

The epidemiology and disease burden potential relating to private water supplies in Scotland



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Glossary

Dose	The number of pathogenic micro-organisms ingested.
Deterministic	It tells us that some future event (e.g., illness from a PWS) can be calculated exactly, without the involvement of randomness.
Epidemiology	The study of diseases and their causes in populations.
Gastroenteritis	An infection of the gut whose symptoms may include diarrhoea, vomiting and abdominal pain.
Hazard	A potential source of harm or adverse health effect on a person or persons.
Hydraulic conductivity	Describes the ease with which a fluid (usually water) can move through pore spaces or fractures.
Immunocompromised	Have a reduced ability to fight infections and other diseases.
<i>in silico</i>	Conducted or produced by means of computer modelling or computer simulation.
Macropores	In soil, macropores are defined as cavities that are larger than 75 μm .
Microtopography	The surface features of a material, or of the earth or other body, on a small or microscopic scale.
Pathogen	A bacterium, virus, or other microorganism that can cause disease.
Sequela	Any abnormality following or resulting from a disease or injury or treatment (plural – sequelae).
Sorption	A physical and chemical process by which one substance becomes attached to another.
Source attribution	Human illness source attribution is defined as the partitioning of the human disease burden of one or more foodborne infections to specific sources (e.g., cattle, sheep, pigs etc).
Stochastic	Random, specifically involving a random variable. This is the opposite of "deterministic".
Zoonoses	Infectious diseases of animals (usually vertebrates) that can naturally be transmitted to humans.

Executive Summary

Research aim

This project sought to develop an understanding of the epidemiology and disease burden contribution of private water supplies in Scotland on the public health of the populations (indigenous and transient) exposed to these supplies.

Background

Around 3.3% of Scotland's population (182,516) are served by private water supplies (PWS) together with transient visitors such as tourists. In 2019 there were 3,837 Regulated private water supplies and 18,616 Type B (domestic) supplies registered with local authorities¹. Recent reports from the Drinking Water Quality Regulator (DWQR)¹ highlight that improvements need to be made, in particular to PWS. Furthermore, there is a need to understand the epidemiology and disease burden that PWS contribute to in terms of the potential impact on public health. It is accepted that there are multiple exposure routes to disease-causing microorganisms, meaning health surveillance alone may not readily identify clusters of illness attributable to waterborne contaminants. The impact of exposure of waterborne disease on transient populations is also less well understood and this will vary with season.

Research undertaken

This report comprises a risk profile (Part A) to provide current knowledge about the risks of gastrointestinal pathogens associated with private water supplies in Scotland. This is supplemented with preliminary work looking at the linkage of human gastrointestinal illness with private water supply microbiological failures (Part B) and the scoping out of a quantitative microbiological risk assessment for PWS in Scotland (Part C).

Key findings and recommendations

Risks to Scottish Consumers

Key findings

- *Campylobacter* causes the largest number of reported gastrointestinal cases in Scotland, followed by viruses (norovirus, rotavirus, adenovirus) and then *Salmonella*, with protozoa (*Cryptosporidium*, *Giardia*) and Verotoxigenic *Escherichia coli* (VTEC) being comparatively rare.

- In terms of hospitalisation the largest number of reported gastrointestinal cases in Scotland are from *Campylobacter* (8.9 hospital admissions/100,000 people) with the remainder of the gastrointestinal pathogens comprising <2 hospital admissions/100,000 in total.
- An accurate estimate of the number of cases caused by these pathogens due to exposure from PWS does not yet exist. However, case control data and Quantitative Microbial Risk Assessment (QMRA) models show that around 8% of *Campylobacter* (NE Scotland) and <8% of *E. coli* O157 cases (across whole of Scotland) may be due to PWS. In addition, outbreaks show that during 2005-2014 only one VTEC outbreak was associated with a PWS in Scotland.
- There are differences in infectivity, survival, inactivation, and occurrence of the pathogens. There are also some differences in these characteristics when these pathogens are subdivided into their different sub-types.
- Establishing the extent of Scottish PWS contribution to the burden of Gastrointestinal (GI) disease needs further investigation, as the quantitative analysis of the risk is still in its infancy.
- There are several modelling/analytical approaches that can be used to estimate the number of cases due to PWS. These include quantitative risk assessment, quantitative risk factor approaches using regression analysis on disease data, case-control studies, source attribution models and a combination of the aforementioned techniques.

Recommendation

- There is a need to develop and apply quantitative risk assessment in combination with epidemiological approaches to quantify the contribution of PWS to the human disease burden in Scotland.

Infectious disease transmission from animal reservoirs to water to humans

Key findings:

- Understanding the pathways taken by pathogens from animal reservoirs to PWS to human disease is essential in identifying how to manage the risk of illness.
- There is a growing body of knowledge on the prevalence and concentration of pathogens in both farm and wild animal reservoirs but there are still gaps that need to be filled, particularly in the latter. There is also progress on understanding and modelling the

¹ <https://dwqr.scot/information/annual-report/>

mechanisms of pathogen transfer from animal faeces, through soil or runoff to surface and ground waters. However, the application of this to PWS in Scotland on a regional or national scale has not yet been attempted.

- Knowledge exists in part on treatment of water supplies as well as failure rates to indicator organisms. There have been some studies looking for pathogens in PWS but only one in NE Scotland looking at *Campylobacter* was of a reasonable size (i.e., >100 PWS tested).
- In general, the PWS supplies most at risk are surface water abstracted supplies, particularly burns and rivers which are untreated and in areas where there are both animal reservoirs excreting pathogens and mechanisms by which these pathogens can enter the PWS.
- Studies have been conducted that have estimated consumption of water from PWS by humans and dose response models have been developed for the main pathogens of concern. The spectrum of illness for these pathogens is also known in the general population but not for specific PWS associated cases.
- The QMRA method can be used to model the transmission of pathogens from animal reservoirs to PWS. This is scoped out in Part C.

Recommendation

- There is a need to develop risk assessment models which follow the movement of pathogens from animal sources, through the environment, to water supplies and then to the human population.

Risk Management Options

Key findings

- A range of risk management options are available and include catchment management measures, adequate barriers and security around the raw water intake, appropriate treatment to ensure quality, good maintenance, and operation, underpinned by appropriate monitoring.
- The effectiveness of the risk interventions needs to be demonstrated through the risk assessments, water safety plans and monitoring programme for each supply.
- These options also need to be considered in terms of their practicality of implementation, cost, and acceptability to users.

Recommendations

- It is important that local authorities ensure risk assessments are carried out as per the regulations so

that the appropriate risk management options can be identified and implemented.

Linkage of human gastrointestinal illness with private water supply microbiological failures

Key findings

- A low percentage of gastrointestinal illnesses (0.7% for Regulated private water and 1.0% for Type B) were potentially linked to microbiological failures of PWS. It is possible that these linked cases may not have acquired illness from PWS but from other sources (e.g., contact with farm animals or travel abroad).
- Around one quarter of GI illnesses could not be included because of lack of postcode information and the analysis could involve only those PWS where microbiological sampling had been conducted.

Recommendation

- Explore potential approaches outlined in the report for further data analysis e.g., time series analysis to look at the time between a PWS failure and human infection.

Scoping a Quantitative Microbiological Risk Assessment approach

Key findings

- A spreadsheet model is proposed which would predict the probability of illness from drinking a glass of water from a PWS.
- The outline of the model was compiled as part of this project to identify:
 - o Where existing data can be used as part of the risk assessment and to validate it where appropriate.
 - o Those data that are missing and need to be collated (e.g., pathogen shedding by wild animals, transport of pathogens from animal faeces to the PWS, efficacy of PWS treatment systems on reducing levels of gastrointestinal pathogens etc.).
 - o A number of steps that will require model building (e.g., transport models of pathogen movement from animal sources to PWS etc).

Recommendation

- Data gaps need to be addressed for each of the pathogens of interest so that QMRA models can be developed.

Conclusions

In summary there is a need to:

1. Make better use of the available epidemiological information, where relevant to supplement this with additional studies, to obtain realistic estimates of the importance of PWS as a source of human disease.
2. Develop a QMRA model that describes the transmission of pathogens from animal reservoirs to private water supplies from catchment to regional and national scales.
3. Fill data gaps that are required to implement the above model.
4. Use the model to evaluate the efficacy of risk management strategies to help reduce the incidence of disease.

1.0 Introduction

1.1 Background

Access to safe drinking water continues to be a priority for the Scottish Government. Around 3.3% of Scotland's population (182,516 people) are served by private water supplies (PWS) together with transient visitors such as tourists. In 2019 there were 3,837 Regulated private water supplies and 18,616 Type B (domestic) supplies registered with local authorities². Recent reports from the Drinking Water Quality Regulator (DWQR)³ highlight that improvements need to be made in PWS to improve their microbiological safety. Furthermore, there is a need to understand the epidemiology and disease burden that PWS contribute to in terms of the potential impact on public health. It is accepted that there are multiple exposure routes to disease-causing microbiological entities, meaning health surveillance alone may not readily identify clusters of illness attributable to waterborne contaminants. The impact of exposure of waterborne disease on transient populations is also less well understood and this will vary with seasonality.

1.2 Aim and objectives

This project sought to develop an understanding of the epidemiology and disease burden contribution of private supplies on the public health of the populations (indigenous and transient) exposed to the drinking water supplies. The objectives were to:

1. Explore the existing body of research on disease burden and epidemiology of small rural drinking water supplies.
2. Review existing data to include existing water quality data and health surveillance reports.
3. Investigate the potential of linking water quality failures against clusters of illness within the community.
4. Scope out a Quantitative Microbiological Risk Assessment approach to assessing the disease burden of small rural supplies which can be used to inform future regulation.
5. Scope out a series of metrics that can be used to measure the effectiveness of treatment of rural water systems and the risk to public health.
6. Make recommendations which inform improvements to management of private supplies.

² Numbers refer to known registered PWS. It is assumed that some PWS may not be registered.

³ <https://dwqr.scot/information/annual-report/>

1.3 Report structure

This report comprises a risk profile (Part A) to provide current knowledge about the risks of gastrointestinal pathogens associated with private water supplies (PWS) in Scotland. This is supplemented with preliminary work looking at the linkage of human gastrointestinal illness with private water supply microbiological failures (Part B) and the scoping out of a quantitative microbiological risk assessment for PWS in Scotland (Part C).

Part A: Risk Profile

A.1 Introduction

The purpose of this risk profile is to provide current knowledge on the risks of gastrointestinal pathogens associated with private water supplies in Scotland. This should be of interest to both regulators and consumers. The former to inform risk management decisions and to assess their likely impact *in silico* prior to implementation. The latter to identify potential risks associated with their private water supplies and to give insight into what can be done to reduce these risks.

Risk profiles have previously been developed for a number of food/pathogen combinations (Lake, et al. 2005, Mataragas, et al. 2008). A risk profile can be considered as part of step 1 of an iterative risk management framework (Step 1. Evaluate Risks, Step 2. Assess Risk Management Options, Step 3. Implement Risk Management Decision and Step 4. Monitor and Review) (Anon. 2000).

The risk profile collates and summarises the current state of knowledge into the four primary steps of risk assessment (Codex 2007):

1. Hazard identification—identifies the pathogenic microorganism(s) of concern and the vehicle (e.g., water, food) with which it is associated.
2. Exposure assessment—aims to determine the mechanisms through which the human population is exposed. In a risk profile this may include both qualitative and quantitative data on the levels of pathogens and also the amount and frequency of the vehicle consumed.
3. Hazard characterisation—gives a quantitative or qualitative assessment of the adverse effects of the pathogen to humans. More specifically a dose–response model can be implemented which mathematically models the variability in impact (response) following exposure to different doses (McNab 1997).
4. Risk characterisation—gives a probability of occurrence of the illness and also the severity of the health effects in

a given population. In a risk profile this usually relies on surveillance data.

The purpose of the risk profile is to inform risk management. The information contained within a risk profile enables decisions to be made on whether to conduct a quantitative risk assessment, apply risk management strategies and/or collate additional data. This risk profile concerns the full range of gastrointestinal pathogens that can be found in private water supplies in Scotland. The final section provides the main conclusions and recommendations for further action.

A.2 Hazard identification: the organisms

Hazard identification is the identification of the agents (in this case gastrointestinal pathogens) which can cause human illness (e.g., gastroenteritis) as well as the vehicle by which these are transmitted to humans (in this case private water supplies).

2.1 Inventory of gastrointestinal pathogens that can be found in private water supplies

Gastrointestinal pathogens in drinking waters (including PWS) can be classified into three categories: bacteria, viruses, and protozoa. Table 1 presents a list of waterborne pathogens associated with gastroenteritis, including their public health significance from an international perspective. Viruses are found mostly in sewage and surface waters, which are the main sources of pollution for public waters (Grondahl-Rosado et al. 2014; Maunula et al. 2005; Riera-Montes, et al. 2011). Viruses that infect humans predominantly come from human faeces (Ashbolt 2004). Viruses were not considered further in this report for two reasons. First, the risk to PWS in rural areas from human faeces is predominantly via septic tanks and much work has been done on this previously (Wallender et al. 2014). Second, there have been no outbreaks associated with PWS in Scotland as far as the authors are aware.

Giardia is considered as one the four most important pathogens for drinking water in Europe. However, in Scotland the incidence of *Giardia* disease is relatively low (4 cases/100,000 people per year) and data from Health Protection Scotland suggest that the infection is associated with foreign travel, particularly to Egypt and the Indian sub-continent (Smith-Palmer et al. 2012). Also, a quantitative microbial risk assessment in small PWS in England suggests that the median annual risk for *Giardia* is relatively low (0.4% to 0.7% per annum) (Hunter et al. 2011). Therefore, *Giardia* has not been studied in detail in this report.

The pathogens and indicator organisms covered by this report are as follows and a detailed appendix is provided for each:

Bacteria

- *Salmonella* (Appendix I)
- Verocytotoxin-producing *Escherichia coli* (VTEC) (Appendix II)
- *Campylobacter* (Appendix III)
- Indicator organisms (Appendix IV)

Protozoa

- *Cryptosporidium* (Appendix V)

Table 1. Waterborne gastrointestinal pathogens of concern in drinking waters*. Pathogens covered in this report are shown in blue text.

Name of micro-organism	Health significance ^a	Persistence in water supplies ^b	Resistance to chlorine ^c	Dose at which probability of illness is 50%: ID ₅₀ (95% CIs) ^d	Primary sources
Bacteria					
<i>Salmonella</i>	High	Moderate	Low	6.65 (0.69 – 5.89x10 ⁴) cfu ^e	Human and animal faeces
VTEC	High	Moderate	Low	5.4 x 10 ⁵ (10 – 9 x 10 ¹²) cfu ^f	Human and animal faeces
<i>Campylobacter (jejuni and coli)</i>	High	Moderate	Low	5.8 (1.0 – 9.0) cfu ^g	Human and animal faeces
Enteric viruses					
rotaviruses	High	Long	Moderate	1500 (370 – 2.2 x 10 ⁴) ffu ^h	Human faeces
adenoviruses	Moderate	Long	Moderate	Unknown	Human faeces
astroviruses	Moderate	Long	Moderate	Unknown	Human faeces
norovirus	High	Long	Moderate	Unknown	Fomites and water
Protozoa					
<i>Cryptosporidium homonis, C. parvum</i>	High	Long	High	165 (85 – 200) oocysts ⁱ	Human and other animal faeces
<i>Giardia lamblia</i>	High	Moderate	High	34.8 (12 – 158) cysts ^j	Animal faeces

*Adapted from (Ashbolt 2004) and (WHO 2011).

^aHealth significance relates to the incidence and severity of disease, including association with outbreaks.

^bDetection period for infective stage in water at 20 °C: short, up to 1 week; moderate, 1 week to 1 month; long, over 1 month.

^cWhen the infective stage, is freely suspended in water, treated at conventional doses and contact times and pH between 7 and 8. Low means 99% inactivation at 20 °C generally in < 1 min, moderate 1–30 min and high > 30 min.

^dID₅₀ is the average ingested dose needed to cause infection in 50% of the population exposed to hazard

^e(Teunis et al. 2010)

^fThe ID₅₀ was calculated for *E. coli* O157 from outbreak and environmental data (Strachan, et al. 2005).

^gThe ID₅₀ was calculated for *Campylobacter* from an outbreak in children which consumed milk at a farm visit (P. Teunis, Van den Brandhof et al. 2005)

^hThe ID₅₀ was calculated for rotavirus data from a healthy volunteers study, in 62 adults, the subjects ingesting between 9 x 10⁻³ to 9 x 10⁴ focus-forming units (ffu) (Haas et al. 1993)

ⁱThe ID₅₀ was calculated for *Cryptosporidium* data from a healthy volunteers study (Haas 2000)

^jThe ID₅₀ was calculated for *Giardia* data from a healthy volunteers study (Teunis et al. 2002; Teunis et al. 1996)

2.2 Hazard identification of the pathogens studied: summary

Detailed hazard identification information is provided for each pathogen in Section 1.0 of their respective appendices, under the following sub-sections:

- The organism
- Growth and survival
- Inactivation (critical control points and hurdles)
- Sources

Each pathogen can be subdivided up into a number of different types (e.g., serotypes for *Salmonella* and VTEC

and sequence types for *Campylobacter*. The occurrence and behaviour (e.g., growth, behaviour, and survival) of these types may differ. All of the pathogens studied have non-human reservoir hosts and a number have several (e.g., *Campylobacter* and *Salmonella* have poultry, ruminants, wild animals etc). These pathogens may or may not grow outwith a host (e.g., *Salmonella* and VTEC can grow outwith but *Cryptosporidium* and *Campylobacter* cannot). There is the potential to inactivate the pathogens by various means but there are significant differences in disinfection efficacy between pathogens (e.g., chlorination readily inactivates the bacterial pathogens but not *Cryptosporidium*). As a result of the differences between

the pathogens and also within (e.g., serotypes etc) it is important to use this information in the risk assessment process and also take account of this when considering risk mitigation strategies.

A.3 Hazard identification: private water supplies

The regulations place a duty on the local authorities to conduct risk assessments on all regulated supplies which supply commercial or public activities or serve greater than 50 people (Regulated private water supplies). These risk assessments must be conducted every five years. In addition, the local authorities are required to provide assistance on risk assessments on all other smaller, domestic supplies (Type B) on request. The purpose of the risk assessments is to determine risks to health and corrective actions that will improve the drinking water quality of each supply. The risk assessments are a critical part of the regulatory framework which provides an overview of the full water supply system. It provides much more insight into the risks than the water quality testing which is only done once a year. It is intended that the risk assessments will, in future, form the basis of a water safety plan for each supply. This section explores the risks identified with the PWS and highlights the number of supplies by council area in Scotland. Overall, 58% of Regulated private water supplies have risk assessments completed in 2019.

3.1 Relevant characteristics of the private water supply

The vast majority of private supplies are located in rural areas of Scotland, although a very small number are located in urban centres (for example 0.1% of the Aberdeen City population are served by private supplies). The PWS are abstracted from either ground or surface water and the more common quality failures are microbiology, metals (typically iron, aluminium, manganese and in some cases lead), turbidity, colour and pH. Surface waters have a higher risk of microbiology failure than ground water sources, but this varies with seasonality, flow, treatment and activity near the surface water source. Some groundwater sources also fail for microbiology and this may be attributed to borehole depth, security of the well and treatment.

Regulated private water supplies were found to be 87.1% compliant for *E. coli* compared to 89% compliant in 2018 (DWQR 2020).

3.1.1 Inventory of PWS in Scotland (Table of PWS or properties on PWS by council)

Each local authority area has responsibility for identifying and regulating the Regulated private water supplies and B supplies within their area. The number of private supplies has increased and Table 2 sets out the number of recorded private water supplies by council area in 2019 (DWQR 2020).

Table 2. Summary of private water supplies by local authority area (DWQR 2020).

Local Authority	Regulated Supplies	Risk Assessed Supplies	Percentage of Risk Assessed Supplies	Population on Regulated Supplies	Population on Reg Risk Assessed Supplies	Pop. on Reg Supplies with no Risk Assessment
Aberdeen City	8	8	100%	55	55	0
Aberdeenshire	301	288	96%	4,160	4,090	70
Angus	40	38	95%	1,556	1,550	6
Argyll and Bute	512	492	96%	15,653	15,600	54
City of Edinburgh	6	1	17%	93	45	48
Clackmannanshire	6	6	100%	147	147	
Comhairle nan Eilean Siar	15	10	67%	236	223	13
Dumfries and Galloway	552	219	40%	18,528	18,082	446
Dundee City	No Regulated Supplies					
East Ayrshire	25	24	96%	340	190	150
East Dunbartonshire	1	1	100%	34	34	0
East Lothian	21	9	43%	483	110	373
East Renfrewshire	7	7	100%	858	858	0
Falkirk	1					
Fife	72	60	83%	2,471	2,357	114
Glasgow City	No Regulated Supplies					
Highlands	854	760	89%	26,040	25,654	386
Inverclyde	11	5	45%	818	592	226
Midlothian	37	2	5%	288	16	272
Moray	265	50	19%	2,903	352	2,551
North Ayrshire	23	3	13%	1,721	25	1,696
North Lanarkshire	8	2	25%	65	4	61

A.4 Hazard characterisation: adverse health effects

Hazard characterisation details the dose response and the spectrum of human illness/disease symptoms (e.g., gastroenteritis) associated with specific pathogenic microorganisms.

Detailed information on hazard characterisation is provided for each pathogen in Section 2.0 of their respective appendices, under the following sub-sections:

- Disease symptoms
- Dose response
- Susceptible population
- Particular subtypes found in both humans and PWS

A.5 Exposure assessment

The exposure assessment aims to determine the path of exposure and the frequency and the intensity of the exposure.

