering). ^a A serving of lettuce (40 g) might hold 5 mL of recycled water, and other produce might hold up to 1 mL per serving. Calculated frequencies are based on ABS data. ^b
		1 (other raw produce)	50	
Food crop consumption (commercial)	Ingestion	5 (lettuce)	70	100 g of lettuce leaves hold 10.8 mL water and cucumbers 0.4 mL at worst case (immediately post-watering). ^a A serving of lettuce (40 g) might hold 5 mL of recycled water, and other produce might hold up to 1 mL per serving. Calculated frequencies are based on ABS data. ^c
		1 (other raw produce)	140	
Toilet flushing	Ingestion of sprays	0.01	1 100	Frequency based on 3 uses of home toilet per day. Aerosol volumes are less than those produced by garden irrigation.
Washing machine use	Ingestion of sprays	0.01	100	Assumes one member of household exposed. Calculated frequency based on ABS data. ^d Aerosol volumes are less than those produced by garden irrigation (machines usually closed during operation).
Firefighting	Ingestion of water and sprays	20	50	Median ingestion for firefighters estimated at 20 mL per fire, with a maximum number of fires fought within area served by recycled water of 50 per year. ^e
Cross-connection of dual-reticulation systems with drinking-water mains	Ingestion	1 000/day	1/1 000 houses	Total consumption is assumed to be 2 L per day, of which 1 L is consumed cold. ^f Affected individuals may consume water 365 days per year. A conservative estimate of 1/1 000 houses has been considered.

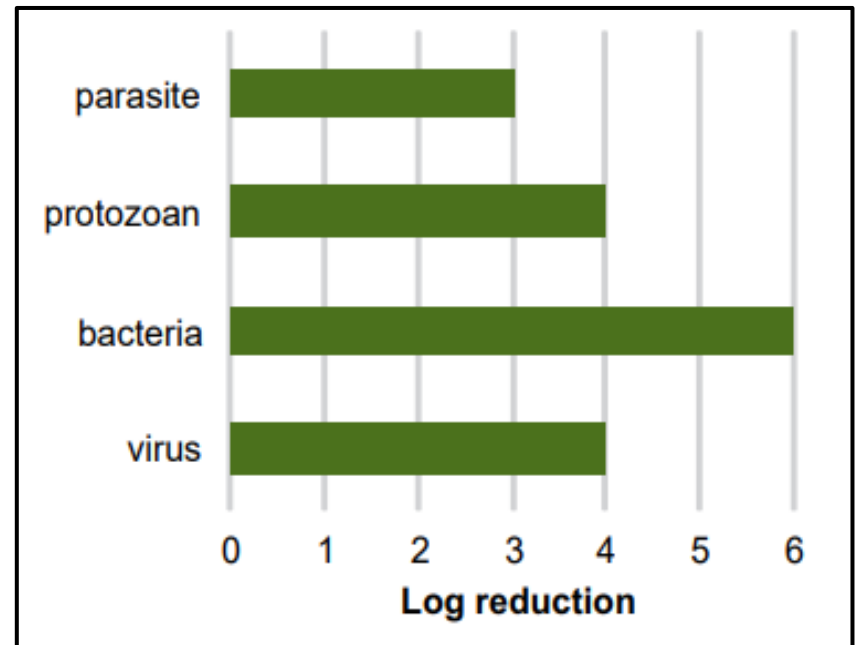
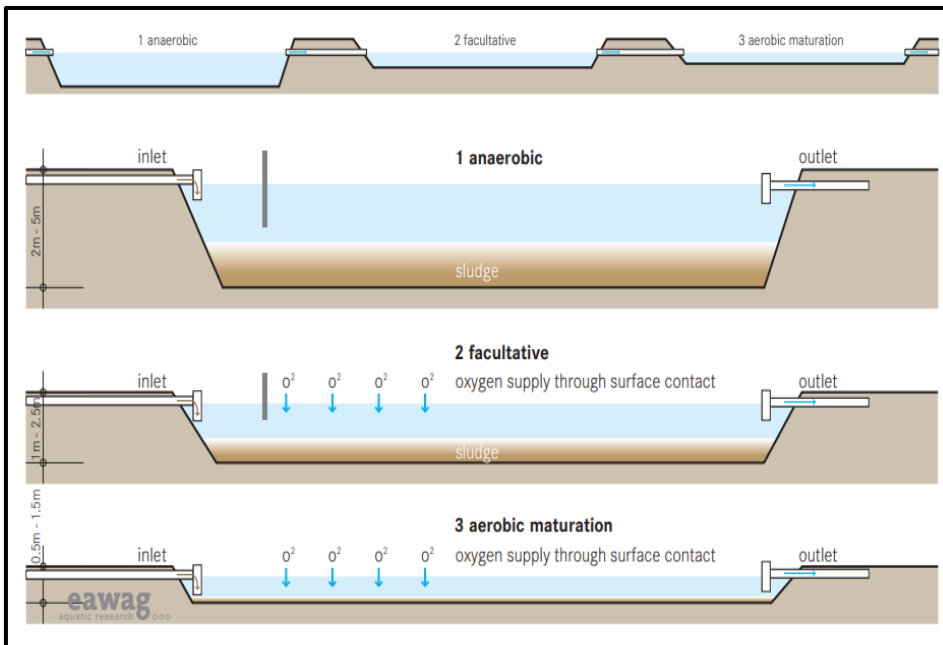
Table A5.5 Indicative log₁₀ removals of enteric pathogens and indicator organisms

