# Human health risks of faecal pollution from different sources: A review of the literature

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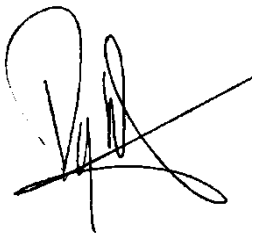

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# ABBREVIATIONS

| | |
|---|---|
| CFU | Colony forming unit |
| EHEC | Enterohaemorraghic *Escherichia coli* |
| EPEC | Enteropathogenic *E. coli* |
| ESBL | Extended-spectrum beta-lactamase |
| ESC | Cephalosporinase *E. coli* |
| FIB | Faecal indicator bacteria |
| GC | Gene copies |
| MPN | Most probable number |
| PCR | Polymerase Chain Reaction |
| PFU | Plaque forming unit |
| RT-PCR | Reverse-transcriptase Polymerase Chain Reaction |
| STEC | Shiga-toxin-producing *E. coli* |
| Stx1 | Shiga toxin 1 gene |
| Stx2 | Shiga toxin 2 gene |

# EXECUTIVE SUMMARY

Faecal pollution of surface water can come from a number of sources including raw human sewage, treated sewage, farm animals such as cows and sheep, domestic animals, wildfowl and many other sources. The aim of this project was to explore the evidence available in the literature as to how contact with surface water contaminated with these sources of pollution may affect human health risk (e.g. during swimming or secondary contact activities, such as boating). In particular, the goal was to assess whether non-human sources (e.g. wildfowl) could affect human health, given that previous research has shown high levels of non-human pathogen sources in Christchurch waterways (Moriarty & Gilpin 2015). The first approach taken in this project was to explore the presence and levels of indicator bacteria (such as *Escherichia coli* and enterococci) and pathogens in a range of animal faeces relevant to the New Zealand environment. We then evaluated published Quantitative Microbial Risk Assessments (QMRA) of the risk to human health of water contaminated with faeces from different sources.

All animal and wildfowl faeces are potential human health risks. Many studies of pathogens in animal faeces have been presence/absence based, and have used methods that detect all species or subtypes of a particular genus or species without differentiating between the pathogenic and non-pathogenic types. Future studies will increasingly make greater use of molecular techniques to identify pathogenicity which will build a clearer picture of the risk to human health of microorganisms from different species. In addition differences in methodology, regional differences, and the small number of samples analysed in many studies, limits the robustness of comparisons between sources. With these caveats in mind, we make the following observations:

- *Campylobacter* have been identifed in human, sheep, cattle, dairy cow, dog, cat, black swan, duck, canada geese, and gull faeces.
- *Cryptosporidium* have been identifed in human, sheep, cattle, dairy cow, dog, cat, goats, duck and geese faeces.
- *Giardia* have been identifed in human, sheep, cattle, dairy cow, dog, cat, goat, duck and geese faeces.
- *Salmonella* have been identifed in human, sheep, cattle, dairy cow, dog, cat, duck, pigeon and geese faeces.
- Pathogenic *Escherichia coli* have been identifed in human, sheep, cattle, dairy cow, dog, cat, pigeon and geese faeces.

- Viruses of importance to human health are only found in human faeces.

There is a requirement for on-going monitoring of faecal sources for the emergence of new pathogens or changes in virulence or prevalence of existing pathogens, which will impact human health risk.

Published Quantitative Microbial Risk Assessment (QMRA) studies suggest that human faeces has the greatest health risk, including when it is only a minor component of faecal pollution in water. Keeping human faeces out of recreational and drinking water must remain a priority. Treated human sewage needs to be evaluated on the basis of individual treatment processes to assess the pathogen inactivation rates and likely impacts on the ratio of indicator to pathogen.

All animal and wildfowl faeces are potential human health risks, particularly to children and immunocompromised individuals. Amongst the animals characterised by risk modelling in international studies, cattle/dairy cow sources appear to have the highest risk, which is driven by the presence of *Campylobacter*, pathogenic *E. coli* and *Cryptosporidium*. The health risk associated with poultry appears to be mainly driven by *Campylobacter,* making poultry of lower risk than human and cattle/dairy cow source, but higher risk than wildfowl. Notable in these QMRA studies, is the absence of information on health risks from sheep and lambs which represent a significant portion of the faecal contamination observed in rural NZ rivers and streams. The QMRA studies have also only be undertaken using gull faeces as a wildfowl source.

A key conclusion from these QMRA studies is that in water containing the same level of faecal indicator from different sources, there is potentially a lower risk of human illness when the water is impacted by chicken, gull and pig faecal material, than human or cattle faeces. Further extrapolation of this work suggests that if the indicator organisms in water are **entirely** from chicken, pig or gull sources, acceptable levels of indicator organisms could be three to 50 times higher than if from a human source.