5.1 Pathways from sources of pathogens to private water supplies

The fate of faecal pathogens once deposited on land is a complex issue (Jamieson et al. 2002; Akoumianaki et al., 2016). It is clear, however, that hydrological processes are key in allowing pathogens to be transported from sources of pathogens (e.g. faecal material) to private water supplies, and several potential pathways have been identified (Oliver et al. 2005; Reddy et al. 1981). These pathways will be discussed below.

5.1.1 Surface pathways

The transport of pathogens over the soil surface by overland flow has largely been identified as the hydrological pathway which generates the greatest risk of faecal contamination to private water supplies (Jamieson et al. 2002; Tyrrel & Quinton 2003). In general, the orders of magnitude increase in faecal indicator organism (FIO) concentrations that are often seen during major storm events are attributed to the development of greater hydrological connectivity due to overland flow initiation, and to the greater velocity and efficiency of pathogen transport which these flows permit (Rodgers et al. 2003; Kay et al. 2008; Tetzlaff et al. 2010). It has also been suggested that overland flow causes a larger risk of contamination because the processes of straining / filtering and sorption can prevent pathogens from entering the soil, thus increasing the availability of pathogens at the soil surface for transport by overland flow (Reddy et al. 1981).

For pathogens to be transported by overland flow, they must first be detached from their source on the soil surface or directly from the soil surface after previously having been deposited by overland flow, and then entrained by the flow (Tyrrel & Quinton 2003). Detachment and entrainment occur because of kinetic energy exerted by raindrop impact or by the flow itself (Tyrrel & Quinton 2003). Faecal pathogens may travel in one of three possible states when in overland flow: as free cells or cell clumps, attached to soil particles, or attached to faecal particles (Tyrrel & Quinton 2003). At present, it is largely uncertain what governs the mode by which faecal pathogens are transported in overland flow (Jamieson et al. 2002; Tyrrel & Quinton 2003; Collins et al. 2005). A recent study (Pachepsky et al. 2008) suggested that strain-dependent differences in *E. coli* surface properties may cause different strains of *E. coli* to preferentially attach to certain soil particle sizes, which in turn may affect the ease by which pathogenic and non-pathogenic strains can be transported in overland flow.

Detachment of faecal pathogens and entrainment by overland flow does not necessarily mean that pathogens will reach a private water supply, however. For example, it is possible for pathogens in overland flow to become restrained by the microtopography of the soil surface (Oliver et al., 2005). It is also possible that overland flow may infiltrate into the soil before reaching a private water supply, causing either the deposition of faecal pathogens on the soil surface or their infiltration into the soil as well (Collins et al. 2005).

5.1.2 Sub-surface pathways

Whilst it was mentioned in the last section that the processes of straining / filtration and sorption can prevent faecal pathogens from entering the soil system, it is nonetheless possible for this to occur. Effective infiltration and movement of pathogens through the soil is most likely to occur in the presence of macropores (Guber et al. 2009). Macropores are larger conduits within the soil structure that provide preferential flow pathways for potentially rapid sub-surface flows, and which may be formed, for example, by the action of soil fauna or plant roots (Beven & German 1982; Jamieson et al. 2002). When moving through macropores, faecal pathogens may once again be transported as free cells or cell clumps or attached to soil or faecal particles (Oliver et al. 2005). In addition, however, transport of pathogens through macropores may be aided by the action of soil fauna, with it being possible for pathogens to attach to the fauna which then moves through the soil (Oliver et al. 2005).

Whilst macropores offer a more efficient sub-surface pathway for the transport of faecal pathogens compared with transport through the soil matrix itself, it is possible

for pathogen transport through macropores to be slowed. The characteristics of the soil on the inside of a macropore may promote pathogen retention (Oliver et al. 2005). Recent work (Guber et al. 2009) has found, however, that this effect may be reduced when soils are closer to saturation and if pathogens are attached to faecal particles, whilst it has been suggested (Reddy et al. 1981) that pathogen retention in macropores may be reduced in soils with a lower clay content. Motility of bacteria and their subsequent movement against the direction of macropore flow may also be a factor which restricts pathogen transport in macropores (Oliver et al. 2005).

Unlike macropore flow pathways, flow pathways through the soil matrix are more likely to promote the attenuation of faecal pathogen transport, and this is largely due to the processes of straining / filtration and sorption (Collins et al. 2005). For example, it has been reported (Weldeyohannes et al. 2018) that during the movement of untreated effluent through the unsaturated zone to groundwater, there was a 2-3 \log_{10} reduction in the most probable number of *E. coli* per 100 ml. Sorption refers simply to the process whereby faecal pathogens become attached to the soil, with this process becoming more prevalent in clay- and organic-rich soils (Oliver et al. 2005). Straining / filtration, meanwhile, occurs when pathogens become trapped because of pore sizes in the soil matrix being smaller than the size of the faecal pathogens carried in the soil water (Oliver et al. 2005). In addition to the initial trapping of pathogens by this process, it has also been suggested (Thullner et al. 2002) that this bioclogging causes a reduction in local soil hydraulic conductivity, which may further limit the movement of soil water and its associated pathogens. It should be noted, however, that as straining / filtration is a function of soil pore size and the size of the faecal pathogen, this process may be less likely to affect smaller pathogens such as viruses (Jamieson et al. 2002). Where pathogen transport does occur in the soil matrix, it is likely to be more efficient in coarser soils with reduced clay content (Hagedorn et al. 1978).

A final sub-surface pathway that should be mentioned is an artificial one: field drains. If faecal pathogens can infiltrate into the soil and then percolate vertically to field drains, it has been suggested that this pathway may be a significant contributor to contamination risk in agricultural areas (Jamieson et al. 2002). Flow rate of field drains, time since animals were last grazing, and whether manure is applied to the land have all been identified as important factors in the risk of contamination from field drains (Evans & Owens 1972). There has also been recent work (Rieke et al. 2018), where 16S-rRNA gene sequencing was used to determine the likely transport pathways of different pathogens. It was identified that whilst field drains could effectively transport a diverse range of pathogens, there were examples where certain genera,

such as *Enterobacter*, could be found in significantly higher concentrations in drainage water than overland flow; this suggests that artificial drains may be a more important pathway for some types of faecal bacteria than others.

5.1.3 Direct defecation and in-channel sources

The preceding sections have dealt with the major surface and sub-surface pathways of pathogens from sources on the soil surface to private water supplies. It is also important to recognise, however, that animals may defecate directly into water courses (Collins et al. 2005). This may occur if water courses are directly accessible to animals and livestock, or if livestock must cross water courses due to specific farming practices (e.g., being moved for milking; Davies-Colley et al. 2004). The interaction of animals with a water body could also indirectly give rise to a further pathway for pathogens. If animals frequently cross or congregate close to a body of water, the soils close to the water body could become compacted and prone to saturation. This, in turn, could give rise to chronic seepages of FIOs and pathogens into a water body, which could contribute to sustained background levels of faecal contamination (Neill et al. 2018).

It is also important to recognise that in-channel stores of faecal pathogens in water courses feeding private water supplies may contribute significantly to contamination risk (McKergow & Davies-Colley 2010). Stores of pathogens in streambed sediments can accumulate due to direct defecation by animals, or through the deposition of sediment and associated pathogens from the water column (Oliver et al. 2005). Once deposited, it has been found that streambed sediment may offer suitable conditions for the survival of faecal pathogens for several weeks, by providing soluble organic material, nutrients and protection from predation and UV-B light (Oliver et al. 2005, Kim et al. 2010). It has recently been shown in some studies that the resuspension of sediment and the associated release of stored pathogens can be a greater contributing factor to impaired microbial water quality than overland flow pathways (Stocker et al. 2018). Re-suspension of in-channel sources is most likely to occur during periods of high flow, as observed previously in an artificial flood experiment (Muirhead et al., 2004). It was further observed (Jamieson et al. 2002) that re-suspension occurred only above a shear-stress threshold, and that this threshold was most likely to be exceeded during the rising limb of a flood event. However, there has also been work (Pachepsky et al. 2008) that has suggested that mixing during baseflow conditions can lead to the transfer of FIOs from streambed sediments to the water column to sustain FIO concentrations in streams during these periods. Overall, it has been increasingly recognised that

considering in-channel sediments as a source of pathogens is vital to the management of microbial water quality (Droppo et al. 2009).

5.1.4 Survival of pathogens

In addition to understanding the pathways which may exist to connect faecal pathogen sources to private water supplies, it is also necessary to consider the factors that influence the survival of faecal pathogens, as this will affect the likelihood of pathogens initially being available for transport, and then the likelihood of them surviving the journey to a private water supply (Reddy et al. 1981; Oliver et al. 2005). Very briefly, reviews (Jamieson et al. 2002, Oliver et al. 2005) suggest the following as important factors affecting faecal pathogen survival:

- **Soil moisture:** A key control on the survival of faecal bacteria, with generally greater soil moisture content increasing survival.
- **Soil type:** This affects water retention, and thus soil moisture.
- **Temperature:** Higher temperatures appear to decrease survival of faecal bacteria – die-off rate may be defined as doubling with a 10°C increase in temperature, in the range of 5-30°C.
- **Organic matter:** Organic matter promotes survival and potentially re-growth of faecal bacteria by increasing

nutrient and water retention, as well as providing a source of carbon.

- **Competition:** Faecal bacteria in the soil system must compete with resident bacteria. This is also true for faecal bacteria in water courses where they must compete with better-adapted aquatic microbes for nutrients.
- **UV radiation:** UV-B may damage the DNA of faecal bacteria.
- **Predation:** Faecal bacteria may be preyed upon by protozoa.
- **Soil pH:** Optimum pH for minimal faecal bacteria die-off is found to be between 6 and 7.

5.2 The hazard in the Scottish private water supply

During the reporting year for 2019, 24.6% of the Regulated private water supplies sampled failed to meet compliance standards for coliforms and 12.9% for *E. coli* (DWQR 2020). This means that 1 or more coliforms/*E. coli* were detected per 100 ml potable water. Another faecal indicator organism, Enterococci, was present in 9.6% of Regulated private water supplies. Failures due to *E. coli* by local authority are given in Table 3 (DWQR 2020).

Table 3. Results of testing of Regulated private water supplies for microbiological quality in 2020 (DWQR 2020)

Local Authority	<i>E.coli</i>	Enterococci	<i>Clostridium perfringens</i>	Coliform Bacteria
Aberdeen City	0%	100%	100%	0%
Aberdeenshire	95%	95%	99%	85%
Angus	N/A	N/A	N/A	0%
Argyll and Bute	90%	90%	93%	75%
City of Edinburgh	0%	100%	100%	0%
Clackmannanshire	100%	100%	88%	63%
Comhairle nan Eilean Siar	78%	0%	82%	61%
Dumfries and Galloway	89%	93%	91%	78%
Dundee City	No regulated supplies			
East Ayrshire*	N/A	N/A	N/A	N/A
East Dunbartonshire	100%	100%	100%	N/A
East Lothian	75%	73%	93%	65%
East Renfrewshire	89%	89%	100%	89%
Falkirk	100%	100%	100%	100%
Fife	90%	89%	92%	74%
Glasgow City	No regulated supplies			
Highland	89%	94%	92%	80%
Inverclyde	76%	92%	92%	68%
Midlothian	86%	86%	92%	70%
Moray	87%	87%	95%	72%
North Ayrshire	85%	85%	92%	82%
North Lanarkshire	50%	50%	100%	50%
Orkney	92%	89%	89%	79%
Perth and Kinross	70%	76%	89%	60%
Renfrewshire	100%	100%	100%	100%
Scottish Borders	81%	88%	92%	70%
Shetland	100%	100%	100%	100%
South Ayrshire	98%	98%	98%	84%
South Lanarkshire	88%	88%	83%	81%
Stirling	92%	99%	90%	84%
West Dunbartonshire	100%	100%	100%	86%
West Lothian	100%	100%	100%	100%
Total	87.1%	90.4%	92.90%	75.4%

* N/A denotes that the local authority provided no samples for the specific parameters. East Ayrshire only reported failing samples and therefore was treated as not having provided any samples for compliance calculation purposes.

For Type B supplies, failure rates were 43% and 23 % respectively for coliforms and *E. coli*. The degree of compliance is heavily influenced by the type of water source, as shown (Table 4) for scheduled samples (DWQR 2020). These supplies failed with respect to one or more of the following: coliforms, *E. coli*, *Clostridium perfringens*, Enterococci, irrespective of type of treatment on the supply. Groundwater sources are less likely to be microbiologically contaminated, however they are not completely compliant.

Table 4. Microbiological compliance of Regulated private water and Type B supplies by source type (DWQR 2020)

Source Type	Compliance (%)	
	Regulated private water	Type B
Groundwater borehole	95	87
Groundwater spring	88	71
Groundwater well	85	73
Surface water loch	85	72
Surface water rainwater	100	-
Surface water watercourse	85	71

Table 5. Microbiological data from private water supplies in Scotland 2013-14. Data are confirmed counts for tap water and presumptive counts for source water. One sample of source and tap water was taken per supply per quarter (Avery et al. 2016 and Avery – unpublished data).

Quarter (sampling month)	Water	<i>E. coli</i> CFU/100 ml (number positive/number of supplies)	Total coliforms CFU/100 ml (number positive/number of supplies)	<i>Clostridium</i> CFU/100 ml (number positive/number of supplies)	Enterococci CFU/100 ml (number positive/number of supplies)
Q1 (October)	Tap	0-10 (2/34)	0-80 (3/34)	0-8 (3/33)	0-4 (2/35)
	Source	0-800 (15/33)	0-9800 (31/33)	*	*
Q2 (Feb-Mar)	Tap	0-3 (3/34)	0-570 (4/34)	0-5 (5/34)	0-2 (1/34)
	Source	0-10 (8/33)	0-2100 (29/33)	*	*
Q3 (May)	Tap	0-<10 (1/33)	0-660 (4/33)	0-2 (4/33)	0-6 (2/33)
	Source	0-37 (15/32)	0->10,000 (28/32)	*	*
Q4 (July-Aug)	Tap	0-2 (3/32)	0-120 (3/32)	0-1 (2/32)	0-2 (3/32)
	Source	0-5000 (26/32)	0-520,000 (31/32)	*	*

There are few reports in the peer-reviewed literature on the prevalence of specific pathogens in private supplies in Scotland. It has been reported (Licence, et al. 2001) on cases of *E. coli* O157:H7-associated illness, noting that in the week prior to the first case, the water supply (a spring supplying a campsite) was tested and found to contain total coliforms (11 CFU/100 ml) and *E. coli* (15 CFU/100 ml). Samples taken during the outbreak continued to show the presence of FIOs. Analysis of PWS data across Aberdeenshire between 1992 and 1998 (Reid et al. 2003) reported failure rates rather than absolute microbiological counts, thus these are superseded by the data held currently by local authorities and DWQR. They did, however, note that microbiological failure rates displayed a seasonal trend being greater during the latter half of the year. A study of over 30 private water supplies across Scotland (Avery et al. 2016) involved quarterly sampling of raw (source) and potable (tap) water and results are presented in Table 5. Analyses comprised confirmed counts for coliforms, *E. coli*, *Clostridium* and Enterococci for potable waters and presumptive counts for *E. coli* and Enterococci on raw waters.

5.3 Water consumption from PWS by the Scottish population

5.3.1 Total PWS consumption per person (per/day)

A telephone-based exposure assessment questionnaire of a representative (by age, population density, socioeconomic status) subsample of the Grampian health board population (990 questionnaires from a population 519,979) was conducted by Aberdeen University in 2009. From this survey water consumption was estimated from the number of unboiled glasses of water drunk per person per day (Figure 1) (MacRitchie et al. 2013). On average the 979 respondents consumed 2.8 glasses of water/day

and assuming one glass of water contains 190 ml (Hunter et al. 2011), the average daily water consumption is 532 ml. This agrees with USA survey data (daily consumption range – 430 -2900 ml) (Gale 2001).

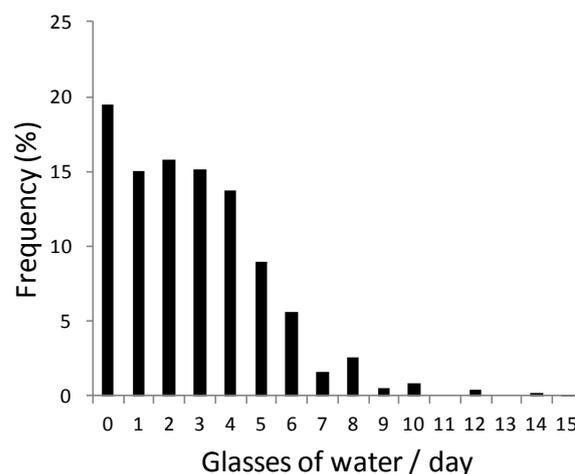


Figure 1. The distribution of number of glasses of water consumed daily per person in Grampian during 2009. The distribution is left skewed (average – 2.8 glasses/day, median – 2 glasses/day, mode – 0 glasses/day, range – 0 to 15 glasses/day).

Other consumption of drinking water include cleaning teeth, having a shower, swimming, other recreational water activities, other activities using water (e.g. irrigation in agriculture) and also preparing food (e.g., washing fruit or vegetables).

A QMRA study of *Cryptosporidium* in China estimated that people ingest between 7 to 71 ml of tap water per session of tooth-brushing (An et al. 2011). Water inhaled in lungs via aerosols was reported either by showering (1.9 ml/event) or hosing (1.9 ml) (Ahmed et al. 2010). It was also estimated that people ingest 1ml of water during hosing (Ahmed et al. 2010).

Although there is much data in the literature on the amount of water ingested during swimming, surfing, diving and other recreational water activities, they are

not given here because it is considered that they are not relevant for this study (Crabtree et al. 1997; Vinten et al. 2009). This is despite that there may be some swimming pools and spas on private water supplies but it is considered that there will be low numbers of these and certainly the swimming pool water should be treated and that disease in humans is only likely to follow on failure of the treatment system.

5.4 Quantitative estimate of exposure

Detailed information on quantitative estimate of exposure is provided for each pathogen in Section 3.0 of their respective Appendices, under the following sub-sections:

- Contamination prevalence/frequency, concentration, survival/growth in water.
- Dose ingested.

5.4.1 Exposure summary

There is a lack of data on the exposure to waterborne pathogens from PWS in Scotland except for *E. coli* O157. Data on PWS water consumption exist and can be readily used in QMRAs (see sub-sections 5.3.1 and 5.4.1). However, in terms of prevalence, concentration, survival, and dose ingested there are data gaps for most of the pathogens (*Salmonella*, *Campylobacter* and *Cryptosporidium*) discussed in this report. For *Cryptosporidium* there is prevalence and concentration data from a UK wide study (including two PWS from Scotland) which can potentially be adapted to Scotland. Table 6 summarises the availability of prevalence and concentration data for PWS in Scotland.

Table 6. Prevalence and concentration data availability in PWS in Scotland.

Prevalence and concentration in PWS		
Pathogen	Prevalence	Concentration
<i>Salmonella</i>	No	No
<i>E. coli</i> O157	Yes	Yes (estimated)
<i>Campylobacter</i>	Yes	No
<i>Cryptosporidium</i>	No ^a	No ^b

^{a,b}Data from a UK wide study exist (including two PWS from Scotland) (Hunter et al., 2011).

5.4.2 Number of exposure events (e.g., per day)

5.4.2.1 Total Scottish population

Based on a telephone survey conducted in Grampian (see subsection 5.3.1) the proportion of people who drink

water daily is 80.5 %. The number of exposures per day can be calculated by multiplying the number of people on PWS and the proportion of people drinking water daily. There are approximately 80,000 people in Scotland who regularly use private water supplies such as wells, springs, burns and boreholes to provide their drinking water. Hence, a reasonable estimate of the number of exposed people per day will be 64,000 (= 80.5% x 80,000). In the same survey, 47.5% of respondents declared that their PWS was treated. No treatment efficiency was provided. Assuming that the treatment efficiency was 100%, which will be an overestimate, the number potentially contaminated water exposures per year from a PWS was 12 million (= 64,000 x 365 x 52.5%, note: 64,000 – number of people on PWS exposed daily, 365 – days, 52.5% – proportion of PWS not treated).

5.4.2.2 Vulnerable Scottish population

The telephone-based survey in Grampian (see subsection 5.3.1 above) shows a different water consumption pattern between age groups (Figure 2). The average water consumption in the population ≥ 60 years old (2.4 glasses/day, 95%CI: (2.20 – 2.67)) is significantly lower (P<0.05) than the average water consumption in the population < 60 years old (3.2 glasses/day, 95%CI: (2.96 – 3.43)). To account for this water consumption difference in QMRAs, the distribution by age of the Scottish population drinking from PWS needs to be determined. This can be estimated either from the existing survey data in Grampian, or from a future survey which can be done at national scale. There is no information available on the number of daily exposures for other vulnerable populations (e.g., immunocompromised, young children etc.).