Treatment	Indicative log ₁₀ reductions ^a							
	<i>E. coli</i>	Bacterial pathogens (including <i>Campylobacter</i>)	Viruses (including adenoviruses, rotaviruses and enteroviruses)	Phage	<i>Giardia</i>	<i>Cryptosporidium</i>	<i>Clostridium perfringens</i>	Helminths
Primary treatment	0–0.5	0–0.5	0–0.1	N/A	0.5–1.0	0–0.5	0–0.5	0–2.0
Secondary treatment	1.0–3.0	1.0–3.0	0.5–2.0	0.5–2.5	0.5–1.5	0.5–1.0	0.5–1.0	0–2.0
Dual-media filtration with coagulation	0–1.0	0–1.0	0.5–3.0	1.0–4.0	1.0–3.0	1.5–2.5	0–1.0	2.0–3.0
Membrane filtration	3.5→6.0	3.5→6.0	2.5→6.0	3→6.0	>6.0	>6.0	>6.0	>6.0
Reverse osmosis	>6.0	>6.0	>6.0	>6.0	>6.0	>6.0	>6.0	>6.0
Lagoon storage	1.0–5.0	1.0–5.0	1.0–4.0	1.0–4.0	3.0–4.0	1.0–3.5	N/A	1.5→3.0
Chlorination	2.0–6.0	2.0–6.0	1.0–3.0	0–2.5	0.5–1.5	0–0.5	1.0–2.0	0–1.0
Ozonation	2.0–6.0	2.0–6.0	3.0–6.0	2.0–6.0	N/A	N/A	0–0.5	N/A
UV light	2.0→4.0	2.0→4.0	>1.0 adenovirus >3.0 enterovirus, hepatitis A virus	3.0–6.0	>3.0	>3.0	N/A	N/A
Wetlands – surface flow	1.5–2.5	1.0	N/A	1.5–2.0	0.5–1.5	0.5–1.0	1.5	0–2.0
Wetlands – subsurface flow	0.5–3.0	1.0–3.0	N/A	1.5–2.0	1.5–2.0	0.5–1.0	1.0–3.0	N/A