# 1. INTRODUCTION

Faecal pollution of water can come from a number of sources including raw human sewage, treated sewage, farm animals such as cows and sheep, wildfowl and many other sources. The aim of this report was to review the published literature on levels of indicators and pathogens in a range of sources potentially polluting water in New Zealand.

The scope of this review was restricted to the following sources:

- human sewage (Raw and Treated);
- sheep and cows;
- dogs and cats;
- wildfowl.

During the course of reviewing the literature, information regarding goat faeces was also found and is included. In each of these sources we wanted to identify the presence and levels in faeces of faecal indicator bacteria (*E. coli* and enterococci), and pathogens (*Campylobacter*, *Salmonella*, STEC, Viruses, *Cryptosporidum* and *Giardia*). There was an emphasis on those pathogens known for their zoonotic potential. The European Academies Science Advisory council (EASAC) defines zoonoses as an infection that is naturally transmissible, directly or indirectly, between vertebrate animals and humans. Some zoonoses cause disease in the animal and human, while others are commensal in the animal host.

Between 5 May and 25 June, 2015 we carried out a literature search using Science direct and University of Canterbury database using the following search terms:

- *Cryptosporidium*
- *Giardia*
- Beef cattle
- Dairy
- Sheep
- Lambs
- Cats
- Dogs
- Wildfowl
- Avian
- Birds
- Pets
- Companion animals
- Faecal indicator bacteria
- *Escherichia coli*
- STEC/VTEC
- Enterococci
- Viruses, enterovirus
- Zoonotic/zoonoses
- Toxoplasmosis
- Human wastewater
- Treated wastewater
- New Zealand

We reviewed online abstracts for over 300 papers, and retrieved and reviewed full papers from approximately 100 papers.

# 2. ANIMAL FAECES

This section describes the results of literature searches for the presence and levels of indicator bacteria and pathogens in a range of sources.

## 2.1 AVERAGE DAILY FAECAL OUTPUTS

To enable calculation of the daily output of any microorganism from a particular source requires an estimate of the daily output of faecal material from that source. These varied from between 24.8 kg per day for dairy cows down to 0.05 kg per day for seagulls (TABLE 1). Estimates of daily faecal load were not found for cats or pigs.

**TABLE 1: Published daily faecal outputs from a range of animals**

| Microorganism | Prevalence | References |
|---|---|---|
| Dairy Cow | 24.8 kg day$^{-1}$ | Muirhead et al. (2011) |
| Dairy calf | 1.65 kg day$^{-1}$ | Atwill et al. (2012) |
| Beef cattle | 14.4 kg day$^{-1}$ | |
| Sheep | 1.5 kg day$^{-1}$ | Moriarty et al. (2011a) |
| Large dog | 52 g dw day$^{-1}$ | Wright et al. (2009) |
| Small dog | 7.6 g dw day$^{-1}$ | |
| Gull | 0.05 kg day$^{-1}$ | |
| Canada geese | 0.250 kg day$^{-1}$ | |

## 2.2 BOVINE SOURCES – DAIRY COWS AND BEEF CATTLE

Quantitative data in dairy cow faeces is primarily based on New Zealand studies, with *Campylobacter* frequently detected at high levels, while *Salmonella* were not detected in either of the NZ studies undertaken (TABLE 2). A number of other studies reported the presence/absence of a range of pathogens (TABLE 3).

**TABLE 2: Quantitative data on levels of indicators and pathogens in dairy cow and beef cattle faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size (No. of animals)? | Daily Output/cow | Country | References |
|---|---|---|---|---|---|---|
| *E. coli* | 8.20E+04 | 99.5 | 155 | 2.01E+09 | NZ | Moriarty et al. (2008) |
| Enterococci | 4.50E+02 | 93.3 | 155 | 1.04E+07 | | |
| *Campylobacter* | 4.30E+02 | 63.9 | 155 | 6.81E+06 | | |
| *Salmonella* | 0.00E+00 | 0 | | - | | |
| *Campylobacter* (thermophilic) includes *C. jejuni and C. coli* | 1.3E+02 | 9 | 120 | 2.90E+05 | Denmark | Nielsen (2002) |
| | 6.1E+02 | 89.4 | 360 | 7.85E+06 | UK | Stanley et al. (1998) |
| STEC *E. coli* | 1.0E+05 to 1.0E+08 | 15.9 | 605 | 2.3E+08 to 2.3E+11 | Japan | Fukushima and Seki (2004) |
| *Cryptosporidium parvum* | 3.38E+00 | 0.71 | | 5.95E+04 | USA | Atwill et al. (2003) |

Mean CFU/MPN /(oo)cysts $g^{-1}$ – Mean CFU (colony forming units), MPN (Most probable number) or oocysts (in case of protozoa).