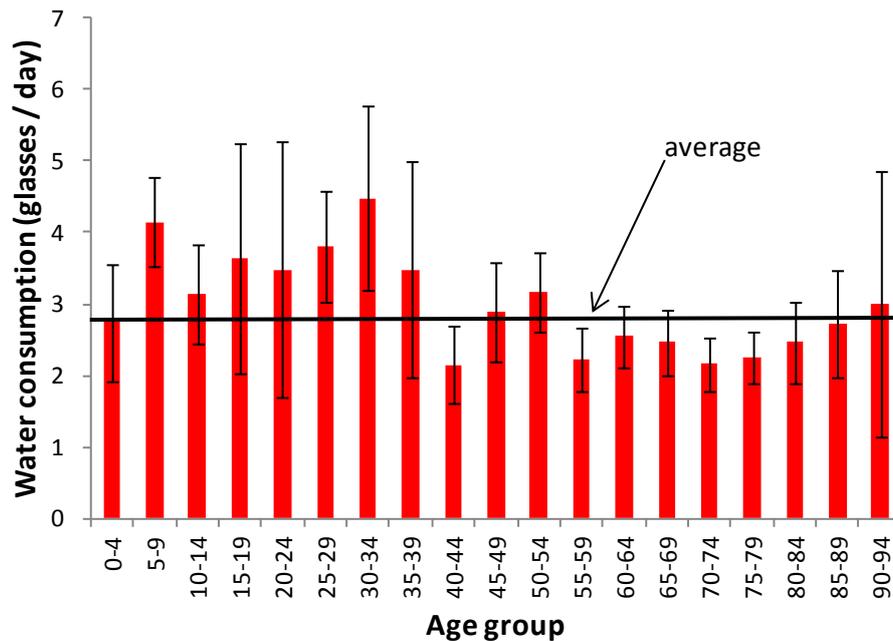


Figure 2. The distribution of water consumption in Grampian health board in 2009, stratified by age group (this includes the whole population, part of which will be on a PWS).

5.4.3 Treatments

Approximately 85% of Regulated private water supplies have some form of treatment applied. This appears to be fairly consistent across years and local authorities (Grzybowski, Scottish Government, 2018 – pers. comm.). Determining the presence and efficacy of treatment on PWS is difficult due to the large number of supplies which are not routinely sampled by the local authorities (Type B supplies). Based on treatment data held by DWQR for Type B supplies in East Ayrshire, Midlothian, Stirling, and Moray, it appears that around 35% of supplies have some form of treatment. Again, this is reasonably consistent across years. The caveats associated with these data are that local authorities may not have supplied DWQR with all of their treatment data or may not have completed returns correctly. DWQR holds treatment data for all local authorities and the compliance data supports that disinfection is only present and consistently effective in less than two thirds of supplies (DWQR 2017). Furthermore, presence of a treatment system does not give an indication of the maintenance of treatment systems. For example, if filtration systems and UV lamps are not regularly replaced and system components cleaned (e.g., lamp quartz sleeves), treatment efficacy decreases.

In a study of 31 Type B private water supplies in Scotland during 2013-14 (Avery et al. 2016), a number of treatment types were being used (Table 7).

Table 7. Treatment of Type B PWS in Scotland during 2013-14 (31 supplies studied).

Description of treatment system	Frequency
20 µm filter, UV, pH, and then into header tank	1
25 µm bag filter, 10 µm 10-inch filter, carbon filter and UV tube	1
5 µm, carbon filter, UV, Fe filter	1
Arsenic, Fe, Mn, ion exchanger, UV filters	1
Burn water – between header tank and house: 50 µm mesh, 5 µm filter, UV (spun wound). Rainwater – 350-gallon tank: from roof to gutter mate, mesh filter, nylon filter mesh.	2
Clean out tank once a year and UV filter	1
Course, fine filter, pH correction, UV, pressure vessel	2
Fe and Mn filters, UV, pH correction	1
Filter, pH, and UV	1
From settlement tank 5500 l through 30 m filter then 5 µm filter then UV filter	1
Mn filter, UV, Pumped from well to holiday cottage tank then down to (owners) house	1
Pressure tank, filter, pH correction then UV	1
Pressure vessel, filter, sand, possible pH, 5 – 10 µm filter and UV	1
Pump to accumulator tank, 20 m filter, 5 µm filter, UV bulb to softener balls to house	1
Pump. Chlorine before holding tank. 20 and 5 µm filter at property level and UV	1
Sediment filter, UV filter, CO ₂ filter,	1
Source pumped to ground level tank, through filter and UV, then into roof space tank	1
Standard general filter, large cylinder for Mn removal, UV lamp	1
Through 2 filters, UV and into taps	1
UV filter, 50 µm filter, then to tap	1
UV, secondary filter	1
UV, storage cupboard	1
Chalk and sand filters, micron filters then UV	1
Cotton filter, pH and UV	1
Header tank, pump, 50 µm filter, pH filter, element filter, UV	1
Media filter, 5 µm, UV, pumped for pressure	1
One filter then UV bulb	1
pH correction, aerator to drop out the iron, iron filters, organic scavenger with a brine backwash, then through a carbon filter and a 5 µm filter, then UV	1
Pre filter, UV bulb, pH crystals, 5 µm filter	1

The treatments associated with passes or fails in the study (Avery, et al. 2016) are summarised below.

The nine supplies which consistently passed microbiological AND chemical parameters were fitted with some form of filter, usually a 5 µm filter, and UV disinfection; some with pH correction but only two out of these nine had any additional treatment. The two supplies with additional treatment had some form of media/element removal filter. The fourteen supplies failing microbiological standards on at least one of four sampling occasions tended to have more complex/less standard treatment systems. This implies that either it is difficult to treat supplies (i.e. requiring more complex treatment) or possibly non-standard installations that tend to fail microbiologically. This is consistent with the understanding that physico-chemical water quality directly affects disinfection processes.

5.4.3.1 Filtration

A 5 µm pre-filter is commonly fitted to most PWS with a treatment system. These comprise mesh or paper-based cartridge filters. The primary intention is to remove particulates such that the UV disinfection can operate effectively (particulates can shield microorganisms against UV penetration (Winward et al. 2008)). There is a paucity of information on the effects of 5 µm (or coarser) filters on removal of microorganisms in PWS, presumably because they are not intended for this purpose, although it is inevitable that some removal will take place, particularly as filters age and pores become clogged.

5.4.3.2 UV

The most common disinfection treatment present on PWS in Scotland is UV irradiation. In a correctly installed system, the UV dose delivered is calculated based on the flow rate. Table 8 (modified from Avery et al. 2016), illustrates the UV dose required (mJ/cm²) to give a range of log-inactivations for the bacteria/bacterial forms listed. The standard specification of UV fluence for private water supplies is currently 40 mJ/cm². Older systems may have been installed to a specification of 30 mJ/cm².

Table 8. UV dose (mJ/cm²) required to give 1.0, 2.0, 3.0 and 4.0-log inactivation of different bacteria/bacterial spores.

Target	Log ₁₀ inactivation			
	1.0	2.0	3.0	4.0
Spores				
<i>B. subtilis</i> spores ¹	28	39	50	62
<i>B. subtilis</i> spores ²	56	111	167	222
Bacteria				
<i>Campylobacteri jejuni</i> ³	-	-	-	4.6
<i>Campylobacteri jejuni</i> ²	3	7	10	14
<i>Clostridium perfringens</i> ²	45	95	145	-
<i>Clostridium perfringens</i> ³	-	-	-	23.5
<i>Enterobacter cloacase</i> ³	-	-	-	10 (33)
<i>Enterolítica faecium</i> ³	-	-	-	17 (20)
<i>E. coli</i> ¹	3	4.8	6.7	8.4
<i>E. coli</i> O157:H7 ³	-	-	-	6 (25)
<i>E. coli</i> O157 ²	5	9	14	19
<i>E. coli</i> wild type ³	-	-	-	8.1
<i>E.coli</i> wild type ⁴	-	-	-	6-8.5
<i>E.coli</i> wild type ²	5	9	14	19
<i>Klebsiella pneumoniae</i> ³	-	-	-	20 (31)
<i>Legionella pneumophila</i> ³	-	-	-	9.4
<i>Legionella pneumophila</i> ²	3-8	6-15	8-23	11-30
<i>Mycobacterium smegmatis</i> ³	-	-	-	20 (27)
<i>Pseudomonas aeruginosa</i> ³	-	-	-	11 (19)
<i>Salmonella typhi</i> ³	-	-	-	8.2
<i>Salmonella typhi</i> ²	6	12	17	51
<i>Shigella dysenteriae</i> ATTC29027 ³	-	-	-	3

¹ (USEPA 2010), ²(Hijnen, et al. 2006), ³(Bolton, 1983), ⁴(Bucheli-Witschel, et al. 2010).

Table 9. Water disinfection studies with UV used for inactivation of Giardia cysts and Cryptosporidium oocysts – adapted from (Betancourt & Rose 2004).

Protozoan	UV dose (mJ/cm ²)	Water condition	Log10 inactivation
<i>C. parvum</i> ¹	50	Treated filtered surface water	3.9
<i>C. parvum</i> ²	1	Buffered saline	1.5
<i>C. parvum</i> ²	3	Buffered saline	>3.2
<i>G. muris</i> ³	5	Milli Q water	2
<i>G. lamblia</i> ⁴	1	Buffered saline	>4

¹(Bukhari et al. 1999); ²(Zimmer et al. 2003); ³(Craik et al. 2000); ⁴(Linden et al. 2002).

The efficacy of UV disinfection is governed by: the correct installation of the system (i.e., appropriate UV dose delivered for flow rate); maintenance of the system (aging lamps fail to deliver the correct UV dose and lamp fouling (coating) can prevent UV penetration throughout the water passing through the UV tube) and, importantly, water quality (Avery et al. 2016). Water is generally suitable for disinfection by UV (40 mJ/cm²) if:

- UV transmittance (UVT) of the water to be disinfected is >75 %.
- The colour of the water to be disinfected is 20 H or lower as colour reduces UVT.
- The turbidity of the water should be < 4 NTU as turbidity reduces UVT.
- The concentration of iron in the water is < 50 g l⁻¹ (to minimise lamp fouling).
- The concentration of manganese is <20 g l⁻¹ (to minimise lamp fouling).
- Hardness is <120 mg l⁻¹ CaCO₃ (unlikely to be an issue in Scotland).
- Water storage (tanks, etc.) should be prior to UV treatment to prevent post treatment re-activation (Avery, et al. 2016).

Determining whether water quality is indeed suitable for UV disinfection or what treatments need to be installed to mitigate poor (physico-chemical) water quality is not straight forward due to the effects of weather and seasonality on PWS. For example, presence of indicator organisms tends to rise in PWS following several days of rainfall (Auld et al. 2004). In a recent study on the impact of water quality and maintenance on UV disinfection efficacy on PWS, it was evident that in general, four samples throughout the year gave sufficient information to deduce the degree of water quality variation (when comparing with monthly samples), however a single sample did not provide sufficient information (Avery et al. 2016). It is usually impractical to wait for a year's worth of quarterly samples to determine the breadth of water quality variation; therefore, local authorities and most householders rely on risk assessment profiles along with a single water sample.

5.4.3.3. Other treatment methods

Media filters

While frequently referred to as filters in the industry, media "filters" are essentially vessels containing media (usually activated carbon) which adsorb pollutants. They are frequently used to remove metals and other non-microbial contaminants. They are not designed to remove pathogens, although it is likely that microorganisms do indeed adsorb to the activated carbon particles, therefore, some removal is likely to occur. For example,

Hijnen et al. 2006, evaluated the removal of range of indicator organisms from water and reported that *E. coli* and the anaerobic spores were removed to a degree from ≤0.1–1.1 log. MS2 phages were not removed and (oo)cysts of *C. parvum* and *G. lamblia* were removed significantly (1.3–2.7 log). However, some studies suggest that activated carbon filters can become colonised by microorganisms including coliforms (Tobin et al. 1981; McFeters et al. 1985).

Settling tanks

Settling tanks again are not specifically intended to remove pathogens, rather, they allow particles to settle out. Microorganisms present in drinking water supplies are commonly associated with particulates (Liu et al. 2016), therefore, a proportion are likely to be removed during settling. The number and size of particles is dependent on the influent water quality and the number of particle-associated bacteria is also highly variable dependent on the number and type of bacteria present and the physico-chemical characteristics of both particle and water matrix. The rate of settling for particles of a given size can be estimated using a mathematical model (Stoke's law). Planktonic (i.e. free bacteria/viruses in the water column) can usually be considered as a colloidal particles unlikely to settle during the residence time in the tank (Liu et al. 2016).

Ion exchange resin

Ions with the same charge are exchanged between the water and a solid-phase resin. Cation exchange is used for water softening and removal of certain heavy metals. Anionic exchange is used to remove nitrate (WHO 2004). Literature searches, (WoS; search terms "water ion exchange bacteria/viruses/protozoa/*Cryptosporidium/coli* – in title – all years"), did not yield any relevant information on losses of pathogenic or indicator microorganisms during water treatment through ion exchange.

Chlorine dosing

Chlorine may be used in several ways in PWS: firstly as the primary disinfectant, in which chlorine is dosed into the system with the intention of generating a chlorine residual and both disinfecting and preventing regrowth of microorganisms. Secondly it can be used as part of a system cleaning regime e.g., when tanks are cleaned out, a single dose of chlorine is applied (and may also be flushed into the pipework and retained for a given contact time) to disinfect. This latter approach may also be applied following an event, such as a pipe-break, where ingress of soil or other contaminated material is known to occur. Finally, chlorine dosing may be used in parallel with UV disinfection to ensure a more robust process. Chlorination has the disadvantage that it can form harmful disinfection by-products, depending on other water quality parameters (Li et al. 2019).

A.6 Risk characterisation

6.1 Approach

Risk characterisation can be qualitative or quantitative and aims to estimate the risk of an adverse health effect based on exposure (e.g., from drinking a glass of water from a PWS which is contaminated with gastrointestinal pathogens). Risk characterisation integrates the hazard identification, hazard characterisation and dose response, in order to estimate the magnitude of the public health problem.

Risk characterisation for the main pathogens involved in pathogenic infections from drinking water in Scotland (*Salmonella*, *Verocytotoxin-producing Escherichia coli* (VTEC), *Campylobacter*, *Cryptosporidium*) is summarised in Section 4.0 of their respective appendices, under the following sub-sections:

- Incidence
- Clinical consequences of infection
- Outbreaks
- Sporadic cases
- Risk assessments
- Qualitative/quantitative assessment of risk
- Risk categorisation
 - o Disease incidence
 - o Disease severity

Where information does not exist from Scotland, data were sought from elsewhere in the UK.

6.2 Risk characterisation summary

Depending on data availability, ideally from Scotland or alternately elsewhere in the UK, the risk characterisation for the pathogens discussed in this study was performed using the following: disease incidence; clinical consequences of infection; outbreaks; sporadic cases and risk assessment and risk categorisation.

Disease incidence

The disease incidence for GI pathogens in Scotland is well documented and data exists for at least the last decade. Ranking the disease incidence data puts *Campylobacter* first, followed by enteric viruses, *Salmonella* and *Cryptosporidium*, with VTEC and *Giardia* coming last (see Table A.6 in Appendix VI). However, there is no information on what proportion of overall GI cases is related to PWS, except for some estimates for individual pathogens (e.g., VTEC and *Campylobacter*, see 6.2.8 and 6.3.8).

Clinical consequences of infection

In terms of the clinical consequences of infection, VTEC (30-57% hospitalisation) and *Campylobacter* (8% hospitalisation) are the most harmful GI pathogens, followed by *Salmonella* (3.6% hospitalisation). Severe health complications occur most frequently for VTEC and *Campylobacter*. There appear to be a lack of recent data on the severity of *Cryptosporidium* cases in Scotland. Also, there are no data published to establish if GI cases associated with PWS consumption have the same spectrum of symptoms as those acquired from elsewhere.

Outbreaks

In Scotland outbreaks are dominated by viruses, with over 95% of those reported to HPS having norovirus (NV) as the main cause (Smith-Palmer & Cowden 2010). No general outbreaks (defined as more than one household) associated with norovirus have been reported in Scotland and for single household outbreaks it would be difficult to identify a source of infection. Although for the majority of the NV outbreaks the suspected vehicle is unknown, the outbreaks occur mainly in public institutions and hospitals, the virus spreads mainly by person to person transmission, water and in particular PWS being a less probable vehicle (Smith-Palmer & Cowden 2010; Smith-Palmer & Cowden 2013). The remaining GI outbreaks (>160 in the last 10 years) are caused by pathogenic bacteria, with VTEC playing the most important role (83 VTEC, 41 *Salmonella*, 20 *Cryptosporidium* and 13 *Campylobacter*). However, only (7/167) were water related and a single one (VTEC) had PWS as the suspected vehicle.

Sporadic cases

Sporadic cases of *Campylobacter* and *Cryptosporidium* have been associated with PWS exposure (see Appendix III subsection 4.5 and Appendix V subsection 4.5 respectively). However, for VTEC the data that exists is not supportive of a significant association.

Risk assessments

No risk assessment for *Salmonella*, *Campylobacter* and *Cryptosporidium* from PWS exist in Scotland. However, two risk models were developed for *E. coli* O157 in Scotland (Rotariu et al. 2012) which found that <8% of people contract the disease from PWS.

Risk categorisation

The burden of disease caused by drinking water from private water supplies in Scotland is unknown. There are also no risk models (excepting for *E. coli* O157) which determine the incidence of disease by transmission pathway, source, or any other risk factor. One case-control study for *Campylobacter* in NE Scotland (Smith-Palmer & Cowden 2010) and one generalised linear model for *Cryptosporidium* across Scotland (Pollock et al. 2010) did not offer enough information to perform

risk categorisation. Therefore, the overall incidence, from all sources, for each pathogen was used in this review for risk categorisation (see Appendix VI for details). This puts *Campylobacter* in pole position, followed by viruses and *Salmonella*, then *Cryptosporidium*, with VTEC being classified as the lowest risk. The risk categorisation in terms of the severity of disease from waterborne infections is also unknown.

A.7 Risk management information for private water supplies

Quantitative Microbial Risk Assessment Models use bottom-up approaches, linking the source to the receptor (individuals) via one or more pathways. In terms of risk management, it is of key importance to identify protective barriers to stop the disease. The barriers can act either on source, by reducing the amount of pathogens excreted, on the pathways, trying to control/stop the amount exposure to the pathogens, or on receptors (e.g., by vaccination or by behaviour change to limit exposure), to reduce the incidence of the disease.

7.1 Relevant controls in water

The PWS owner has an obligation under the regulations to ensure that the drinking water is fit to drink and meets the quality standards specified in Schedule 2 (available at www.legislation.gov.uk/ssi/2017/282/contents/made). To that end the PWS owner needs to make sure that adequate catchment management measures are in place to protect the source water. In many cases the source water is ground water and in such cases the principal controls are adequate security of the borehole which will include secure capping of the well head, double barrier fencing and maintenance of the structure to prevent ingress of faecal contamination, small mammals and other carriers of possible microbiological contamination. Surface water sources are more at risk (see DWQR 2020) which shows that groundwater sources, and in particular boreholes, are microbiologically more compliant than surface water sources and would generally require robust protection of the raw water inlet that will include filtration and the establishment of buffer zones around the water intake, to prevent contamination. Risks to surface water abstraction include: contamination from wild deer, small mammals, runoff of farm animal waste, birds, sediments, flooding, etc. The surface water abstraction points will also be at higher risk of *Cryptosporidium* particularly from cross inoculation of the wild deer population in certain areas of Scotland.

7.2 Regulations in Scotland with respect to pathogens/indicator organisms from private water supplies

The regulations governing PWS are “The Water Intended for Human Consumption (Private Supplies)(Scotland) Regulations 2017” (available at www.legislation.gov.uk/ssi/2017/282/contents/made) which in part replace “The Private Water Supply (Scotland) Regulations 2006”. The regulations cover all aspects of PWS ownership and operation, including registration, risk assessments, enforcement, maintenance, and other relevant activities associated with ensuring the drinking water quality. The regulations have associated guidance documents for both the local authorities who enforce the regulations and the PWS owners. The regulations and associated guidance documents are available through the DWQR website, the relevant local authority website, the Scottish Government and Citizens Advice Scotland.

Schedule 2 Part A of the regulations covers the requirements for microbiology and states that the limit for Enterococci and *E. coli* are 0 cfu/100ml. In other words, the water needs to be free of these indicator organisms.

The supply owner is required by the regulations to supply information which covers the risk assessment, remediation action, water quality results and any other information which demonstrates compliance with the regulation. This may include action plans for recovering the quality of water during a failure event. There is an obligation to ensure that the PWS owner manages the full system to ensure safe drinking water. This includes adequate provision for catchment management measures, abstraction, adequate treatment, storage, and distribution of the water. The PWS owner must identify the most appropriate means of mitigating risk to human health.

7.2.1 Water standards

The regulations define the basic water quality standards that need to be maintained to allow the water to be classified as safe to drink. The regulations specify a series of chemical, physical, and microbiological parameters which must be monitored on a regular basis for Regulated private water supplies. The regulations also specify the methods of analysis which helps to ensure that there is confidence in the results produced. Schedules 2 to 4 in the regulations specify the parameter and the values, the monitoring requirements, and the methods of analysis, respectively. All data generated through the monitoring program is reported back to DWQR through the local authorities. Any water quality failure must be investigated, and appropriate mitigations identified and implemented.

7.2.3 Guidance for PWS owners and users

Advice and guidance for owners and users on private water supplies can be found at [mygov.scot⁴](https://www.mygov.scot/housing-local-services/water-supplies-sewerage/private-water-supplies/). This is the centralised location for information on how to look after a domestic or commercial private water supply. The range of information includes everything from applying for a private water supply grant through to risk assessments and testing the private water supplies.

7.3 Adverse economic effects from infection with pathogens from PWS

There is a lack of information on the economic effects of pathogen infection from private water supplies. This is problematic to determine because the annual number of human cases per year for each pathogen is unknown. However, if this were known then it would be possible to determine costs of disease. For example, this could be done by estimating the number of Disability Adjusted Life Years (DALYs) through illness as well as the other costs (healthcare, days lost at work etc.). A DALY can be thought of as one year lost of healthy life. This type of analysis has been performed in the Netherlands for pathogens associated with food and has the potential to be used to determine the costs of disease from PWS in Scotland (Mangen et al. 2015).