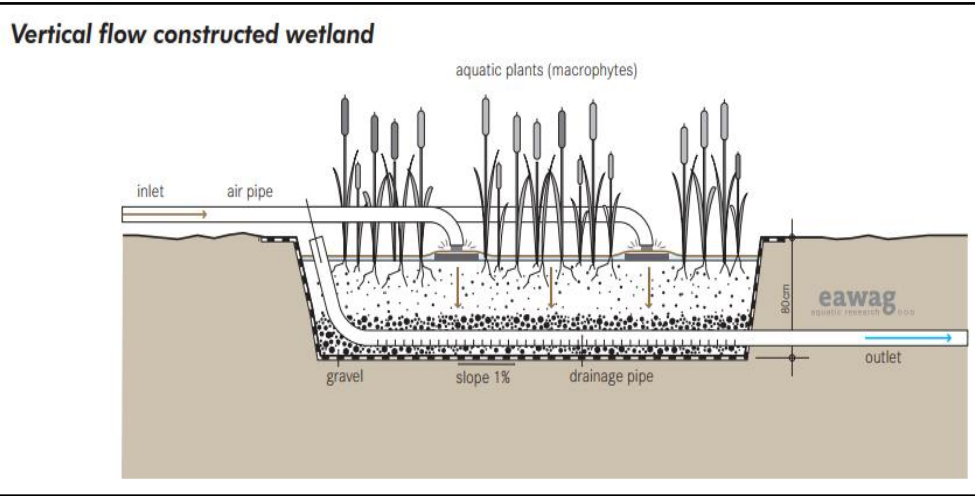
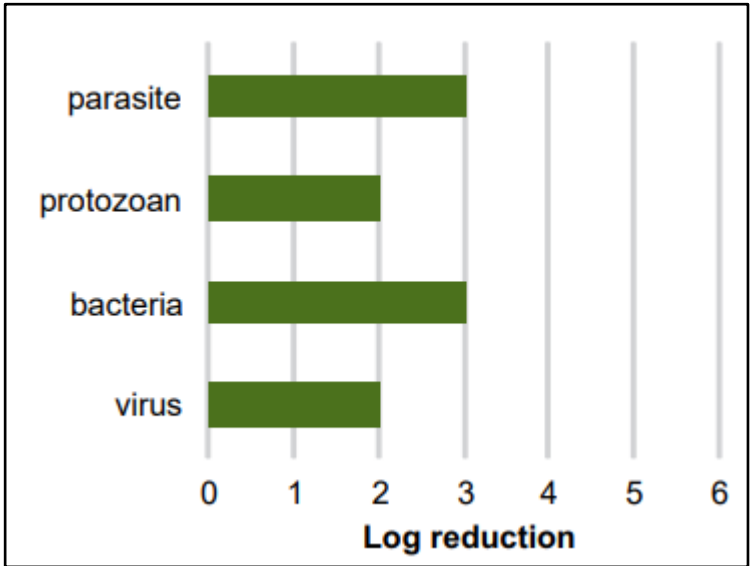
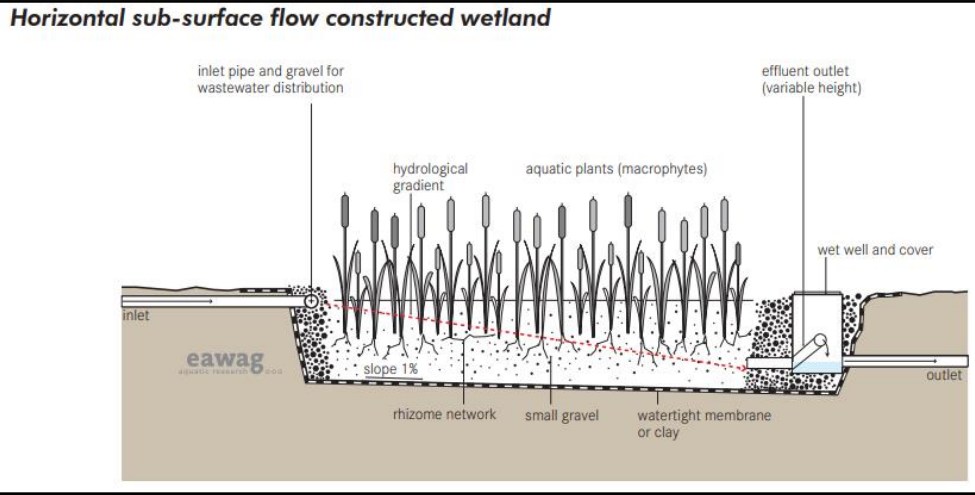
Treatment Barrier: Septic Tank