**TABLE 3: Prevalence data for indicators and pathogens in dairy cow faeces**

| Microorganism | Prevalence % | Study size (No of animals)? | Country | References |
|---|---|---|---|---|
| *Campylobacter* spp. | 36 | 161 | NZ | Grinberg et al. (2005) |
| *C. jejuni* | 6.8 | 161 | | |
| *Campylobacter* spp. | 40 | pooled n = 496 | UK | Brown et al. (2004) |
| *C. jejuni*<br>*C. coli* | 31<br>6 | 311 | USA | Bae et al. (2005) |
| *Salmonella enterica* | 9.6 | 960 | USA | Callaway et al. (2005) |
| *Salmonella enterica* | 56 | 16 herds | | |
| STEC *E. coli* | 1.3 | 155 | NZ | Moriarty et al. (2008) |
| STEC *E. coli* | *stx*1 4<br>*stx*2 6<br>*eae* 7 | 72 | NZ | Cookson et al. (2006a) |
| STEC *E. coli* | 100 in all farms | Per Farm Organic (n = 60) and Conventional farms (n =60) | Switzerland | Kuhnert et al. (2005) |
| *E. coli* O157 | 25 organic<br>17 conventional | | | |
| STEC-<br>*E. coli* O157- | 58<br>4.6 | 500 cows | | |
| non *E .coli* O157 STECs | 20.7 | 82 herds | Spain | Oporto et al. 2008 |
| *E. coli* O157 :H7 | 7 | 82 herds | | |
| *STEC E. coli* | *stx*1 30-47<br>*stx*2 30-53<br>*eae* 64-76 | Approx. 9000 faecal samples | USA | Lambertini et al., (2015 |
| *Cryptosporidium* spp. | 5.2 | 155 | NZ | Moriarty et al. 2008 |
| *Cryptosporidium* spp. | 7.3 | 288 | Ireland | Moriarty et al. 2005 |
| *C. parvum* | 0.6 | 354 | NZ | Learmonth et al. 2003 |

**TABLE 4: Presence/Absence data indicators and pathogens in beef cattle faeces**

| Microorganism | Prevalence % | Study size | Country | References |
|---|---|---|---|---|
| *E. coli* O157 :H7 | 6.70 | 30 | Spain | Oporto et al. 2008 |
| *E. coli* O157 :H8 | 1.60 | 124 herds | Spain | Oporto et al. 2008 |
| Shiga toxin *E. coli* (STEC) non *E .coli* O157 | 46 | 124 herds | Spain | Oporto et al. 2008 |
| *Salmonella* | 6.20 | 130 | Italy | European Food Safety Agency, 2010 |
| *Salmonella* | 5.40 | 707 | Italy | |
| *Salmonella* | 0.3- 1.3 | 199-386 animal per year over 12 years | Slovenia | |
| *Cryptosporidium parvum* | 8.4 | 379 | Spain | Castro-Hermida et al. 2007 |
| *Giardia duodenalis* (Assemblage A) | 7.30 | 110 | Germany | Gillhuber et al. 2013 |
| *Giardia enteris* (zoonotic) | 0 | | | |
| *Giardia duodenalis* | 26.6 | 379 | Spain | Castro-Hermida et al. 2007 |

Younger animals have been found to have higher levels of *Cryptosporidium* and higher prevalence of both and *Cryptosporidium* and *Giardia* (TABLE 5).

**TABLE 5: Indicators and pathogens in calf faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output/ calf | Country | References |
|---|---|---|---|---|---|---|
| *Campylobacter* (thermophilic) includes *C. jejuni and C. coli* | ≤4 months old 2.5E+04 | 42 | 107 | 1.73E+06 | Denmark | Nielsen (2002) |
| | >4 months old 7.90E+02 | 20 | 105 | 1.36E+06 | | |
| *C. jejuni*<br>*C. coli* | | 24<br>20 | 105 | | USA | Bae et al. (2005) |
| *Salmonella* | 0.00E+00 | 0 | 156 | 0 | NZ | Grinberg et al. 2005 |
| *E. coli* O157:H7 | | 23-26 | 52 | | Canada | (Gannon et al., 2002) |
| STEC *E. coli* and enteropathogenic E. coli (EPEC)(*eae* gene) | | *stx*1 2<br>*stx*2 19<br>*eae* 44 | 91 | | NZ | Cookson et al. (2006a) |
| STEC *E. coli* and E. coli (EPEC)(*eae* gene) | | STEC 2.6<br>Atypical EPEC 12.3 | 299 | | NZ | Irshad et al. (2014) |
| *E. coli* O157 | | 17.7 and 23.8 farms | 309 calves, Farms n=197 | | NZ | Irshad et al., (2012) |
| *C. parvum* | 3.00E+06 | 10-80 | | 1.50E+10 | | Atwill et al. (2012) |
| *C. parvum* | | 10.9 | 304 | | NZ | Learmonth et al. (2003) |
| *C. parvum* | | 21.2 | 156 | | NZ | Grinberg et al. (2005) |
| *Giardia* | | 4.5 | | 1.00E+03 | NZ | Moriarty et al. (2008) |

## 2.4 SHEEP AND LAMB FAECES

A number of studies have been completed on indicators and pathogens in sheep and lamb faeces (TABLES 6-9).