7.4 Risk management options

Preventative risk management is critical and the PWS owner working with the community and local authority has an obligation to ensure that identified interventions are applied to the PWS to reduce or eliminate the risk of provision of drinking water that is unfit for consumption. The risk management interventions should be identified through the risk assessments and documents in a water safety plan which will be unique to the individual PWS. The risk management options include but are not limited to:

- Catchment management interventions; for example, reduce livestock in the vicinity of the raw water source. This is underpinned by the data presented earlier in the report on prevalence and shedding of pathogens by farmed and wild animals.
- Adequate barriers and security around the raw water intake; for example, DWQR and the regulations already recommend a protective double fence around the source and boreholes should be properly capped and locked.
- Appropriate treatment to ensure quality (filtration, UV disinfection, etc). This is underpinned by the data

presented on the efficacy of these methods and test results from PWS in Scotland and in other previous studies (e.g., Avery et al. 2016).

- Sealed storage tanks.
- Adequate distribution systems.
- Adequate system maintenance and operational plans. The regulations require all Regulated private water supplies to have been risk assessed with action plans to mitigate risks. It is also a requirement for Type B supplies to be risk assessed.

The effectiveness of the risk interventions needs to be demonstrated through the risk assessments, water safety plans and monitoring programme for each supply. These options also need to be considered in terms of their practicality of implementation, cost and acceptability to users.

A.8 Part A Conclusions

8.1 Risks to Scottish consumers

Campylobacter causes the largest number of reported gastrointestinal cases in Scotland, followed by viruses (norovirus, rotavirus, adenovirus) and then *Salmonella*, with protozoa (*Cryptosporidium*, *Giardia*) and VTEC being comparatively rare. In terms of hospitalisation, the largest number are from *Campylobacter* (8.9 hospital admissions/100,000 people) with the remainder of the gastrointestinal pathogens comprising <2 hospital admissions/100,000 in total.

An accurate estimate of the number of cases caused by these pathogens due to exposure from PWS does not yet exist. However, case control data and QMRA models show that around 8% of *Campylobacter* (NE Scotland) and <8% of *E. coli* O157 cases (across whole of Scotland) may be due to PWS. In addition, outbreaks show that during 2005-2014 only one VTEC outbreak was associated with a PWS in Scotland.

There are several modelling/analytical approaches that can be used to estimate the number of cases due to PWS. These include quantitative risk assessments, quantitative risk factor approaches using regression analysis on disease data, case-control studies, source attribution models and a combination of the aforementioned techniques (Roux et al. 2013; Bessell et al. 2012; Mughini et al. 2012).

Establishing the extent of Scottish PWS contribution to the burden of GI disease needs further investigation, as the quantitative analysis of the risk is still in its infancy. Therefore, there is a need to apply the approaches mentioned above to quantify the contribution of PWS to the human disease burden in Scotland.

⁴ <https://www.mygov.scot/housing-local-services/water-supplies-sewerage/private-water-supplies/>

8.2 Infectious disease transmission from animal reservoirs to water to humans

Understanding the pathways taken by pathogens from animal reservoirs to PWS to human disease is essential in identifying how to manage the risk of illness. There is a growing body of knowledge on the prevalence and concentration of pathogens in both farm and wild animal reservoirs but there are still gaps that need to be filled. There is also progress on understanding and modelling the mechanisms of pathogen transfer from animal faeces, through soil or runoff to surface and ground waters. However, the application of this to PWS in Scotland on a regional or national scale has not yet been attempted. Knowledge exists in part on treatment of water supplies as well as failure rates to indicator organisms. There have been some studies looking for pathogens in PWS but only one in NE Scotland looking at *Campylobacter* was of a reasonable size (>100 PWS tested). In general, the PWS supplies most at risk are surface water abstracted supplies, particularly burns and rivers which are untreated and in areas where there are both animal reservoirs excreting pathogens and mechanisms by which these pathogens can enter the PWS.

Studies have been conducted that have estimated consumption of waters from PWS by humans and dose response models have been developed for the main pathogens of concern. The spectrum of illness for these pathogens is also known in the general population but not for specific PWS associated cases.

The QMRA method can be used to model the transmission of pathogens from animal reservoirs to PWS. This is developed in Part C: "Scope out microbiological risk assessments for gastrointestinal (GI) pathogenic infections from PWS in Scotland".

8.3 Commentary on risk management options

A range of risk management options are available and include catchment management measures, adequate security of the source, adequate treatment; good maintenance and operation, underpinned by appropriate monitoring. The combination and robustness of risk management intervention should be deemed adequate to ensure compliance with The Water Intended for Human Consumption (Private Supplies) (Scotland) Regulations 2017). The risk management plan should address any risks identified during the risk assessment of the supply. The risk assessment should be reviewed on a regular basis to ensure that the any changes to the environment, source, catchment activities, etc., are considered. The risk management plan should then be amended to mitigate the new or emerging risks.

Part B: Human gastrointestinal illness and private water supply failures

B.1 Introduction

Exposure to inadequately treated water from a private water supply is a risk factor for human infection through the gastrointestinal route (Food Standards Scotland 2016)⁵. Public Health Scotland routinely receives information via ECOSS (Electronic Communication of Surveillance in Scotland) (Health Protection Scotland 2020) on all laboratory positive identifications of all organisms covered by the Public Health (Scotland) Act 2008 and a number of other pathogens of public health interest. This includes the gastrointestinal pathogens of interest for this work, mainly shiga toxin producing *E. coli* (STEC) (both O157 and non-O157), *Campylobacter*, *Cryptosporidium* and *Giardia*. However, the ECOSS database does not capture information on exposure factors such as: overseas travel, animal contact or private water supplies, in individuals with these infections. This project aims to look at the links between areas with known private water supply failures and human infection in Scotland between 2009 and 2013.

B.2 Methods

Data on *Campylobacter*, *Cryptosporidium*, *Giardia* and STEC (O157 and non-O157) cases from 2009-2013 were extracted from the ECOSS database. Mean incidence rates for each pathogen were calculated using population data (National Records of Scotland 2019)⁶ for the equivalent years. Using postcodes (where available), ECOSS data was linked to both Regulated private water and Type B PWS failures reported by the DWQR from 2009-2013. Maps were created in Tableau version 10.4 and analysis was undertaken in SPSS® version 24.

⁵ The second study of infectious intestinal disease in the community (IID2 Study). Available at: <https://www.food.gov.uk/research/research-projects/the-second-study-of-infectious-intestinal-disease-in-the-community-iid2-study>

⁶ National Records of Scotland. Available at: <https://www.nrscotland.gov.uk/statistics-and-data/statistics/statistics-by-theme/population/population-estimates/mid-year-population-estimates>

B.3 Results

There was a mean of 6374 cases of *Campylobacter* (120.6 cases per 100,000 population), 557 cases of *Cryptosporidium* (10.5 cases per 100,000 population), 233 cases of *E. coli* O157 (4.4 cases per 100,000 population), 195 *Giardia* (3.7 cases per 100,000 population) and four cases of *E. coli* non-O157 (0.1 cases per 100,000 population) per year during the study period. However, this incidence varied by NHS board location (Table 10). During the study period, *Campylobacter* had a higher incidence in NHS Grampian (210.7 cases per 100,000 population) and NHS Tayside (151.2 cases per 100,000 population), *Cryptosporidium* had a higher incidence in NHS Dumfries and Galloway (22.1 cases per 100,000 population) and NHS Orkney (19.7 cases per 100,000 population), *Giardia* had the highest incidence in NHS Borders (11.4 cases per 100,000 population) and NHS Lothian (7.9 cases per 100,000 population) while *E. coli* had the highest rates in NHS Orkney (19.7 cases per 100,000 population for O157, 91.9 cases per 100,000 population for non-O157) and NHS Dumfries and Galloway (10.6 cases per 100,000 population for O157, 7.0 cases per 100,000 population for non-O157). However, the rates in the Island NHS boards should be viewed with caution due to the impact of the small population size.

Table 10. Incidence (per 10,000) of *Campylobacter*, *Cryptosporidium*, *Giardia*, and *E. coli* in Scotland by NHS board, 2009-2013.

NHS Board	<i>Campylobacter</i>	<i>Cryptosporidium</i>	<i>Giardia</i>	<i>E. coli</i> O157	<i>E. coli</i> non-O157
Ayrshire & Arran	128.9	11.9	4.1	5.5	1.5
Borders	137.0	15.1	11.4	5.6	4.9
Dumfries & Galloway	115.4	22.1	2.8	10.6	7.0
Fife	79.5	11.4	0.6	4.1	1.1
Forth Valley	137.1	6.3	1.1	4.4	1.5
Grampian	210.7	15.4	3.2	7.0	1.2
Greater Glasgow & Clyde	65.5	6.3	1.9	3.3	0.3
Highland	108.7	9.7	1.6	4.3	1.3
Lanarkshire	131.6	10.8	5.5	3.4	0.5
Lothian	131.2	11.0	7.9	2.8	0.3
Orkney	123.7	19.7	3.7	19.7	91.9
Shetland	25.1	2.6	1.7	0.9	3.7
Tayside	151.2	11.4	2.8	5.2	1.3
Western Isles	61.7	4.4	0.0	0.0	0.0
Total	120.6	10.5	3.7	4.4	0.1

Around three quarters (17366/22692; 76.5%) of ECOSS data had a valid postcode available. Of these, 88.0% (15279/17366) had a unique postcode for each pathogen and were able to be mapped (Figure 3).

Results show that human gastrointestinal infections occur in most postcode areas throughout Scotland with infections reported across the central belt, borders, eastern, northern and the island areas of Scotland. *Campylobacter* was reported in the greatest number of postcode areas, however there were a number of postcode areas in Dumfries and Galloway and Highland that had no reports of *Campylobacter*. In contrast, *Cryptosporidium* (although less common) was reported from more postcode areas in Dumfries and Galloway (Figure 3).

Data linkage: ECOSS to Regulated private water PWS failure data

ECOSS and PWS data linkage showed that between 2009 and 2013, 104 cases (0.7%) of human gastrointestinal disease occurred in postcodes that also had a Regulated private water microbiological private water supply failure during the same time period. The most commonly reported pathogens were *Campylobacter* (88 cases; 84.6%) and *Cryptosporidium* (7 cases; 6.7%), (Table 11). During the study period there were 1411 postcodes with a recorded Regulated private water PWS failure, 104 (8.8%) of which had a gastrointestinal infection reported at the same postcode.

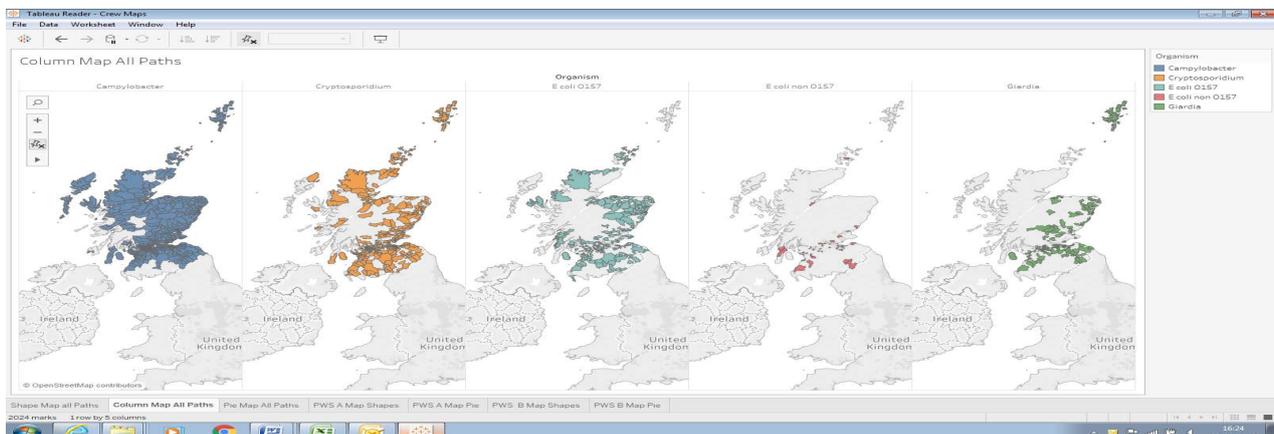


Figure 3. Location of human gastrointestinal infections in Scotland, 2009-2013.

Table 11. Number, and percentage, of cases of gastrointestinal infection linked to Regulated private water PWS microbiological failure, 2009-2013.

Organism	Number of cases linked to Regulated private water PWS failures	Percent of Regulated private water PWS failures	Percent of all ECOSS records
<i>Campylobacter</i> (n=13222)	88	84.6	0.7
<i>Cryptosporidium</i> (n=1063)	7	6.7	0.7
<i>E. coli</i> O157 (n=491)	4	3.8	0.8
<i>Giardia</i> (n=425)	3	2.9	0.7
<i>E. coli</i> non-O157 (n=78)	2	1.9	2.6
Total (n=15279)	104	100	0.7

As a percentage of all ECOSS records with a unique postcode, *E. coli* non-O157 was the most commonly reported pathogen associated with a Regulated private water PWS failure (2/78; 2.6%) (Table 11) and *E. coli* non-O157 cases were more likely (RR=3.8, 95% CI 1.1-12.2, p=0.04) to have a link to a Regulated private water PWS failure than cases with other gastrointestinal infections.

Results (map not presented to preserve anonymity) show that human disease with a matching postcode to Regulated private water PWS failures were more frequently identified in NHS Highland, NHS Grampian and NHS Tayside, which perhaps reflects the higher number of Regulated private water PWS in these areas of Scotland.

Data linkage: ECOSS to Type B PWS failure data

ECOSS data and PWS data linkage showed that between 2009 and 2013, 160 (1.0%) cases of human gastrointestinal disease occurred in postcodes that also had a Type B microbiological private water supply failure during the same time period. The most commonly reported pathogen was *Campylobacter* (122 cases; 76.3%) and *E. coli* O157 (17 cases; 10.6%) (Table 12). During the study period there were 1910 postcodes with a recorded Type B PWS failure, 160 (8.4%) of which had a gastrointestinal infection reported at the same postcode.

As a percentage of all ECOSS records with a unique postcode, *E. coli* non-O157 (3/78; 3.8%) and *E. coli* O157 (15/491; 3.1%) were the most commonly reported pathogen associated with Type B PWS failures (Table 12).

In addition, *E. coli* non-O157 cases (RR=3.7, 95% CI 1.2-11.4, p=0.01) and *E. coli* O157 cases (RR=3.1, 95% CI 1.8-5.3, p<0.01) were more likely to have a Type B PWS failure than cases with other gastrointestinal infections.

Results (map not presented to ensure anonymity) show that human disease with a matching postcode to Type B PWS failures were more frequently identified in NHS Grampian, NHS Highland and NHS Borders, which perhaps reflects the higher number of Type B PWS in these areas of Scotland.

B.4 Part B Conclusions

Gastrointestinal disease is regularly identified in Scotland with *Campylobacter* and *Cryptosporidium* most frequently reported in this study. However, the number and rate of each pathogen varied across the 14 NHS boards and postcode areas. Very few of these human cases of illness had a link via postcode to a reported PWS microbiological failure with 0.7% and 1.0% for Regulated private water and Type B failures, respectively. Where there was a postcode link noted, *Campylobacter* was the most frequently identified pathogen in both PWS failure types.

There was no significant difference observed in the overall level of selected human gastrointestinal infections when comparing Regulated private water PWS with Type B PWS microbiological failures. In addition, there were no significant differences in the distribution of *Campylobacter*, *Cryptosporidium*, *Giardia* and *E. coli*

Table 12. Number, and percentage, of cases of gastrointestinal infection linked to Type B PWS microbiological failure, 2009-2013.

Organism	Number of cases linked to Type B PWS failures	Percent of all Type B PWS failures	Percent of all ECOSS records
<i>Campylobacter</i> (n=13222)	122	76.3	0.9
<i>Cryptosporidium</i> (n=1063)	17	10.6	1.6
<i>E. coli</i> O157 (n=491)	15	9.4	3.1
<i>Giardia</i> (n=425)	3	1.9	0.7
<i>E. coli</i> non-O157 (n=78)	3	1.9	3.8
Total (n=15279)	160	100	1.0

non-O157 between Regulated private water and B PWS failures. However, *E. coli* O157 infection was three times higher amongst Type B water supply failures (17/160; 10.6% versus 4/104; 3.8%, RR=3.0 95% CI 1.2-7.6, $P<0.05$) than Regulated private water PWS failures. In addition, *E. coli* non-O157 cases were four times more likely to have a link to a Regulated private water or Type B PWS failure than cases with other gastrointestinal infections.

Linked cases were more frequent in NHS Highland and NHS Grampian than other NHS boards, possibly reflecting the higher number of PWS in these boards compared to elsewhere in Scotland. It is important to note that as a single postcode will cover multiple households, the case may not necessarily have been in the same household as the PWS microbiological failure, likewise there was insufficient information to determine time parameters between the PWS microbiological failure and onset of infection among the individual.

Removal of the duplicate postcodes with the same pathogen may have accounted for those cases where onward household transmission was believed to have occurred. However, it may also have removed co-primary cases or cases within different households within the same postcode.

No exposure information is collected via ECOSS for these infections, such as: overseas travel, animal contact, environmental exposures, food consumption history, or drinking from a private water supply. Therefore, as well as having a postcode link to a PWS failure, these cases may have acquired their infection through other routes, this is an important point as previous research has shown other risk factors to be strongly associated with particular pathogens, for example, the strong association between consumption of chicken and *Campylobacter* infection. In contrast, a case may have been exposed to a PWS but not in the postcode in which they reside (e.g., when visiting friends and relatives or travelling).

Almost a quarter of the ECOSS data had missing postcodes (23.5%). Some of these cases may have been in areas with a PWS and so this may represent an underestimation of the link to PWS failures. Moreover, the cases captured in ECOSS are those which are laboratory confirmed, under-ascertainment and under-reporting of gastrointestinal disease is well documented⁷; not all cases will seek healthcare, and when healthcare is sought not all cases will have samples taken for laboratory testing and confirmation.

Further work would involve extending the analysis to calculate the rate of gastrointestinal infection within the number of private water supplies (and failures) within each postcode, to assess the risk of infection associated with PWS types and how if the risk differs between those

with failures and those without reported failures. The analysis presented here did not capture the number of microbiological failures that a particular supply had during the study period which could also be factored into any future analysis. Additionally, a time series analysis could be undertaken to look at the time between a PWS failure and human infection, whilst recognising the limitations of such analysis: as the testing date may be determined by the testing cycle of the local authority and not be related to human illness; and the microbial quality of the water at the time of testing may not reflect the quality at the time any individual within the households acquires a gastrointestinal infection. Additional analysis could consider the seasonality of gastrointestinal infection in areas with high incidence of PWS (and PWS with failures) compared to areas without PWS, as the microbial water quality could be impacted by rainfall. In the future, repeating the analysis with more recent PWS and ECOSS data should make the results more current and up to date. Those individuals with PWS are likely to live in rural communities, possibly with direct animal contact. Further analysis could be undertaken with the more recent years data to also include animal density data, as not only may this be a direct factor for human infection but may also, via run off water, contaminate PWS and be a potential source of infection.

7 <https://dwqr.scot/information/annual-report/>

Part C: Scope out microbiological risk assessments for gastrointestinal (GI) pathogenic infections from PWS in Scotland

C.1 Introduction

The purpose of this objective is to identify the main steps and data required to develop quantitative microbial risk assessments (QMRA) for GI pathogens from drinking water from PWS in Scotland. This is done separately for each pathogen of interest: *Salmonella*, *E. coli* O157, *Campylobacter* and *Cryptosporidium*.

This scoping study shows how it can be possible to determine the risk of GI infection from (i) consuming a glass of water drunk from a PWS and (ii) estimation of the number of GI cases, per year, in the Scottish population, attributable to drinking from PWS.

C.2 Approach

The quantitative risk assessment is a process model that involves a number of steps: (1) pathogen sources, (2) transport of pathogen to PWS and types of PWS, (3) survival of pathogen in PWS, (4) treatment of PWS, (5) dose response, (6) consumption and (7) calculation of disease burden in Scotland. Appendices tables A.1 (*Salmonella*), A.2 (*E. coli* O157), A.3 (*Campylobacter*) and A.5 (*Cryptosporidium*) cover steps (1) – (5) for the respective pathogens.

Table A.7 in Appendix VII deals with the risk characterisation, which is the same methodology for all pathogens, and encompasses steps (6) & (7).

C.3 Part C Findings and conclusions

The tables listed above identify those data that are available as well as those that are missing and need to be gathered. They also identify the steps that will require model building (e.g., transport models of pathogen movement from animal sources to PWS etc.). This needs to be addressed for each of the pathogens of interest so that QMRA models can be developed.

In summary, for step 1 (pathogen sources) there are a number of studies that provide data on prevalence and concentration of pathogens in farm animals, however for wild animals the data are generally lacking. There is a lack of data on the prevalence/concentration of pathogens in PWS. It would be expected that the prevalence would be low for pathogens, so a large number of samples would need to be analysed to obtain robust data.

There has been considerable research into the transport processes (step 2) of bacteria (usually indicator organisms) that include detachment, leaching, overland flow etc. This work has the potential to be readily applied to risk assessments of PWS assuming that the relevant topography and hydrological information are known for the particular PWS. There is also available data for survival of these pathogens in water (step 3), though not necessarily in water from PWS.