Treatment Barrier: Waste stabilization ponds

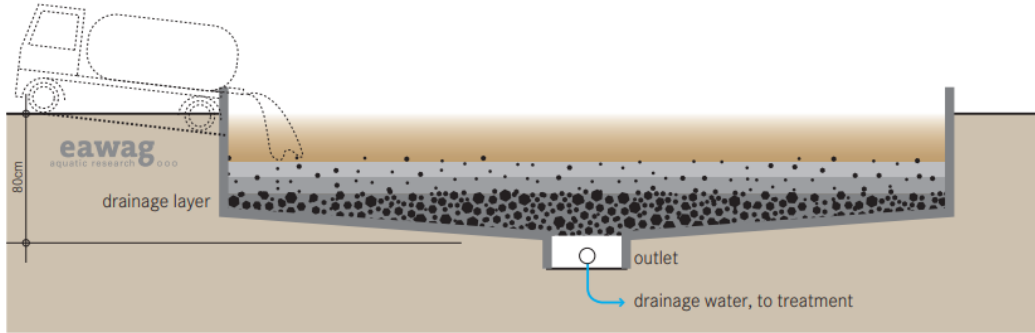


Treatment Barrier: Constructed wetland

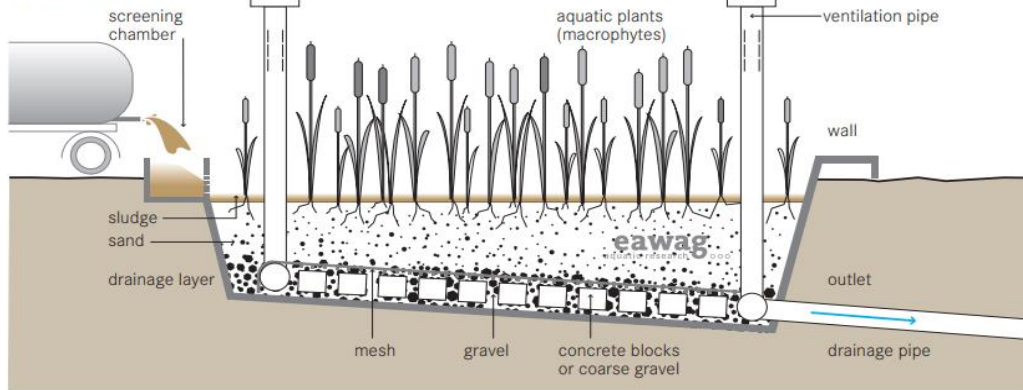


Treatment Barrier: Planted & unplanted drying bed

Unplanted drying beds

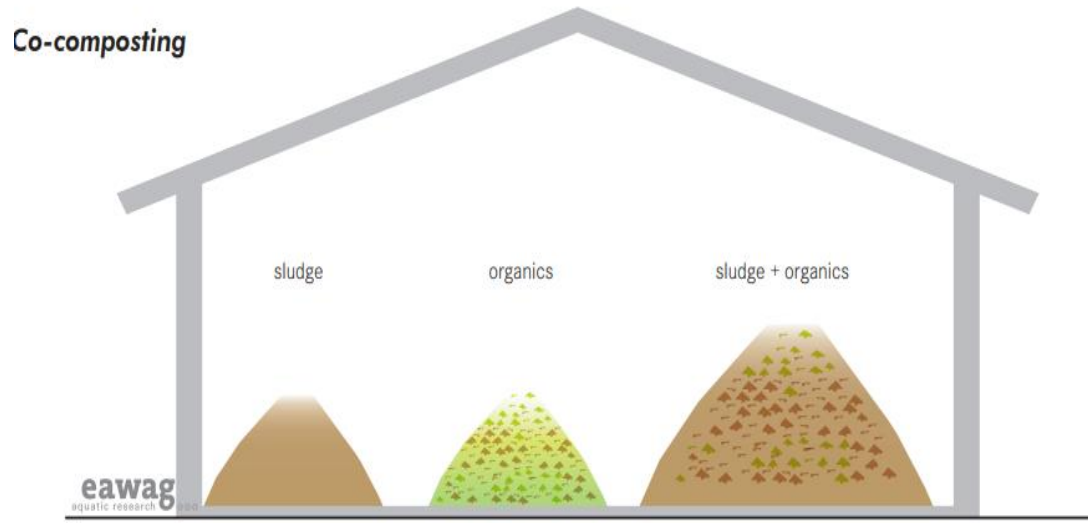


Planted drying bed



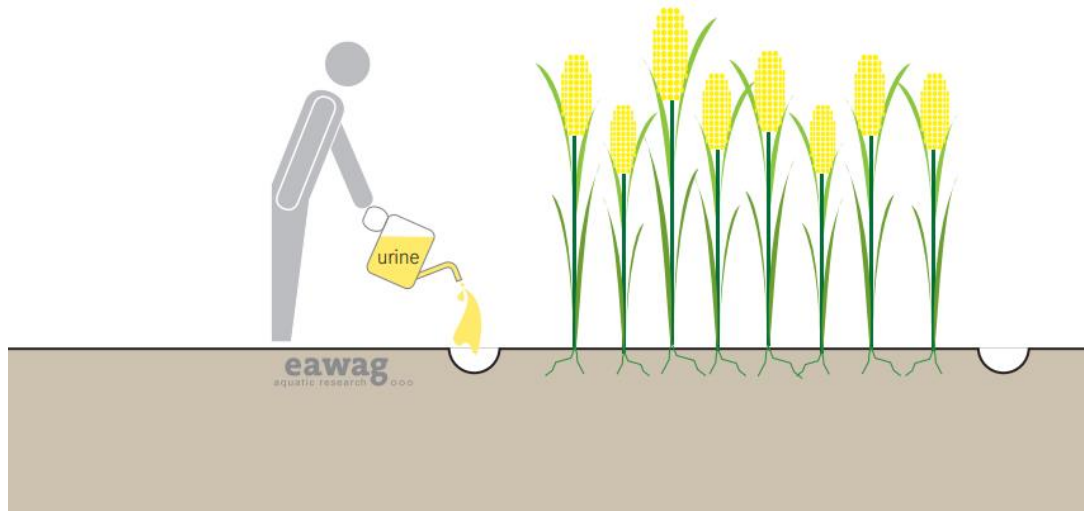
Viruses	1- <6
Bacteria	
Protozoa	
Helminths	1-3

Treatment Barrier: Co-composting



Viruses	2- <6
Bacteria	1.8- <6
Protozoa	2.5
Helminths	1-2

Treatment Barrier:



Storage temp	Storage time	Possible pathogen in the urine mixture	Recommended crops
4°C	> 1 month	Viruses, protozoa	Food and fodder crops that are to be processed
4°C	> 6 months	Viruses	Food crops that are to be processed ^c
20°C	> 1 month	Viruses	food crops that are to be processed, fodder crops ^c
20°C	> 6 months	Probably none	all crops ^d

Adapted from Hoglund (2001)