Regarding information on the proportion of PWS that are treated (step 4), there are data available on this, particularly for Regulated private water supplies, but this is sparser for Type B supplies. Considering the efficacy of the different types of treatment, information is available for UV treatment and also some information is available for filtration and chlorine dosing (see section 5.4.3). However, these data are not complete for all pathogens studied and there is therefore a need to fill the gaps.

Dose response models exist for all of the pathogens that were focussed upon in this report (Step 5). However, it is worth noting that the dose response may vary by subtype but there is a lack of information available in this area at the moment. There are a number of studies that describe the consumption of water from PWS (step 6). The final estimate of the disease burden (step 7) can be generated by the model using the previous 6 steps of the model.

The potential of developing process based quantitative microbiological risk assessment models for PWS has been established. However, there are data at several of the steps of the model, and for a number of the different pathogens, that need to be collected before these models can be successfully implemented.

Conclusions and recommendations

This project sought to develop an understanding of the epidemiology and disease burden contribution of private water supplies on the public health of the populations (indigenous and transient) exposed. This final section of the report provides conclusions and recommendations against the project objectives.

This report provides a review of existing research on disease burden and epidemiology of small rural drinking water supplies (Objective 1) and then extends this with a review of data including existing water quality data and health surveillance reports (Objective 2). An accurate estimate of the number of cases caused by gastrointestinal pathogens due to exposure from PWS does not yet exist. However, case control data and QMRA models show that around 8% of *Campylobacter* (NE Scotland) and <8% of *E. coli* O157 cases (across whole of Scotland) may be due to PWS. In addition, outbreaks show that during 2005-2014 only one VTEC outbreak was associated with a PWS in Scotland. It is recommended that there is a need to:

- Develop and apply the approaches mentioned above, and in more detail in the report, to quantify the contribution of PWS to the human disease burden in Scotland.

In general, the PWS supplies most at risk are surface water abstracted supplies, particularly burns and rivers which are untreated and in areas where there are both animal reservoirs excreting pathogens and mechanisms by which these pathogens can enter the PWS. There have been some studies to detect pathogens in PWS but only one in NE Scotland, looking at *Campylobacter*, was of a reasonable size.

Objective 3 explores linkages between water quality failures and clusters of illness. It was found (Part B) that a low percentage of gastrointestinal illnesses (0.7% for Regulated private water and 1.0% for Type B) were potentially linked to microbiological failures of PWS. It is possible that these linked cases may not have acquired illness from PWS but from other sources (e.g., contact with farm animals or travel abroad). Also, only around one quarter of illnesses could not be included because of lack of postcode information and the analysis could involve only those PWS where microbiological sampling had been conducted. Further analyses of these datasets are readily possible. It is recommended that there is a need to:

- Explore potential approaches outlined in the report for further data analysis e.g., time series analysis to look at the time between a PWS failure and human infection.

Part C reports the scoping out of a Quantitative Microbiological Risk Assessment approach (Objective 4) to assess the disease burden of small rural supplies which can be used to inform future regulation. A spreadsheet model is proposed which would predict the probability of illness from drinking a glass of water from a PWS. The tables compiled as part of this project identify: where existing data can be used as part of the risk assessment and to validate it where appropriate; those data that are missing and need to be collated (e.g., pathogen shedding by wild animals, transport of pathogens from animal faeces to the PWS etc); and a number of steps that will require model building (e.g., transport models of pathogen movement from animal sources to PWS etc.). It is recommended that:

- Risk assessment models (QMRA) be developed which follow the movement of pathogens from animal sources, through the environment, to water supplies and then to the human population.
- Data gaps be addressed for each of the pathogens of interest so that QMRA models can be developed.

Data were collected on the efficacy of PWS treatment methods. However, although some robust data was available for some treatments (e.g., UV) overall there were gaps in the data for the range of pathogens being considered. Dose response model data were available for all of the pathogens under study, but information was not available on how dose response varied by pathogen subtype (e.g., different serotypes of VTEC). Hence, it was not possible at this stage to develop metrics that can be used to measure the effectiveness of treatment of rural water systems and the risk to public health (Objective 5).

Objective 6 involved recommendations to inform improvements to management of private supplies. A number of risk management options for PWS were listed with underpinning evidence where available and these included but are not limited to catchment management interventions; adequate barriers and security around the raw water intake; appropriate treatment to ensure quality (filtration, UV disinfection, etc.); sealed storage tanks; adequate distribution systems; and adequate system maintenance and operational plans. The effectiveness of the risk interventions needs to be demonstrated through the risk assessments, water safety plans and monitoring programme for each supply. These options also need to be considered in terms of their practicality of implementation, cost and acceptability to users. In particular, it is recommended that:

- The combination and robustness of risk management intervention should be deemed adequate to ensure compliance with The Water Intended for Human Consumption (Private Supplies) (Scotland) Regulations 2017.

- The risk assessment and management plan should be reviewed on a regular basis to ensure that any changes to the environment, source, catchment activities, etc., are mitigated against.

In summary there is a need to:

1. Make better use of the available epidemiological information, where relevant to supplement this with additional studies, to obtain realistic estimates of the importance of PWS as a source of human disease.
2. Develop a QMRA model that describes the transmission of pathogens from animal reservoirs to private water supplies from catchment to regional and national scales.
3. Fill data gaps that are required to implement the above model.
4. Use the model to evaluate the efficacy of risk management strategies to help reduce the incidence of disease.

References

- ABBASZADEGAN, M., HASAN, M., GERBA, C., ROESSLER, P., WILSON, E., KUENNEN, R. and VANDELLEN, E., 1997. The disinfection efficacy of a point-of-use water treatment system against bacterial, viral and protozoan waterborne pathogens. *Water Research*, **31**(3), pp. 574-582.
- ADAK, G.K., LONG, S.M. and O'BRIEN, S.J., 2002. Trends in indigenous foodborne disease and deaths, England and Wales: 1992 to 2000. *Gut*, **51**(6), pp. 832-841.
- AGNEW, D., LIMA, A., NEWMAN, R., WUHIB, T., MOORE, R., GUERRANT, R. and SEARS, C., 1998. Cryptosporidiosis in northeastern Brazilian children: Association with increased diarrhoea morbidity. *Journal of Infectious Diseases*, **177**(3), pp. 754-760.
- AGULLO-BARCELO, M., CASAS-MANGAS, R. and LUCENA, F., 2012. Direct and indirect QMRA of infectious *Cryptosporidium* oocysts in reclaimed water. *Journal of Water and Health*, **10**(4), pp. 539-548.
- AHMAD, F., TOURLOUSSE, D.M., STEDTFELD, R.D., SEYRIG, G., HERZOG, A.B., BHADURI, P. and HASHSHAM, S.A., 2009. Detection and occurrence of indicator organisms and pathogens. *Water Environment Research*, **81**(10), pp. 959-980.
- AHMED, W., VIERITZ, A., GOONETILLEKE, A. and GARDNER, T., 2010. Health risk from the use of roof-harvested rainwater in Southeast Queensland, Australia, as potable or nonpotable water, determined using quantitative microbial risk assessment. *Applied and Environmental Microbiology*, **76**(22), pp. 7382-7391.
- AKOUMIANAKI, I., POTTS, J., BAGGIO, A., GIMONA, A., SPEZIA, L., SAMPLE, J., VINTEN, A., AND J. MACDONALD 2016. Developing a method to monitor the rural diffuse pollution plan: providing a framework for interpreting catchment data, CRW2014/13.
- AILES, E., BUDGE, P., SHANKAR, M., COLLIER, S., BRINTON, W., CRONQUIST, A., CHEN, M., THORNTON, A., BEACH, M.J. and BRUNKARD, J.M., 2013. Economic and health impacts associated with a *Salmonella* Typhimurium drinking water outbreak-Alamosa, CO, 2008. *PloS one*, **8**(3), pp. e57439.
- ALLOS, B.M. and BLASER, M.J., 1995. *Campylobacter jejuni* and the expanding spectrum of related infections. *Clinical infectious diseases : an official publication of the Infectious Diseases Society of America*, **20**(5), pp. 1092-9; quiz 1100-1.
- ALTEKRUSE, S.F., STERN, N.J., FIELDS, P.I. and SWERDLOW, D.L., 1999. *Campylobacter jejuni*: an emerging foodborne pathogen. *Emerging infectious diseases (Print); Emerging infectious diseases*, **5**(1), pp. 28-35.
- ALUM, A., ABSAR, I.M., ASAAD, H., RUBINO, J.R. and IJAZ, M.K., 2014. Impact of environmental conditions on the survival of *Cryptosporidium* and *Giardia* on environmental surfaces. *Interdisciplinary perspectives on infectious diseases*, **2014**, pp. 210385.
- AN, W., ZHANG, D., XIAO, S., YU, J. and YANG, M., 2011. *Environmental Science and Technology*, **45**(11), pp. 4951-4958.
- ANONYMOUS, 2000. *New Zealand's Food Safety Risk Management Framework*.
- ANONYMOUS, 2016. Gastro-intestinal and foodborne infections: General outbreaks of infectious intestinal disease reported to HPS during 2005 to 2014 inclusive, annual report.
- ANONYMOUS, 1986. Epidemiologic notes and reports thrombotic thrombocytopenic purpura associated with *E. coli* O157:H7. *MMWR. Morbidity and mortality weekly report*, **35**(34), pp. 549-551.
- ANONYMOUS, 1971. A waterborne epidemic of salmonellosis in Riverside, California, 1965 epidemiologic aspects a collaborative report. *American Journal of Epidemiology*, **93**(1), pp. 33-48.
- ARNOLD, M.E., PAPADOPOULOU, C., DAVIES, R.H., CARRIQUE-MAS, J.J., EVANS, S.J. and HOINVILLE, L.J., 2010. Estimation of *Salmonella* prevalence in UK egg-laying holdings. *Preventive veterinary medicine*, **94**(3-4), pp. 306-309.

- ARNAUD, E., BEST, A., PARKER, B.L., ARAVENA, R. and DUNFIELD, K., 2015. Transport of *Escherichia coli* through a thick vadose zone. *Journal of Environmental Quality*, **44**(5), pp. 1424-1434.
- ARROWOOD, M., 2002. In vitro cultivation of *Cryptosporidium* species. *Clinical microbiology reviews*, **15**(3), pp. 390-+.
- ARTZ, R., TOWNEND, J., BROWN, K., TOWERS, W. and KILLHAM, K., 2005. Soil macropores and compaction control the leaching potential of *Escherichia coli* O157 : H7. *Environmental Microbiology*, **7**(2), pp. 241-248.
- ASHBOLT, N.J., 2004. Microbial contamination of drinking water and disease outcomes in developing regions. *Toxicology*, **198**(1-3), pp. 229-238.
- AULD, H., MACIVER, D. and KLAASSEN, J., 2004. Heavy rainfall and waterborne disease outbreaks: The Walkerton example. *Journal of Toxicology and Environmental Health-Part A-Current Issues*, **67**(20-22), pp. 1879-1887.
- AVERY, L., HOUGH, R., POTTS, J., NEWMAN, G., LILLY, A., LUMSDON, D., ABEL, C., DODD, N., COOK, Y. and MCINTYRE, S., 2016. *Effect of maintenance and different raw water quality parameters on ultraviolet (UV) disinfection in private water supplies in Scotland*. CR/2012/01. Available at: <https://dwqr.scot/media/28687/research-crew-effect-of-maintenance-and-different-raw-water-quality-parameters-on-ultraviolet-uv-disinfection-in-private-water-supplies-in-scotland.pdf> (accessed June 2021)
- BACH, S.J., MCALLISTER, T.A., VEIRA, D.M., GANNON, V.P.J. and HOLLEY, R.A., 2002. Transmission and control of *Escherichia coli* O157 : H7 - A review. *Canadian Journal of Animal Science*, **82**(4), pp. 475-490.
- BAKER, K. and HEGARTY, J., 1997. Detection and occurrence of indicator organisms and pathogens. *Water Environment Research*, **69**(4), pp. 403-415.
- BANMAIRUROY, P., CHAICHANA, P., PULSRIKARN, C. and NUANUALSUWAN, S., 2014. Quantitative microbial risk assessment of *Salmonella* in surface water as a source of tap water. *Thai Journal of Veterinary Medicine*, **44**(1), pp. 95-106.
- BARROW, P.A. and METHNER, U., 2013. *Salmonella in Domestic Animals (2nd Edition)*. CABI Publishing. Wallingford, Oxon, England, UK.
- BARWICK, R.S., MOHAMMED, H.O., WHITE, M.E. and BRYANT, R.B., 2003. Prevalence of *Giardia* spp. and *Cryptosporidium* spp. on dairy farms in southeastern New York state. *Preventive veterinary medicine*, **59**(1-2), pp. 1-11.
- BERN, C., ORTEGA, Y., CHECKLEY, W., ROBERTS, J., LESCANO, A., CABRERA, L., VERASTEGUI, M., BLACK, R., STERLING, C. and GILMAN, R., 2002. Epidemiologic differences between cyclosporiasis and cryptosporidiosis in Peruvian children. *Emerging Infectious Diseases*, **8**(6), pp. 581-585.
- BERRIMAN, A.D., CLANCY, D., CLOUGH, H.E. and CHRISTLEY, R.M., 2013. Semi-stochastic models for *Salmonella* infection within finishing pig units in the UK. *Mathematical biosciences*, **245**(2), pp. 148-156.
- BESSELL, P.R., ROTARIU, O., INNOCENT, G.T., SMITH-PALMER, A., STRACHAN, N.J.C., FORBES, K.J., COWDEN, J.M., REID, S.W.J. and MATTHEWS, L., 2012. Using sequence data to identify alternative routes and risk of infection: a case-study of *Campylobacter* in Scotland. *BMC Infectious Diseases*, **12**, pp. 80.
- BETANCOURT, W.Q. and ROSE, J.B., 2004. Drinking water treatment processes for removal of *Cryptosporidium* and *Giardia*. *Veterinary parasitology*, **126**(1-2), pp. 219-234.
- BEUTIN, L., ZIMMERMANN, S. and GLEIER, K., 1998. Human infections with Shiga toxin-producing *Escherichia coli* other than serogroup O157 in Germany. *Emerging infectious diseases*, **4**(4), pp. 635-639.
- BEVEN, K. and GERMANN, P., 1982. Macropores and Water-Flow in Soils. *Water Resources Research*, **18**(5), pp. 1311-1325.
- BHUNIA, A.J., 2008. *Salmonella enterica*. In: A. BHUNIA, ed, *Foodborne Microbial Pathogens*. pp. 201-216.
- BLACK, R.E., LEVINE, M.M., CLEMENTS, M.L., HUGHES, T.P. and BLASER, M.J., 1988. Experimental *Campylobacter jejuni* infection in humans. *The Journal of infectious diseases*, **157**(3), pp. 472-479.
- BLASER, M.J., 1997. Epidemiologic and clinical features of *Campylobacter jejuni* infections. *The Journal of infectious diseases*, **176** (Suppl 2), pp. S103-105.
- BLASER, M., SMITH, P., WANG, W. and HOFF, J., 1986. Inactivation of *Campylobacter jejuni* by chlorine and monochloramine. *Applied and Environmental Microbiology*, **51**(2), pp. 307-311.

- BLAUSTEIN, R.A., PACHEPSKY, Y.A., HILL, R.L. and SHELTON, D.R., 2015a. Solid manure as a source of faecal indicator microorganisms: release under simulated rainfall. *Environmental Science and Technology*, **49**, pp. 7860-7869.
- BLAUSTEIN, R.A., PACHEPSKY, Y.A., SHELTON, D.R. and HILL, R.L., 2015b. Release and removal of microorganisms from land-deposited animal waste and animal manures: a review of data and models. *Journal of Environmental Quality*, **44**(5), pp. 1338-1354.
- BOES, J., NERSTING, L., NIELSEN, E.M., KRANKER, S., ENOE, C., WACHMANN, H.C. and BAGGESEN, D.L., 2005. Prevalence and diversity of *Campylobacter jejuni* in pig herds on farms with and without cattle or poultry. *Journal of food protection*, **68**(4), pp. 722-727.
- BOLTON, F.J. and COATES, D., 1983. A comparison of microaerobic systems for the culture of *Campylobacter jejuni* and *Campylobacter coli*. *European journal of clinical microbiology*, **2**(2), pp. 105-110.
- BOLTON, F.J., SURMAN, S.B., MARTIN, K., WAREING, D.R. and HUMPHREY, T.J., 1999. Presence of *Campylobacter* and *Salmonella* in sand from bathing beaches. *Epidemiology and infection*, **122**(1), pp. 7-13.
- BONADONNA, L., BRIANCESCO, R., OTTAVIANI, M. and VESCHETTI, E., 2002. Occurrence of *Cryptosporidium* oocysts in sewage effluents and correlation with microbial, chemical and physical water variables. *Environmental monitoring and assessment*, **75**(3), pp. 241-252.
- BOUZID, M., HUNTER, P.R., CHALMERS, R.M. and TYLER, K.M., 2013. *Cryptosporidium* pathogenicity and virulence. *Clinical microbiology reviews*, **26**(1), pp. 115-134.
- BOYER, D.G., KUCZYNSKA, E. and FAYER, R., 2009. Transport, fate, and infectivity of *Cryptosporidium parvum* oocysts released from manure and leached through macroporous soil. *Environmental Geology*, **58**(5), pp. 1011-1019.
- BROPHY, S., JONES, K.H., RAHMAN, M.A., ZHOU, S., JOHN, A., ATKINSON, M.D., FRANCIS, N., LYONS, R.A. and DUNSTAN, F., 2013. Incidence of *Campylobacter* and *Salmonella* infections following first prescription for PPI: A cohort study using routine data. *American Journal of Gastroenterology*, **108**(7), pp. 1094-1100.
- BRENNAN, F.P., KRAMERS, G., GRANT, J., O'FLAHERTY, V., HOLDEN, N.M. and RICHARDS, K., 2012. Evaluating *E. coli* transport risk in soil using dye and bromide tracers. *Soil Science Society of America Journal*, **76**(2), pp. 663-673.
- BROOK, E., HART, C.A., FRENCH, N. and CHRISTLEY, R., 2008. Prevalence and risk factors for *Cryptosporidium* spp. infection in young calves. *Veterinary Parasitology*, **152**(1-2), pp. 46-52.
- BROWNING, L., SMITH-PALMER, A. and BROWNLIE, S., 2015. Gastro-intestinal and foodborne infections: Laboratory reports of *Salmonella* and *Campylobacter* reported to HPS: 2014. *HPS Weekly Report*, **49**, pp. 5-5.
- BUCHANAN, R.L. and BAGI, L.K., 1994. Expansion of response surface models for the growth of *Escherichia coli* O157:H7 to include sodium nitrite as a variable. *International journal of food microbiology*, **23**(3-4), pp. 317-332.
- BUCHELI-WITSCHERL, M., BASSIN, C. and EGLI, T., 2010. UV-C inactivation in *Escherichia coli* is affected by growth conditions preceding irradiation, in particular by the specific growth rate. *Journal of applied microbiology*, **109**(5), pp. 1733-1744.
- BUKHARI, Z., HARGY, T., BOLTON, J., DUSSERT, B. and CLANCY, J., 1999. Medium-pressure UV for oocyst inactivation. *Journal American Water Works Association*, **91**(3), pp. 86-94.
- BUKHARI, Z., SMITH, H., SYKES, N., HUMPHREYS, S., PATON, C., GIRDWOOD, R. and FRICKER, C., 1997. Occurrence of *Cryptosporidium* spp oocysts and *Giardia* spp cysts in sewage influents and effluents from treatment plants in England. *Water Science and Technology*, **35**(11-12), pp. 385-390.
- BULL, S.A., ALLEN, V.M., DOMINGUE, G., JORGENSEN, F., FROST, J.A., URE, R., WHYTE, R., TINKER, D., CORRY, J.E., GILLARD-KING, J. and HUMPHREY, T.J., 2006. Sources of *Campylobacter* spp. colonizing housed broiler flocks during rearing. *Applied and Environmental Microbiology*, **72**(1), pp. 645-652.
- CASADEVALL, A. and PIROFSKI, L.A., 2018. What Is a Host? Attributes of Individual Susceptibility. *Infection and immunity*, **86**(2), pp. 10.1128/IAI.00636-17. Print 2018 Feb.

- CHALMERS, R.M., 2008. *Cryptosporidium*: From laboratory diagnosis to surveillance and outbreaks. *Parasite-Journal De La Societe Francaise De Parasitologie*, **15**(3), pp. 372-378.
- CHECKLEY, W., WHITE, A.C., Jr, JAGANATH, D., ARROWOOD, M.J., CHALMERS, R.M., CHEN, X.M., FAYER, R., GRIFFITHS, J.K., GUERRANT, R.L., HEDSTROM, L., HUSTON, C.D., KOTLOFF, K.L., KANG, G., MEAD, J.R., MILLER, M., PETRI, W.A., Jr, PRIEST, J.W., ROOS, D.S., STRIPEIN, B., THOMPSON, R.C., WARD, H.D., VAN VOORHIS, W.A., XIAO, L., ZHU, G. and HOUP, E.R., 2015. A review of the global burden, novel diagnostics, therapeutics, and vaccine targets for *Cryptosporidium*. *The Lancet Infectious diseases*, **15**(1), pp. 85-94.
- CHEN, R.Z., CRAIK, S.A. and BOLTON, J.R., 2009. Comparison of the action spectra and relative DNA absorbance spectra of microorganisms: Information important for the determination of germicidal fluence (UV dose) in an ultraviolet disinfection of water. *Water Research*, **43**(20), pp. 5087-5096.
- CLAVERO, M.R. and BEUCHAT, L.R., 1996. Survival of *Escherichia coli* O157:H7 in broth and processed salami as influenced by pH, water activity, and temperature and suitability of media for its recovery. *Applied and Environmental Microbiology*, **62**(8), pp. 2735-2740.
- CODEX, 2007. *Working principles for risk analysis for food safety for application by governments*. CAC/GL 62-2007.
- COIA, J.E., 1998. Clinical, microbiological and epidemiological aspects of *Escherichia coli* O157 infection. *FEMS immunology and medical microbiology*, **20**(1), pp. 1-9.
- COLLES, F.M., JONES, K., HARDING, R.M. and MAIDEN, M.C., 2003. Genetic diversity of *Campylobacter jejuni* isolates from farm animals and the farm environment. *Applied and Environmental Microbiology*, **69**(12), pp. 7409-7413.
- COLLINS, R., ELLIOTT, S. and ADAMS, R., 2005. Overland flow delivery of faecal bacteria to a headwater pastoral stream. *Journal of applied microbiology*, **99**(1), pp. 126-132.
- CONNELLY, L., CRAIG, B.H., JONES, B. and ALEXANDER, C.L., 2013. Genetic Diversity of *Cryptosporidium* spp. within a Remote Population of Soay Sheep on St. Kilda Islands, Scotland. *Applied and Environmental Microbiology*, **79**(7), pp. 2240-2246.
- CONNER, D.E. and KOTROLA, J.S., 1995. Growth and survival of *Escherichia coli* O157:H7 under acidic conditions. *Applied and Environmental Microbiology*, **61**(1), pp. 382-385.
- CRABTREE, K., GERBA, C., ROSE, J. and HAAS, C., 1997. Waterborne adenovirus: A risk assessment. *Water Science and Technology*, **35**(11-12), pp. 1-6.
- CRAIK, S., FINCH, G., BOLTON, J. and BELOSEVIC, M., 2000. Inactivation of *Giardia muris* cysts using medium-pressure ultraviolet radiation in filtered drinking water. *Water research*, **34**(18), pp. 4325-4332.
- CRAWFORD, F.G. and VERMUND, S.H., 1988. Human cryptosporidiosis. *Critical reviews in microbiology*, **16**(2), pp. 113-159.
- CURRENT, W.L. and GARCIA, L.S., 1991. Cryptosporidiosis. *Clinical microbiology reviews*, **4**(3), pp. 325-358.
- DALLMAN, T.J., ASHTON, P.M., BYRNE, L., PERRY, N.T., PETROVSKA, L., ELLIS, R., ALLISON, L., HANSON, M., HOLMES, A., GUNN, G.J., CHASE-TOPPING, M.E., WOOLHOUSE, M.E., GRANT, K.A., GALLY, D.L., WAIN, J. and JENKINS, C., 2015. Applying phylogenomics to understand the emergence of Shiga-toxin-producing *Escherichia coli* O157:H7 strains causing severe human disease in the UK. *Microbial genomics*, **1**(3), pp. e000029.
- DAVIES-COLLEY, R., NAGELS, J., SMITH, R., YOUNG, R. and PHILLIPS, C., 2004. Water quality impact of a dairy cow herd crossing a stream. *New Zealand Journal of Marine and Freshwater Research*, **38**(4), pp. 569-576.
- DE HAAN, C.P., LAMPEN, K., CORANDER, J. and HANNINEN, M.L., 2013. Multilocus sequence types of environmental *Campylobacter jejuni* isolates and their similarities to those of human, poultry and bovine *C. jejuni* isolates. *Zoonoses and public health*, **60**(2), pp. 125-133.
- DESHPANDE, A., ALEXANDER, C.L., COYNE, M., BROWNLIE, S., SMITH-PALMER, A. and JONES, B.L., 2015a. Molecular diversity of Scottish *Cryptosporidium hominis* isolates. *Epidemiology and infection*, **143**(6), pp. 1219-1224.

- DESHPANDE, A.P., JONES, B.L., CONNELLY, L., POLLOCK, K.G., BROWNLIE, S. and ALEXANDER, C.L., 2015b. Molecular characterization of *Cryptosporidium parvum* isolates from human cryptosporidiosis cases in Scotland. *Parasitology*, **142**(2), pp. 318-325.
- DEV, V.J., MAIN, M. and GOULD, I., 1991. Waterborne outbreak of *Escherichia coli* O157. *Lancet (London, England)*, **337**(8754), pp. 1412.
- DONNENBERG, M.S. and WHITTAM, T.S., 2001. Pathogenesis and evolution of virulence in enteropathogenic and enterohemorrhagic *Escherichia coli*. *The Journal of clinical investigation*, **107**(5), pp. 539-548.
- DROPPO, I.G., LISS, S.N., WILLIAMS, D., NELSON, T., JASKOT, C. and TRAPP, B., 2009. Dynamic existence of waterborne pathogens within river sediment compartments. Implications for Water Quality Regulatory Affairs. *Environmental science & technology*, **43**(6), pp. 1737-1743.
- DUKE, L.A., BREATHNACH, A.S., JENKINS, D.R., HARKIS, B.A. and CODD, A.W., 1996. A mixed outbreak of *Cryptosporidium* and *Campylobacter* infection associated with a private water supply. *Epidemiology and infection*, **116**(3), pp. 303-308.
- DUNDAS, S., TODD, W.T., STEWART, A.I., MURDOCH, P.S., CHAUDHURI, A.K. and HUTCHINSON, S.J., 2001. The central Scotland *Escherichia coli* O157:H7 outbreak: risk factors for the hemolytic uremic syndrome and death among hospitalized patients. *Clinical infectious diseases : an official publication of the Infectious Diseases Society of America*, **33**(7), pp. 923-931.
- DUPONT, H.L., CHAPPELL, C.L., STERLING, C.R., OKHUYSEN, P.C., ROSE, J.B. and JAKUBOWSKI, W., 1995. The infectivity of *Cryptosporidium parvum* in healthy volunteers. *The New England journal of medicine*, **332**(13), pp. 855-859.
- DWQR, 2018. *Drinking Water Quality in Scotland 2017, Private Water Supplies*. 1.
- DWQR, 2020. DWQR Annual report 2019. (<https://dwqr.scot/information/annual-report/> accessed June 2021)
- ECDC, 2007. *The first European Communicable Disease Epidemiological Report*.
- EVANS, M.R., RIBEIRO, C.D. and SALMON, R.L., 2003. Hazards of healthy living: bottled water and salad vegetables as risk factors for *Campylobacter* infection. *Emerging infectious diseases*, **9**(10), pp. 1219-1225.
- EVANS, M.R., ROBERTS, R.J., RIBEIRO, C.D., GARDNER, D. and KEMBREY, D., 1996. A milk-borne *Campylobacter* outbreak following an educational farm visit. *Epidemiology and infection*, **117**(3), pp. 457-462.
- EVANS, M. and OWENS, J., 1972. Factors affecting concentration of fecal bacteria in land-drainage water. *Journal of General Microbiology*, **71**(AUG), pp. 477-&.
- FAYER, R., 1994. Effect of high temperature on infectivity of *Cryptosporidium parvum* oocysts in water. *Applied and Environmental Microbiology*, **60**(8), pp. 2732-2735.
- FERNANDEZ, T.F., 2008. *E. coli* O157:H7. *Veterinary World*, **1**(3), pp. 83-87.
- FLANIGAN, T., WHALEN, C., TURNER, J., SOAVE, R., TOERNER, J., HAVLIR, D. and KOTLER, D., 1992. *Cryptosporidium* infection and cd4 counts. *Annals of Internal Medicine*, **116**(10), pp. 840-842.
- FUECHSLIN, H.P., KOETZSCH, S. and EGLI, T., 2012. *Cryptosporidium* spp. in drinking water. *Swiss Medical Weekly*, **142**, pp. w13683.
- GALE, P., 2001. Developments in microbiological risk assessment for drinking water. *Journal of applied microbiology*, **91**(2), pp. 191-205.
- GALLAS-LINDEMANN, C., SOTIRIADOU, I., PLUTZER, J. and KARANIS, P., 2013. Prevalence and distribution of *Cryptosporidium* and *Giardia* in wastewater and the surface, drinking and ground waters in the Lower Rhine, Germany. *Epidemiology and infection*, **141**(1), pp. 9-21.

- GAVRIEL, A.A., LANDRE, J.P. and LAMB, A.J., 1998. Incidence of mesophilic *Aeromonas* within a public drinking water supply in north-east Scotland. *Journal of applied microbiology*, **84**(3), pp. 383-392.
- GAYAN, E., SERRANO, M.J., RASO, J., ALVAREZ, I. and CONDON, S., 2012. Inactivation of *Salmonella enterica* by UV-C light alone and in combination with mild temperatures. *Applied and Environmental Microbiology*, **78**(23), pp. 8353-8361.
- GIBBONS, D.W., REID, J.B. and CHAPMAN, R.A., 1993. *The new atlas of breeding birds in Britain and Ireland: 1988-1991*. London, UK: T & AD Poyser Ltd.
- GIL PRIETO, R., GOMEZ ALEJANDRE, C., ALVARO MECA, A., HERNANDEZ BARRERA, V. and GIL DE MIGUEL, A., 2009. Epidemiology of hospital-treated *Salmonella* infection; Data from a national cohort over a ten-year period. *Journal of Infection*, **58**(3), pp. 175-181.
- GONZALEZ, M. and HANNINEN, M.L., 2012. Effect of temperature and antimicrobial resistance on survival of *Campylobacter jejuni* in well water: application of the Weibull model. *Journal of applied microbiology*, **113**(2), pp. 284-293.
- GRIEKSPoor, P., COLLES, F.M., MCCARTHY, N.D., HANSBRO, P.M., ASHHURST-SMITH, C., OLSEN, B., HASSELQUIST, D., MAIDEN, M.C.J. and WALDENSTROM, J., 2013. Marked host specificity and lack of phylogeographic population structure of *Campylobacter jejuni* in wild birds. *Molecular ecology*, **22**(5), pp. 1463-1472.
- GRONDAHL-ROSADO, R.C., YAROVITSYNA, E., TRETENES, E., MYRMEL, M. and ROBERTSON, L.J., 2014. A one year study on the concentrations of norovirus and enteric Adenoviruses in wastewater and a surface drinking water source in Norway. *Food and environmental virology*, **6**(4), pp. 232-245.
- GUBER, A.K., PACHEPSKY, Y.A., SHELTON, D.R. and YU, O., 2009. Association of fecal coliforms with soil aggregates: effect of water content and bovine manure application. *Soil Science*, **174**(10), pp. 543-548.
- GUBER, A.K., FRY, J., IVES, R.L. and ROSE, J.B., 2015. *Escherichia coli* survival in, and release from, white-tailed deer feces. *Applied and Environmental Microbiology*, **81**(3), pp. 1168-1176.
- HAAS, C.N., 2000. Epidemiology, microbiology, and risk assessment of waterborne pathogens including *Cryptosporidium*. *Journal of food protection*, **63**(6), pp. 827-831.
- HAAS, C.N., 1983. Estimation of risk due to low doses of microorganisms: a comparison of alternative methodologies. *American Journal of Epidemiology*, **118**(4), pp. 573-582.
- HAAS, C.N., ROSE, J.B., GERBA, C. and REGLI, S., 1993. Risk assessment of virus in drinking water. *Risk analysis : an official publication of the Society for Risk Analysis*, **13**(5), pp. 545-552.
- HAAS, C.N., THAYYAR-MADABUSI, A., ROSE, J.B. and GERBA, C.P., 2000. Development of a dose-response relationship for *Escherichia coli* O157:H7. *International journal of food microbiology*, **56**(2-3), pp. 153-159.
- HAGEDORN, C., HANSEN, D. and SIMONSON, G., 1978. Survival and movement of fecal indicator bacteria in soil under conditions of saturated flow. *Journal of Environmental Quality*, **7**(1), pp. 55-59.
- HANES, D.E., LORENZO, M.E., HARPER, S.B., SAYLOR, M.L., OFOSU, N.O. and TALL, E., 2003. The food matrix: Effects on infection with *Salmonella enterica* serotype typhimurium. *Abstracts of the General Meeting of the American Society for Microbiology 2003*, pp. P-083.
- HEALTH PROTECTION SCOTLAND 2020. Electronic communication of surveillance in scotland <https://www.hps.scot.nhs.uk/data/> (accessed June 2021)
- HIJNEN, W.A., BEERENDONK, E.F. and MEDEMA, G.J., 2006. Inactivation credit of UV radiation for viruses, bacteria and protozoan (oo)cysts in water: a review. *Water research*, **40**(1), pp. 3-22.
- HOKAJARVI, A., PITKANEN, T., MERILAINEN, P., KAUPPINEN, A., MATIKKA, V., KOVANEN, S., VEPSALAINEN, A. and MIETTINEN, I.T., 2018. Determination of Removal Efficiencies for *Escherichia coli*, Clostridial Spores, and F-Specific Coliphages in Unit Processes of Surface Waterworks for QMRA Applications. *Water*, **10**(11), pp. 1525.

- HOLCOMB, D.L., SMITH, M.A., WARE, G.O., HUNG, Y.C., BRACKETT, R.E. and DOYLE, M.P., 1999. Comparison of six dose-response models for use with food-borne pathogens. *Risk analysis : an official publication of the Society for Risk Analysis*, **19**(6), pp. 1091-1100.
- HUNTER, P.R., 1993. The microbiology of bottled natural mineral waters. *The Journal of applied bacteriology*, **74**(4), pp. 345-352.
- HUNTER, P.R., DE SYLOR, M.A., RISEBRO, H.L., NICHOLS, G.L., KAY, D. and HARTEMANN, P., 2011. Quantitative Microbial Risk Assessment of Cryptosporidiosis and Giardiasis from Very Small Private Water Supplies. *Risk Analysis*, **31**(2), pp. 228-236.
- HYNDS, P.D., GILL, L.W. and MISSTEAR, B.D., 2014. A Quantitative Risk Assessment of Verotoxigenic *E. coli* (VTEC) in Private Groundwater Sources in the Republic of Ireland. *Human and Ecological Risk Assessment*, **20**(6), pp. 1446-1468.
- INNOCENT, G.T., MELLOR, D.J., MCEWEN, S.A., REILLY, W.J., SMALLWOOD, J., LOCKING, M.E., SHAW, D.J., MICHEL, P., TAYLOR, D.J., STEELE, W.B., GUNN, G.J., TERNENT, H.E., WOOLHOUSE, M.E., REID, S.W. and WELLCOME TRUST-FUNDED IPRAVE CONSORTIUM, 2005. Spatial and temporal epidemiology of sporadic human cases of *Escherichia coli* O157 in Scotland, 1996-1999. *Epidemiology and infection*, **133**(6), pp. 1033-1041.
- JAMIESON, R., GORDON, R., SHARPLES, K.E., STRATTON, G.W. and MADANI, A., 2002. Movement and persistence of fecal bacteria in agricultural soils and subsurface drainage water: A review. *Canadian Biosystems Engineering*, **44**(1), pp. 1-9.
- JOHN, D.E. and ROSE, J.B., 2005. Review of factors affecting microbial survival in groundwater. *Environmental science & technology*, **39**(19), pp. 7345-7356.
- JOHNSON, D.C., ENRIQUEZ, C.E., PEPPER, I.L., DAVIS, T.L., GERBA, C.P. and ROSE, J.B., 1997. Survival of *Giardia*, *Cryptosporidium*, poliovirus and *Salmonella* in marine waters. *Water Science and Technology*, **35**, pp. 261-268.
- JONES, K., 2001. *Campylobacter* in water, sewage and the environment. *Journal of Applied Microbiology*, **90**, pp. 1-12.
- JULIO, C., SA, C., FERREIRA, I., MARTINS, S., OLEASTRO, M., ANGELO, H., GUERREIRO, J. and TENREIRO, R., 2012. Waterborne transmission of *Giardia* and *Cryptosporidium* at river beaches in Southern Europe (Portugal). *Journal of Water and Health*, **10**(3), pp. 484-496.
- KAPER, J.B., NATARO, J.P. and MOBLEY, H.L., 2004. Pathogenic *Escherichia coli*. *Nature reviews. Microbiology*, **2**(2), pp. 123-140.
- KARAGIANNIS, I., SIDEROGLOU, T., GKOLFINOPOULOU, K., TSOURI, A., LAMPOUSAKI, D., VELONAKIS, E.N., SCOULICA, E.V., MELLOU, K., PANAGIOTOPOULOS, T. and BONOVAS, S., 2010. A waterborne *Campylobacter jejuni* outbreak on a Greek island. *Epidemiology and infection*, **138**(12), pp. 1726-1734.
- KARANIS, P. and ALDEYARBI, H.M., 2011. Evolution of *Cryptosporidium* in vitro culture. *International journal for parasitology*, **41**(12), pp. 1231-1242.
- KARANIS, P., KOURENTI, C. and SMITH, H., 2007. Waterborne transmission of protozoan parasites: A worldwide review of outbreaks and lessons learnt. *Journal of Water and Health*, **5**(1), pp. 1-38.
- KARMALI, M.A., PETRIC, M., LIM, C., FLEMING, P.C., ARBUS, G.S. and LIOR, H., 1985. The association between idiopathic hemolytic uremic syndrome and infection by verotoxin-producing *Escherichia coli*. *The Journal of infectious diseases*, **151**(5), pp. 775-782.
- KARMALI, M., MASCARENHAS, M., SHEN, S., ZIEBELL, K., JOHNSON, S., REID-SMITH, R., ISAAC-RENTON, J., CLARKS, C., RAHN, K. and KAPER, J., 2003. Association of genomic O(-)island 122 of *Escherichia coli* EDL 933 with verocytotoxin-producing *Escherichia coli* seropathotypes that are linked to epidemic and/or serious disease. *Journal of Clinical Microbiology*, **41**(11), pp. 4930-4940.

- KASSEM, I.I., SANAD, Y., GANGAIAH, D., LILBURN, M., LEJEUNE, J. and RAJASHEKARA, G., 2010. Use of bioluminescence imaging to monitor *Campylobacter* survival in chicken litter. *Journal of applied microbiology*, **109**(6), pp. 1988-1997.
- KAY, D., CROWTHER, J., STAPLETON, C.M., WYER, M.D., FEWTRELL, L., ANTHONY, S., BRADFORD, M., EDWARDS, A., FRANCIS, C.A., HOPKINS, M., KAY, C., MCDONALD, A.T., WATKINS, J. and WILKINSON, J., 2008. Faecal indicator organism concentrations and catchment export coefficients in the UK. *Water Research*, **42**(10-11), pp. 2649-2661.
- KAY, D., WATKINS, J., FRANCIS, C.A., WYN-JONES, A.P., STAPLETON, C.M., FEWTRELL, L., WYER, M.D. and DRURY, D., 2007. The microbiological quality of seven large commercial private water supplies in the United Kingdom. *Journal of Water and Health*, **5**(4), pp. 523-538.
- KHAN, W.A., ROGERS, K.A., KARIM, M.M., AHMED, S., HIBBERD, P.L., CALDERWOOD, S.B., RYAN, E.T. and WARD, H.D., 2004. Cryptosporidiosis among Bangladeshi children with diarrhea: a prospective, matched, case-control study of clinical features, epidemiology and systemic antibody responses. *The American Journal of Tropical Medicine and Hygiene*, **71**(4), pp. 412-419.
- KIM, J., PACHEPSKY, Y.A., SHELTON, D.R. and COPPOCK, C., 2010. Effect of streambed bacteria release on *E. coli* concentrations: Monitoring and modeling with the modified SWAT. *Ecological Modelling*, **221**(12), pp. 1592-1604.
- KING, B.J., HOEFEL, D., DAMINATO, D.P., FANOK, S. and MONIS, P.T., 2008. Solar UV reduces *Cryptosporidium parvum* oocyst infectivity in environmental waters. *Journal of applied microbiology*, **104**(5), pp. 1311-1323.
- KING, B.J. and MONIS, P.T., 2007. Critical processes affecting *Cryptosporidium* oocyst survival in the environment. *Parasitology*, **134**(3), pp. 309-323.
- KIRCHNER, M., MCLAREN, I., CLIFTON-HADLEY, F.A., LIEBANA, E., WALES, A.D. and DAVIES, R.H., 2012. A comparison between longitudinal shedding patterns of *Salmonella* Typhimurium and *Salmonella* Dublin on dairy farms. *The Veterinary record*, **171**(8), pp. 194.
- KORTBEEK, L.M., 2009. *Clinical Presentation in Cryptosporidium-infected Patients*. CABI Publishing, Wallingford, Oxon, England, UK.
- KOTHARY, M. and BABU, U., 2001. Infective dose of foodborne pathogens: A review. *J. Food Safety*, **21**(1), pp. 49-73.
- LAKE, R., HUDSON, A., CRESSEY, P. and GILBERT, S., 2005. *Risk profile: Listeria monocytogenes in low moisture cheeses*. Institute of Environmental Science & Research Limited Christchurch Science Centre (ESR). FW0440.
- LAKE, R., BAKER, M., GARRETT, N., SCOTT, W. and SCOTT, H., 2000. Estimated number of cases of foodborne infectious disease in New Zealand. *New Zealand Medical Journal*, **113**(1113), pp. 278-281.
- LANE, C.R., LEBAGUE, S., ESAN, O.B., AWOFISYO, A.A., ADAMS, N.L., FISHER, I.S., GRANT, K.A., PETERS, T.M., LARKIN, L., DAVIES, R.H. and ADAK, G.K., 2014. *Salmonella* enterica serovar Enteritidis, England and Wales, 1945-2011. *Emerging infectious diseases*, **20**(7), pp. 1097-1104.
- LATIMER, H.K., JAYKUS, L.A., MORALES, R.A., COWEN, P. and CRAWFORD-BROWN, D., 2001. A weighted composite dose-response model for human salmonellosis. *Risk analysis : an official publication of the Society for Risk Analysis*, **21**(2), pp. 295-305.
- LAUBACH, H.E., BENTLEY, C.Z., GINTER, E.L., SPALTER, J.S. and JENSEN, L.A., 2004. A study of risk factors associated with the prevalence of *Cryptosporidium* in villages around Lake Atitlan, Guatemala. *The Brazilian journal of infectious diseases : an official publication of the Brazilian Society of Infectious Diseases*, **8**(4), pp. 319-323.
- LEACH, C.T., KOO, F.C., KUHL, T.L., HILSENBECK, S.G. and JENSON, H.B., 2000. Prevalence of *Cryptosporidium parvum* infection in children along the Texas-Mexico border and associated risk factors. *The American Journal of Tropical Medicine and Hygiene*, **62**(5), pp. 656-661.
- LEE, M.K., BILLINGTON, S.J. and JOENS, L.A., 2004. Potential Virulence and Antimicrobial Resistance in *Campylobacter jejuni* Isolates Obtained from Food and Companion Animals. *Foodborne Pathogenic Diseases*, **1**(4), pp. 223-230.

- LEVALLOIS, P., CHEVALIER, P., GINGRAS, S., DERY, P., PAYMENT, P., MICHEL, P. and RODRIGUEZ, M., 2014. Risk of infectious gastroenteritis in young children living in Quebec rural areas with intensive animal farming: results of a case-control study (2004-2007). *Zoonoses and Public Health*, **61**(1), pp. 28-38.
- LEVANTESI, C., BONADONNA, L., BRIANCESCO, R., GROHMANN, E., TOZE, S. and TANDOI, V., 2012. *Salmonella* in surface and drinking water: Occurrence and water-mediated transmission. *Food Research International*, **61**(1), pp. 28-38.
- LICENCE, K., OATES, K.R., SYNGE, B.A. and REID, T.M., 2001. An outbreak of *E. coli* O157 infection with evidence of spread from animals to man through contamination of a private water supply. *Epidemiology and Infection*, **126**(1), pp. 135-138.
- LINDEN, K., SHIN, G., FAUBERT, G., CAIRNS, W. and SOBSEY, M., 2002. UV disinfection of *Giardia lamblia* cysts in water. *Environmental Science & Technology*, **36**(11), pp. 2519-2522.
- LI, R.A., MCDONALD, J.A., SATHASIVAN, A., KHAN, S.J., 2019. Disinfectant residual stability leading to disinfectant decay and by-product formation in drinking water distribution systems: a systematic review. *Water Research*, **153**, pp. 335-348.
- LIU, G., LING, F.Q., VAN DER MARK, E.J., ZHANG, X.D., KNEZEV, A., VERBERK, J.Q., VAN DER MEER, W.G., MEDEMA, G.J., LIU, W.T. and VAN DIJK, J.C., 2016. Comparison of particle-associated bacteria from a drinking water treatment plant and distribution reservoirs with different water sources. *Scientific reports*, **6**, pp. 20367.
- LOCKING, M., ALLISON, L., RAE, L., POLLOCK, K. and HANSON, M., 2006. VTEC in Scotland 2004: Enhanced surveillance and reference laboratory data *Health Protection Scotland Weekly Report*, **39** (51-52), pp. 290-295.
- LOCKING, M., BROWNING, L., SMITH-PALMER, A. and BROWNLIE, S., 2014. Gastro-intestinal and foodborne infections: Laboratory reports of *E. coli* O157, *Salmonella* and *Campylobacter* reported to HPS: 2013. *HPS Weekly report*, **48**.
- LOCKING, M. and COWDEN, J., 2009. *Escherichia coli* O157. *BMJ (Clinical research ed.)*, **339**, pp. b4076.
- LOCKING, M.E., O'BRIEN, S.J., REILLY, W.J., WRIGHT, E.M., CAMPBELL, D.M., COIA, J.E., BROWNING, L.M. and RAMSAY, C.N., 2001. Risk factors for sporadic cases of *Escherichia coli* O157 infection: the importance of contact with animal excreta. *Epidemiology and Infection*, **127**(2), pp. 215-220.
- LYNN, R.M., O'BRIEN, S.J., TAYLOR, C.M., ADAK, G.K., CHART, H., CHEASTY, T., COIA, J.E., GILLESPIE, I.A., LOCKING, M.E., REILLY, W.J., SMITH, H.R., WATERS, A. and WILLSHAW, G.A., 2005. Childhood hemolytic uremic syndrome, United Kingdom and Ireland. *Emerging Infectious Diseases*, **11**(4), pp. 590-596.
- MACDONALD, I.A., GOULD, I.M. and CURNOW, J., 1996. Epidemiology of infection due to *Escherichia coli* O157: a 3-year prospective study. *Epidemiology and Infection*, **116**(3), pp. 279-284.
- MACRITCHIE, L., HUNTER, C. and STRACHAN, N.J.C., 2013. A population based exposure assessment of risk factors associated with gastrointestinal pathogens: a *Campylobacter* study. *Epidemiology and Infection*, **141**, pp. 97-986.
- MAGNI, V.M., 2010. *Detection of Bacteria, Viruses, Parasites and Fungi*. Dordrecht, Netherlands: Springer.
- MANGEN, M.J., BOUWKNEGT, M., FRIESEMA, I.H.M., HAAGSMA, J.A., KORTBEEK, L.M., TARIQ, L., WILSON, M., VAN PELT, W. and HAVELAAR, A.H., 2015. Cost-of-illness and disease burden of food-related pathogens in the Netherlands, 2011. *International Journal of Food Microbiology*, **196**, pp. 84-93.
- MARIER, E.A., SNOW, L.C., FLOYD, T., MCLAREN, I.M., BIANCHINI, J., COOK, A.J.C. and DAVIES, R.H., 2014. Abattoir based survey of *Salmonella* in finishing pigs in the United Kingdom 2006-2007. *Preventive Veterinary Medicine*, **117**(3-4), pp. 542-553.
- MARTINS, J.M.F., MAJDALANI, S., VITORGE, E., DESAUNAY, A., NAVEL, A., GUINE, V., DAIAN, J.F., VINCE, E., DENIS, H. and GAUDET, J.P., 2013. Role of macropore flow in the transport of *Escherichia coli* cells in undisturbed cores of a brown leached soil. *Environmental Science-Processes & Impacts*, **15**(2), pp. 347-356.

- MATARAGAS, M., SKANDAMIS, P.N. and DROSINOS, E.H., 2008. Risk profiles of pork and poultry meat and risk ratings of various pathogen/product combinations. *International Journal of Food Microbiology*, **126**(1-2), pp. 1-12.
- MAUNULA, L., MIETTINEN, I.T. and VON BONSDORFF, C.H., 2005. Norovirus outbreaks from drinking water. *Emerging Infectious Diseases*, **11**(11), pp. 1716-1721.
- MAWDSLEY, J., BARDGETT, R., MERRY, R., PAIN, B. and THEODOROU, M., 1995. Pathogens in livestock waste, their potential for movement through soil and environmental-pollution. *Applied Soil Ecology*, **2**(1), pp. 1-15.
- MCCULLOUGH, N.B. and EISELE, C.W., 1951a. Experimental human salmonellosis. I. Pathogenicity of strains of *Salmonella* meleagridis and *Salmonella* anatum obtained from spray-dried whole egg. *The Journal of Infectious Diseases*, **88**(3), pp. 278-289.
- MCCULLOUGH, N.B. and EISELE, C.W., 1951b. Experimental human salmonellosis. II. Immunity studies following experimental illness with *Salmonella* meleagridis and *Salmonella* anatum. *Journal of Immunology (Baltimore, Md.: 1950)*, **66**(5), pp. 595-608.
- MCFETERS, G., BROADWAY, S., CAMPER, A., DAVIES, D. and LECHEVALLIER, M., 1985. Transport and survival of bacteria attached to carbon particles in drinking-water. *Journal American Water Works Association*, **77**(4), pp. 51-51.
- MCGECHAN, M., 2002. Transport of particulate and colloid-sorbed contaminants through soil, part 2: Trapping processes and soil pore geometry. *Biosystems Engineering*, **83**(4), pp. 387-395.
- MCGEE, P., BOLTON, D.J., SHERIDAN, J.J., EARLEY, B., KELLY, G. and LEONARD, N., 2002. Survival of *Escherichia coli* O157:H7 in farm water: its role as a vector in the transmission of the organism within herds. *Journal of Applied Microbiology*, **93**(4), pp. 706-713.
- MCKERGOW, L.A. and DAVIES-COLLEY, R.J., 2010. Stormflow dynamics and loads of *Escherichia coli* in a large mixed land use catchment. *Hydrological Processes*, **24**(3), pp. 276-289.
- MCNAB, W.B., 1997. A literature review linking microbial risk assessment, predictive microbiology and dose-response modelling. *Dairy Food and Environ. Sanitation* **17**, pp. 405-416.
- MEDEMA, G.J., TEUNIS, P.F., HAVELAAR, A.H. and HAAS, C.N., 1996. Assessment of the dose-response relationship of *Campylobacter jejuni*. *International journal of food microbiology*, **30**(1-2), pp. 101-111.
- MEINHARDT, P.L., CASEMORE, D.P. and MILLER, K.B., 1996. Epidemiologic aspects of human cryptosporidiosis and the role of waterborne transmission. *Epidemiologic Reviews*, **18**(2), pp. 118-136.
- MILNES, A.S., STEWART, I., CLIFTON-HADLEY, F.A., DAVIES, R.H., NEWELL, D.G., SAYERS, A.R., CHEASTY, T., CASSAR, C., RIDLEY, A., COOK, A.J.C., EVANS, S.J., TEALE, C.J., SMITH, R.P., MCNNALLY, A., TOSZEGHY, M., FUTTER, R., KAY, A. and PAIBA, G.A., 2008. Intestinal carriage of verocytotoxigenic *Escherichia coli* O157, *Salmonella*, thermophilic *Campylobacter* and *Yersinia enterocolitica*, in cattle, sheep and pigs at slaughter in Great Britain during 2003. *Epidemiology and Infection*, **136**(6), pp. 739-751.
- MINDLIN, M.J., LANG, N., MAGUIRE, H., WALSH, B., VERLANDER, N.Q., LANE, C., TAYLOR, C., BISHOP, L.A. and CROOK, P.D., 2013. Outbreak investigation and case-control study: penta-resistant *Salmonella* Typhimurium DT104 associated with biltong in London in 2008. *Epidemiology and Infection*, **141**(9), pp. 1920-1927.
- MOLLOY, S.F., TANNER, C.J., KIRWAN, P., ASAOLU, S.O., SMITH, H.V., NICHOLS, R.A., CONNELLY, L. and HOLLAND, C.V., 2011. Sporadic *Cryptosporidium* infection in Nigerian children: risk factors with species identification. *Epidemiology and Infection*, **139**(6), pp. 946-954.
- MOORE, J.E., CORCORAN, D., DOOLEY, J.S., FANNING, S., LUCEY, B., MATSUDA, M., MCDOWELL, D.A., MEGRAUD, F., MILLAR, B.C., O'MAHONY, R., O'RIORDAN, L., O'ROURKE, M., RAO, J.R., ROONEY, P.J., SAILS, A. and WHYTE, P., 2005. *Campylobacter*. *Veterinary Research*, **36**(3), pp. 351-382.
- MORIARTY, E.M. and GILPIN, B.J., 2014. Leaching of *Escherichia coli* from sheep faeces during simulated rainfall events. *Letters in Applied Microbiology*, **58**(6), pp. 569-575.

- MUGHINI GRAS, L., SMID, J.H., WAGENAAR, J.A., DE BOER, A.G., HAVELAAR, A.H., FRIESEMA, I.H.M., FRENCH, N.P., BUSANI, L. and VAN PELT, W., 2012. Risk factors for campylobacteriosis of chicken, ruminant, and environmental origin: a combined case-control and source attribution analysis. *PLoS One*, **7**(8), pp. e42599.
- MUIRHEAD, R., DAVIES-COLLEY, R.J., DONNISON, A.M., NAGELS, J.W., 2004. Faecal bacteria yields in artificial flood events: quantifying in-stream stores. *Water Research*, **38**(5) pp. 1215-1224
- MUIRHEAD, R., COLLINS, R. and BREMER, P., 2005. Erosion and subsequent transport state of *Escherichia coli* from cowpats. *Applied and Environmental Microbiology*, **71**(6), pp. 2875-2879.
- MUIRHEAD, R., COLLINS, R. and BREMER, P., 2006. Numbers and transported state of *Escherichia coli* in runoff direct from fresh cowpats under simulated rainfall. *Letters in Applied Microbiology*, **42**(2), pp. 83-87.
- NATARO, J. and KAPER, J., 1998. Diarrheagenic *Escherichia coli*. *Clinical Microbiology Reviews*, **11**(1), pp. 142-201.
- NATIONAL RECORDS OF SCOTLAND 2019. Available at: <https://www.nrscotland.gov.uk/statistics-and-data/statistics/statistics-by-theme/population/population-estimates/mid-year-population-estimates> (accessed June 2021)
- NEILL, A.J., TETZLAFF, D., STRACHAN, N.J.C., HOUGH, R.L., AVERY, L.M., WATSON, H. and SOULSBY, C., 2018. Using spatial-stream-network models and long-term data to understand and predict dynamics of faecal contamination in a mixed land-use catchment. *Science of the Total Environment*, **612**, pp. 840-852.
- NICHOLS, R.A., CONNELLY, L., SULLIVAN, C.B. and SMITH, H.V., 2010. Identification of *Cryptosporidium* species and genotypes in Scottish raw and drinking waters during a one-year monitoring period. *Applied and Environmental Microbiology*, **76**(17), pp. 5977-5986.
- O'DONOGHUE, P., 1995. *Cryptosporidium* and Cryptosporidiosis in Man and Animals. *International Journal for Parasitology*, **25**(2), pp. 139-195.
- OGDEN, I.D., DALLAS, J.F., MACRAE, M., ROTARIU, O., REAY, K.W., THOMSON, A.P., SHEPPARD, S.K., MAIDEN, M.C., FORBES, K.J. and STRACHAN, N.J.C., 2009. *Campylobacter* excreted into the environment by animal sources: prevalence, concentration shed and host association. *Environmental Microbiology*, **6**(10), pp. 1161-1170.
- OGDEN, I.D., MACRAE, M. and STRACHAN, N.J., 2004. Is the prevalence and shedding concentrations of *E. coli* O157 in beef cattle in Scotland seasonal? *FEMS Microbiology Letters*, **233**(2), pp. 297-300.
- OGDEN, I.D., MACRAE, M. and STRACHAN, N.J., 2005. Concentration and prevalence of *Escherichia coli* O157 in sheep faeces at pasture in Scotland. *Journal of Applied Microbiology*, **98**(3), pp. 646-651.
- OLIVER, D., CLEGG, C., HAYGARTH, P. and HEATHWAITE, A., 2005. Assessing the potential for pathogen transfer from grassland soils to surface waters. *Advances in Agronomy, Vol 85*, **85**, pp. 125-180.
- OMISAKIN, F., MACRAE, M., OGDEN, I.D. and STRACHAN, N.J., 2003. Concentration and prevalence of *Escherichia coli* O157 in cattle feces at slaughter. *Applied and Environmental Microbiology*, **69**(5), pp. 2444-2447.
- PACHEPSKY, Y.A., BLAUSTEIN, R.A., WHELAN, G. and SHELTON, D.R., 2014. Comparing temperature effects on *Escherichia coli*, *Salmonella*, and *Enterococcus* survival in surface waters. *Letters in Applied Microbiology*, **59**(3), pp. 278-283.
- PACHEPSKY, Y.A., SADEGHI, A.M., BRADFORD, S.A., SHELTON, D.R., GUBER, A.K. and DAO, T., 2006. Transport and fate of manure-borne pathogens: Modeling perspective. *Agricultural Water Management*, **86**(1-2), pp. 81-92.
- PACHEPSKY, Y.A. and SHELTON, D.R., 2011. *Escherichia coli* and fecal coliforms in freshwater and estuarine sediments. *Critical Reviews in Environmental Science and Technology*, **41**(12), pp. 1067-1110.
- PACHEPSKY, Y.A., YU, O., KARNIS, J.S., SHELTON, D.R., GUBER, A.K. and VAN KESSEL, J.S., 2008. Strain-dependent variations in attachment of *E. coli* to soil particles of different sizes. *International Agrophysics*, **22**(1), pp. 61-66.
- PALMER, S., 1990. Cryptosporidiosis in England and Wales - Prevalence and clinical and epidemiologic features. *BMJ*, **300**(6727), pp. 774-777.
- PALMERA-SUAREZ, R., GARCIA, P., GARCIA, A., BARRASA, A., HERRERA, D. and INVESTIGATION TEAM, 2007. *Salmonella* Kottbus outbreak in infants in Gran Canaria (Spain), caused by bottled water, August-November 2006. *Euro surveillance : bulletin Europeen sur les maladies transmissibles = European Communicable Disease Bulletin*, **12**(7), pp. E070712.2.

- PALMGREN, H., ASPAN, A., BROMAN, T., BENGTTSSON, K., BLOMQUIST, L., BERGSTROM, S., SELLIN, M., WOLLIN, R. and OLSEN, B., 2006. *Salmonella* in Black-headed gulls (*Larus ridibundus*); prevalence, genotypes and influence on *Salmonella* epidemiology. *Epidemiology and Infection*, **134**(3), pp. 635-644.
- PANDEY, P.K., SOUPIR, M.L. and REHMANN, C.R., 2012. A model for predicting resuspension of *Escherichia coli* from streambed sediments. *Water Research*, **46**(1), pp. 115-126.
- PARK, S.F., 2002. The physiology of *Campylobacter* species and its relevance to their role as foodborne pathogens. *International Journal of Food Microbiology*, **74**(3), pp. 177-188
- PARK, S., WOROBO, R. and DURST, R., 1999. *Escherichia coli* O157 : H7 as an emerging foodborne pathogen: A literature review. *Critical reviews in Food Science and Nutrition*, **39**(6), pp. 481-502.
- PEBODY, R.G., RYAN, M.J. and WALL, P.G., 1997. Outbreaks of *Campylobacter* infection: rare events for a common pathogen. *Communicable disease report. CDR review*, **7**(3), pp. R33-7.
- PEDERSEN, S.H., WILKINSON, A.L. and MCDERMID, J.M., 2014. *Cryptosporidium* prevalence and risk factors among mothers and infants 0 to 6 months in rural and semi-rural northwest Tanzania: A prospective cohort study. *PLOS Neglected Tropical Diseases*, **8**(10).
- PENG, X., MURPHY, T. and HOLDEN, N.M., 2008. Evaluation of the effect of temperature on the die-off rate for *Cryptosporidium parvum* oocysts in water, soils, and feces. *Applied and Environmental Microbiology*, **74**(23), pp. 7101-7107.
- PETTERSON, S.R. and STENSTROEM, T.A., 2015. Quantification of pathogen inactivation efficacy by free chlorine disinfection of drinking water for QMRA. *Journal of Water and Health*, **13**(3), pp. 625-644.
- PIRES, S.M., EVERS, E.G., VAN PELT, W., AYERS, T., SCALLAN, E., ANGULO, F.J., HAVELAAR, A., HALD, T. and MED-VET-NET WORKPACKAGE 28 WORKING GROUP, 2009. Attributing the human disease burden of foodborne infections to specific sources. *Foodborne Pathogens and Disease*, **6**(4), pp. 417-424.
- POHLEL, I., HELLIWELL, R., SPEZIA, L., 2019. Citizen science evidence from the past century shows that Scottish rivers are warming. *Science of the Total Environment*, **659**, pp. 53-65.
- POLLOCK, K. and HAWKINS, G., 2015. Analysis of long-term sequelae of *Cryptosporidium* infection using data linkage. *Parallel Session D6 - Measuring Improvements in Scotland's Health - 2. Underlying Causes, Faculty of Public Health, Annual Public Health Conference 5-6 November 2015*.
- POLLOCK, K.G., TERNENT, H.E., MELLOR, D.J., CHALMERS, R.M., SMITH, H.V., RAMSAY, C.N. and INNOCENT, G.T., 2010. Spatial and temporal epidemiology of sporadic human cryptosporidiosis in Scotland. *Zoonoses and Public Health*, **57**(7-8), pp. 487-492.
- POLLOCK, K.G.J., LOCKING, M.E., GILCHRIST, L. and YOUNG, D., 2006. Clinical surveillance of haemolytic uraemic syndrome and other thrombotic microangiopathies in Scotland 2003-2005. *Health Protection Scotland Weekly Report*, **31**, pp. 2-3.
- POWELL, L.F., CHENEY, T.E.A., WILLIAMSON, S., GUY, E., SMITH, R.P., DAVIES, R.H. 2016. A prevalence study of *Salmonella* spp., *Yersinia* spp., *Toxoplasma gondii* and porcine reproductive and respiratory syndrome virus in UK pigs at slaughter. *Epidemiology and Infection*, **144**(7), pp. 1538-1549.
- PRITCHARD, J.K., STEPHENS, M. and DONNELLY, P., 2000. Inference of population structure using multilocus genotype data. *Genetics*, **155**(2), pp. 945-959.
- PUI, C.F., WONG, W.C., CHAI, L.C., LEE, H.Y., NOORLIS, A., ZAINAZOR, T.C., TANG, J.Y., GHAZALI, F.M., CHEAH, Y.K., NAKAGUCHI, Y., NISHIBUCHI, M. and RADU, S., 2011. Multiplex PCR for the concurrent detection and differentiation of *Salmonella* spp., *Salmonella* Typhi and *Salmonella* Typhimurium. *Tropical Medicine and Health*, **39**(1), pp. 9-15.
- PUJOL, J.M., EISENBERG, J.E., HAAS, C.N. and KOOPMAN, J.S., 2009. The effect of ongoing exposure dynamics in dose response relationships. *PLoS Computational Biology*, **5**(6), pp. e1000399.

- RAMALHO, R., AFONSO, A., CUNHA, J., TEIXEIRA, P. and GIBBS, P., 2001. Survival characteristics of pathogens inoculated into bottled mineral water. *Food Control*, **12**(5), pp. 311-316.
- REDDY, K., KHALEEL, R. and OVERCASH, M., 1981. Behavior and transport of microbial pathogens and indicator organisms in soils treated with organic wastes. *Journal of Environmental Quality*, **10**(3), pp. 255-266.
- REID, D., EDWARDS, A., COOPER, D., WILSON, E. and MCGAW, B., 2003. The quality of drinking water from private water supplies in Aberdeenshire, UK. *Water Research*, **37**(2), pp. 245-254.
- RICE, E.W., CLARK, R.M. and JOHNSON, C.H., 1999. Chlorine inactivation of *Escherichia coli* O157:H7. *Emerging Infectious Diseases*, **5**(3), pp. 461-463.
- RICHARDSON, H.Y., NICHOLS, G., LANE, C., LAKE, I.R. and HUNTER, P.R., 2009. Microbiological surveillance of private water supplies in England - The impact of environmental and climate factors on water quality. *Water Research*, **43**(8), pp. 2159-2168.
- RIEKE, E.L., MOORMAN, T.B., SOUPIR, M.L., YANG, F. and HOWE, A., 2018. Assessing pathogen presence in an intensively tile drained, agricultural watershed. *Journal of Environmental Quality*, **47**(5), pp. 1033-1041.
- RIERA-MONTES, M., BRUS SJOLANDER, K., ALLESTAM, G., HALLIN, E., HEDLUND, K.O. and LOFDAHL, M., 2011. Waterborne norovirus outbreak in a municipal drinking-water supply in Sweden. *Epidemiology and Infection*, **139**(12), pp. 1928-1935.
- ROBERTSON, L.J., 1996. Severe giardiasis and cryptosporidiosis in Scotland, UK. *Epidemiology and Infection*, **117**(3), pp. 551-561.
- ROBERTSON, L.J., CAMPBELL, A.T. and SMITH, H.V., 1992. Survival of *Cryptosporidium parvum* oocysts under various environmental pressures. *Applied and Environmental Microbiology*, **58**(11), pp. 3494-3500.
- ROBERTSON, L.J. and GJERDE, B.K., 2004. Effects of the Norwegian winter environment on *Giardia* cysts and *Cryptosporidium* oocysts. *Microbial Ecology*, **47**(4), pp. 359-365.
- ROCKHILL, B., NEWMAN, B. and WEINBERG, C., 1998. Use and misuse of population attributable fractions. *American Journal of Public Health*, **88**(1), pp. 15-19.
- RODGERS, P., SOULSBY, C., HUNTER, C. and PETRY, J., 2003. Spatial and temporal bacterial quality of a lowland agricultural stream in northeast Scotland. *Science of the Total Environment*, **314**, pp. 289-302.
- RODRIGUEZ, M., DE DIEGO, I., MARTINEZ, N., ROSARIO RODICIO, M. and CARMEN MENDOZA, M., 2006. Nontyphoidal *Salmonella* causing focal infections in patients admitted at a Spanish general hospital during an 11-year period (1991-2001). *International journal of medical microbiology*, **296**(4-5), pp. 211-222.
- ROTARIU, O., OGDEN, I.D., MACRITCHIE, L., FORBES, K.J., WILLIAMS, A.P., CROSS, P., HUNTER, C.J., TEUNIS, P.F.M. and STRACHAN, N.J.C., 2012. Combining risk assessment and epidemiological risk factors to elucidate the sources of human *E. coli* O157 infection. *Epidemiology and Infection*, **140**(8), pp. 1414-1429.
- ROTARIU, O., SMITH-PALMER, A., COWDEN, J., BESSELL, P.R., INNOCENT, G.T., REID, S.W., MATTHEWS, L., DALLAS, J., OGDEN, I.D., FORBES, K.J. and STRACHAN, N.J., 2010. Putative household outbreaks of campylobacteriosis typically comprise single MLST genotypes. *Epidemiology and Infection*, **138**(12), pp. 1744-1747.
- ROUX, F., SPROSTON, E.L., ROTARIU, O., MACRAE, M., SHEPPARD, S.K., BESSELL, P.R., SMITH-PALMER, A., COWDEN, J.M., MAIDEN, M.C., FORBES, K.J. and STRACHAN, N.J.C., 2013. Elucidating the aetiology of human *Campylobacter coli* infections. *PLoS One*, **8**(5), pp. e64504.
- RYU, J.H., DENG, Y. and BEUCHAT, L.R., 1999. Behavior of acid-adapted and unadapted *Escherichia coli* O157:H7 when exposed to reduced pH achieved with various organic acids. *Journal of Food Protection*, **62**(5), pp. 451-455.
- SAMPSON, R.W., SWIATNICKI, S.A., OSINGA, V.L., SUPITA, J.L., MCDERMOTT, C.M. and KLEINHEINZ, G.T., 2006. Effects of temperature and sand on *E. coli* survival in a northern lake water microcosm. *Journal of Water and Health*, **4**(3), pp. 389-393.

- SANTAMARIA, J. and TORANZOS, G.A., 2003. Enteric pathogens and soil: a short review. *International microbiology : the Official Journal of the Spanish Society for Microbiology*, **6**(1), pp. 5-9.
- SANTAMARIA, J., BRUSSEAU, M.L., ARAUJO, J., OROSZ-COGLAN, P., BLANFORD, W.J. and GERBA, C.P., 2012. Transport and retention of *Cryptosporidium parvum* oocysts in sandy soils. *Journal of Environmental Quality*, **41**(4), pp. 1246-1252.
- SARKAR, R., TATE, J.E., AJJAMPUR, S.S.R., KATTULA, D., JOHN, J., WARD, H.D. and KANG, G., 2014. Burden of Diarrhea, Hospitalization and Mortality Due to Cryptosporidial Infections in Indian Children. *Plos Neglected Tropical Diseases*, **8**(7), pp. e3042.
- SAXENA, T., KAUSHIK, P. and MOHAN, M.K., 2015. Prevalence of *E. coli* O157:H7 in water sources: an overview on associated diseases, outbreaks and detection methods. *Diagnostic Microbiology and Infectious Disease*, **82**(3), pp. 249-264.
- SHEPPARD, S.K., DALLAS, J.F., STRACHAN, N.J.C., MACRAE, M., MCCARTHY, N.D., WILSON, D.J., GORMLEY, F.J., FALUSH, D., OGDEN, I.D., MAIDEN, M.C. and FORBES, K.J., 2009. *Campylobacter* genotyping to determine the source of human infection. *Clinical Infectious Diseases*, **48**(15 April), pp. 1072-1078.
- SILVA, J., LEITE, D., FERNANDES, M., MENA, C., GIBBS, P.A. and TEIXEIRA, P., 2011. *Campylobacter* spp. as a Foodborne Pathogen: A Review. *Frontiers in Microbiology*, **2**, pp. 200.
- SINGLETON, P., 2004. *Bacteria in biology, biotechnology and medicine (6th Edition)*. John Wiley and Sons Ltd., UK.
- SIVAPALASINGAM, S., HOEKSTRA, R.M., MCQUISTON, J.R., FIELDS, P.I. and TAUXE, R.V., 2004. *Salmonella* bacteriuria: an increasing entity in elderly women in the United States. *Epidemiology and Infection*, **132**(5), pp. 897-902.
- SLUTSKER, L., RIES, A.A., MALONEY, K., WELLS, J.G., GREENE, K.D. and GRIFFIN, P.M., 1998. A nationwide case-control study of *Escherichia coli* O157:H7 infection in the United States. *The Journal of Infectious Diseases*, **177**(4), pp. 962-966.
- SMITH, R.P., CLIFTON-HADLEY, F.A., CHENEY, T. and GILES, M., 2014. Prevalence and molecular typing of *Cryptosporidium* in dairy cattle in England and Wales and examination of potential on-farm transmission routes. *Veterinary Parasitology*, **204**(3-4), pp. 111-119.
- SMITH-PALMER, A. and COWDEN, J., 2013. Gastro-intestinal and foodborne infections: General outbreaks of infectious intestinal disease reported to HPS during 2012. *HPS Weekly Report*, **47**, pp. 166-174.
- SMITH-PALMER, A. and COWDEN, J., 2010. *Private water supplies as a risk factor for Campylobacter infection in Aberdeen City and Aberdeenshire*.
- SMITH-PALMER, A., POLLOCK, K., BROWNLIE, S. and COWDEN, J., 2012. Gastro-intestinal and foodborne infections: Norovirus, rotavirus, cryptosporidium and giardia – laboratory reports, 2011. *Health Protection Scotland Weekly Report*, **46**, pp. 73-76.
- SOAVE, R., DANNER, R.L., HONIG, C.L., MA, P., HART, C.C., NASH, T. and ROBERTS, R.B., 1984. Cryptosporidiosis in homosexual men. *Annals of Internal Medicine*, **100**(4), pp. 504-511.
- SOLECKI, O., 2008. Explaining the urban and rural differences of *E. coli* O157 human Infection in Grampian, University of Aberdeen.
- SOLECKI, O., MACRAE, M., STRACHAN, N., LINDSTEDT, B.A. and OGDEN, I., 2009. *E. coli* O157 from sheep in northeast Scotland: prevalence, concentration shed, and molecular characterization by multilocus variable tandem repeat analysis. *Foodborne Pathogens and Disease*, **6**(7), pp. 849-854.
- STANLEY, K. and JONES, K., 2003a. Cattle and sheep farms as reservoirs of *Campylobacter*. *Journal of applied microbiology*, **94** Suppl, pp. 104S-113S.
- STANLEY, K. and JONES, K., 2003b. Cattle and sheep farms as reservoirs of *Campylobacter*. *Journal of Applied Microbiology*, **94** Suppl, pp. 104S-113S.

- STANLEY, K., CUNNINGHAM, R. AND JONES, K., 1998. Isolation of *Campylobacter jejuni* from groundwater. *Journal of Applied Microbiology*, **85**(1), pp. 187-191.
- STERN, N.J. and KOTULA, A.W., 1982. Survival of *Campylobacter jejuni* inoculated into ground beef. *Applied and Environmental Microbiology*, **44**(5), pp. 1150-1153.
- STOCKER, M.D., PENROSE, M. and PACHEPSKY, Y.A., 2018. Spatial patterns of *Escherichia coli* concentrations in sediment before and after high-flow events in a first-order creek. *Journal of Environmental Quality*, **47**(5), pp. 958-966.
- STRACHAN, N.J., DOYLE, M.P., KASUGA, F., ROTARIU, O. and OGDEN, I.D., 2005. Dose response modelling of *Escherichia coli* O157 incorporating data from foodborne and environmental outbreaks. *International Journal of Food Microbiology*, **103**(1), pp. 35-47.
- STRACHAN, N.J., GORMLEY, F.J., ROTARIU, O., OGDEN, I.D., MILLER, G., DUNN, G.M., SHEPPARD, S.K., DALLAS, J.F., REID, T.M., HOWIE, H., MAIDEN, M.C. and FORBES, K.J., 2009. Attribution of *Campylobacter* infections in northeast Scotland to specific sources by use of multilocus sequence typing. *Journal of Infectious Diseases*, **199**(8), pp. 1205-1208.
- STRACHAN, N.J.C., ROTARIU, O., SMITH-PALMER, A., COWDEN, J., SHEPPARD, S.K., O'BRIEN, S.J., MAIDEN, M.C.J., MACRAE, M., BESSELL, P.R., MATTHEWS, L., REID, S.W.J., INNOCENT, G.T., OGDEN, I.D. and FORBES, K.J., 2013. Identifying the seasonal origins of human campylobacteriosis. *Epidemiology and Infection*, **141**(6), pp. 1267-1275.
- STRACHAN, N.J.C., ROTARIU, O., MACRAE, M., SHEPPARD, S.K., SMITH-PALMER, A., COWDEN, J., MAIDEN, M.C.J. and FORBES, K.J., 2013. Operationalising factors that explain the emergence of infectious diseases: A case study of the human campylobacteriosis epidemic. *Plos One*, **8**(11), pp. e79331.
- STRACHAN, N.J.C., ROTARIU, O., LOPES, B., MACRAE, M., FAIRLEY, S., LAING, C., GANNON, V., ALLISON, L.J., HANSON, M.F., DALLMAN, T., ASHTON, P., FRANZ, E., VAN HOEK, A.H.A.M., FRENCH, N.P., GEORGE, T., BIGGS, P.J. and FORBES, K.J., 2015. Whole Genome Sequencing demonstrates that Geographic Variation of *Escherichia coli* O157 Genotypes Dominates Host Association. *Scientific Reports*, **5**, pp. 14145.
- STURDEE, A., BODLEY-TICKELL, A., ARCHER, A. and CHALMERS, R., 2003. Long-term study of *Cryptosporidium* prevalence on a lowland farm in the United Kingdom. *Veterinary Parasitology*, **116**(2), pp. 97-113.
- TAM, C.C., RODRIGUES, L.C., PETERSEN, I., ISLAM, A., HAYWARD, A. and O'BRIEN, S.J., 2006. Incidence of Guillain-Barre syndrome among patients with *Campylobacter* infection: a general practice research database study. *The Journal of infectious diseases*, **194**(1), pp. 95-97.
- TETZLAFF, D., SOULSBY, C. and BIRKEL, C., 2010. Hydrological connectivity and microbiological fluxes in montane catchments: the role of seasonality and climatic variability. *Hydrological Processes*, **24**(9), pp. 1231-1235.
- TEUNIS, P.F.M., VAN DER HEIJDEN, O.G., AN DER GIESSEN, J.W.B. and HAVELAAR, A.H., 1996. *The dose response relation in human volunteers for gastro-intestinal pathogens*. 284550002. The Netherlands: RIVM.
- TEUNIS, P., TAKUMI, K. and SHINAGAWA, K., 2004. Dose response for infection by *Escherichia coli* O157 : H7 from outbreak data. *Risk Analysis*, **24**(2), pp. 401-407.
- TEUNIS, P., VAN DEN BRANDHOF, W., NAUTA, M., WAGENAAR, J., VAN DEN KERKHOF, H. and VAN PELT, W., 2005. A reconsideration of the *Campylobacter* dose-response relation. *Epidemiology and infection*, **133**(4), pp. 583-592.
- TEUNIS, P.F., CHAPPELL, C.L. and OKHUYSEN, P.C., 2002a. *Cryptosporidium* dose response studies: variation between isolates. *Risk analysis : an official publication of the Society for Risk Analysis*, **22**(1), pp. 175-183.
- TEUNIS, P.F., CHAPPELL, C.L. and OKHUYSEN, P.C., 2002b. *Cryptosporidium* dose-response studies: variation between hosts. *Risk analysis : an official publication of the Society for Risk Analysis*, **22**(3), pp. 475-485.
- TEUNIS, P.F. and HAVELAAR, A.H., 2000. The Beta Poisson dose-response model is not a single-hit model. *Risk analysis : an official publication of the Society for Risk Analysis*, **20**(4), pp. 513-520.

- TEUNIS, P.F., KASUGA, F., FAZIL, A., OGDEN, I.D., ROTARIU, O. and STRACHAN, N.J., 2010. Dose-response modeling of *Salmonella* using outbreak data. *International journal of food microbiology*, **144**(2), pp. 243-249.
- TEUNIS, P.F., NAGELKERKE, N.J. and HAAS, C.N., 1999a. Dose response models for infectious gastroenteritis. *Risk analysis : an official publication of the Society for Risk Analysis*, **19**(6), pp. 1251-1260.
- TEUNIS, P.F., NAGELKERKE, N.J. and HAAS, C.N., 1999b. Dose response models for infectious gastroenteritis. *Risk Analysis : Official Publication of the Society for Risk Analysis*, **19**(6), pp. 1251-1260.
- TEUNIS, P.F., OGDEN, I.D. and STRACHAN, N.J., 2008. Hierarchical dose response of *E. coli* O157:H7 from human outbreaks incorporating heterogeneity in exposure. *Epidemiology and Infection*, **136**(6), pp. 761-770.
- THULLNER, M., MAUCLAIRE, L., SCHROTH, M., KINZELBACH, W. and ZEYER, J., 2002. Interaction between water flow and spatial distribution of microbial growth in a two-dimensional flow field in saturated porous media. *Journal of Contaminant Hydrology*, **58**(3-4), pp. 169-189.
- TOBIN, R.S., SMITH, D.K. and LINDSAY, J.A., 1981. Effects of activated carbon and bacteriostatic filters on microbiological quality of drinking water. *Applied and Environmental Microbiology*, **41**(3), pp. 646-651.
- TYRREL, S. and QUINTON, J., 2003. Overland flow transport of pathogens from agricultural land receiving faecal wastes. *Journal of Applied Microbiology*, **94**, pp. 87S-93S.
- USEPA, 2010. *Ultraviolet disinfection guidance manual for the final long term 2 Enhanced Surface Water Treatment Rule EPA 815-R-06-007*.
- VINTEN, A., DOUGLAS, J., LEWIS, D., AITKEN, M. and FENLON, D., 2004. Relative risk of surface water pollution by *E. coli* derived from faeces of grazing animals compared to slurry application. *Soil Use and Management*, **20**(1), pp. 13-22.
- VINTEN, A.J.A., POTTS, J., AVERY, L. and STRACHAN, N.J.C., 2009. Microbial pollution of water by livestock: approaches to risk assessment and mitigation. *Animal*, **3**(5), pp. 744-752.
- WALKER, F. and STEDINGER, J., 1999. Fate and transport model of *Cryptosporidium*. *Journal of Environmental Engineering-Asce*, **125**(4), pp. 325-333.
- WALLENDER, E.K., AILES, E.C., YODER, J.S., ROBERTS, V.A. and BRUNKARD, J.M., 2014. Contributing factors to disease outbreaks associated with untreated groundwater. *Ground Water*, **52**(6), pp. 886-897.
- WASSENAAR, T.M. and BLASER, M.J., 1999. Pathophysiology of *Campylobacter jejuni* infections of humans. *Microbes and Infection*, **1**(12), pp. 1023-1033.
- WELDEYOHANNES, A.O., KACHANOSKI, G. and DYCK, M., 2018. Wastewater flow and pathogen transport from at-grade line sources to shallow groundwater. *Journal of Environmental Quality*, **47**(5), pp. 1051-1057.
- WELLS, B., SHAW, H., HOTCHKISS, E., GILRAY, J., AYTON, R., GREEN, J., KATZER, F., WELLS, A. and INNES, E., 2015. Prevalence, species identification and genotyping *Cryptosporidium* from livestock and deer in a catchment in the Cairngorms with a history of a contaminated public water supply. *Parasites & Vectors*, **8**, pp. 66.
- WHO, 2004. *Guidelines for Drinking-water Quality: Recommendations v. 1 (WHO Water Series)*. World Health Organisation.
- WHO, 2011. *Guidelines for Drinking Water Quality*. World Health Organisation.
- WILLSHAW, G., CHEASTY, T., SMITH, H., O'BRIEN, S. and ADAK, G., 2001. Verocytotoxin-producing *Escherichia coli* (VTEC) O157 and other VTEC from human infections in England and Wales: 1995-1998. *Journal of Medical Microbiology*, **50**(2), pp. 135-142.
- WINWARD, G.P., AVERY, L.M., STEPHENSON, T. and JEFFERSON, B., 2008. Ultraviolet (UV) disinfection of grey water: particle size effects. *Environmental Technology*, **29**(2), pp. 235-244.

- WOODWARD, D.L., KHAKHRIA, R. and JOHNSON, W.M., 1997. Human salmonellosis associated with exotic pets. *Journal of Clinical Microbiology*, **35**(11), pp. 2786-2790.
- WORKMAN, S.N., MATHISON, G.E. and LAVOIE, M.C., 2005. Pet dogs and chicken meat as reservoirs of *Campylobacter* spp. in Barbados. *Journal of Clinical Microbiology*, **43**(6), pp. 2642-2650.
- XIAO, L., 2010. Molecular epidemiology of cryptosporidiosis: an update. *Experimental Parasitology*, **124**(1), pp. 80-89.
- ZHANG, G., ZHOU, R., HUANG, P., QIN, Y., HU, P. and CAO, W., 2012. Development of in vitro cultivation model of *Cryptosporidium parvum* in HCT-8 cells and morphological observation of different developmental stages. *Zhongguo Shouyi Kexue*, **42**(2), pp. 111-118.
- ZIA, S., WAREING, D., SUTTON, C., BOLTON, E., MITCHELL, D. and GOODACRE, J.A., 2003. Health problems following *Campylobacter jejuni* enteritis in a Lancashire population. *Rheumatology (Oxford, England)*, **42**(9), pp. 1083-1088.
- ZIMMER, J.L. and SLAWSON, R.M., 2002. Potential repair of *Escherichia coli* DNA following exposure to UV radiation from both medium- and low-pressure UV sources used in drinking water treatment. *Applied and Environmental Microbiology*, **68**(7), pp. 3293-3299.
- ZIMMER, J., SLAWSON, R. and HUCK, P., 2003. Inactivation and potential repair of *Cryptosporidium parvum* following low- and medium-pressure ultraviolet irradiation. *Water Research*, **37**(14), pp. 3517-3523.

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