**TABLE 6: Quantitative data on levels of indicators and pathogens in sheep faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output/ sheep | Country | References |
|---|---|---|---|---|---|---|
| *E. coli* | 1.67E+07 | 100 | 220 | 2.51E+10 | NZ | Moriarty et al. (2011a) |
| Enterococci | 6.80E+05 | 100 | 220 | 1.02E+09 | | |
| Enterococci | 1.20E+04 | 100 | 7 | 1.80E+07 | NZ | Anderson et al. (1997) |
| *Campylobacter* | 2.08E+03 | 30.4 | 220 | 9.48E+05 | NZ | Moriarty et al. (2011a) |
| *E. coli* O157 | Range <100-1.0E+06 | 6.5 | 15 farms ~50 sheep/farm | 9.8E+03 to 9.8E+07 | Scotland | Ogden et al. (2005) |
| *C. parvum* | 5.30E+01 | 5.3 | 446 | 4.21E+03 | Spain | Castro-Hermida et al. (2007) |
| *Giardia duodenalis* | 3.24E+02 | 19.2 | 446 | 9.33E+04 | | |

**TABLE 7: Prevalence data indicators and pathogens in sheep faeces**

| Microorganism | Prevalence % | Study size | Country | References |
|---|---|---|---|---|
| *C. jejuni* | 25 | 24 | UK | Brown et al. (2004) |
| *C. coli* | 21 | 24 | | |
| *Cryptosporidium* spp. | 3.6 | 220 | NZ | Moriarty et al. (2011a) |
| *Cryptosporidium* spp. | 9.4 - 25.0 | 32 | USA | Santin et al. (2007) |
| *C. parvum* | 3.1 | 32 | | |
| *Cryptosporidium* spp. | 26 | 500 | Australia | Ryan et al (2005) |
| *C. parvum* | 0 | | | |
| *C. hominis* | 0.02 | | | |
| *Giardia* Assemblage A* | 11 | 500 | Australia | Ryan et al (2005) |
| *Giardia* | 18.8-37.5 | 32 | USA | Santin et al. (2007) |
| *E. coli* O157:H7 | 7.3 | 278 individuals | Spain | Oporto et al. (2008) |
| *E. coli* O157:H7 | 8.7 | 122 herds | | |
| Shiga toxin *E. coli* (STEC) but non *E. coli* O157:H7 | 50.8 | 122 herds | | |
| STEC *E. coli* | *stx*1 56<br>*stx*2 18<br>*eae* 22 | 50 | NZ | Cookson et al. (2006a) |
| | | | | |
| Shiga toxin *E. coli* (STEC) | 1 | 220 | NZ | Moriarty et al. (2011a) |
| *Salmonella* | 0 | 220 | | |

*Human infective form of *G. duodenalis*

**TABLE 8: Quantitative data on levels of indicators and pathogens in lamb faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output/ lamb | Country | References |
|---|---|---|---|---|---|---|
| *E. coli* | 6.04E+08 | 100 | 105 | 4.53E+11 | NZ | Moriarty et al. (2011a) |
| Enterococci | 1.44E+07 | 100 | 105 | 1.08E+10 | | |
| *Campylobacter* | 3.33E+05 | 80.9 | | 2.02E+08 | | |
| *Cryptosporidium* spp. | 6.83E+03 | 0.9 | 137 | 4.61E+06 | Belgium | Geurden et al., (2008) |
| *Cryptosporidium* - cervine genotype | 8.90E+03 | 28.6 | 105 | 1.91E+06 | NZ | Moriarty et al. (2011a) |
| *Giardia* spp. | 2.80E+01 | 37.1 | 105 | 7.79E+03 | | |
| *Giardia* | 3.60E+04 | 4.8 | 3142 | 1.31E+06 | Australia | Yang et al. (2014) |
| *Giardia* spp. | 4.58E+03 | 25.5 | 137 | 5.84E+05 | Belgium | Geurden et al., (2008) |

**TABLE 9: Presence/Absence data indicators and pathogens in lamb faeces**

| Microorganism | Prevalence % | Study size | Country | References |
|---|---|---|---|---|
| *C. parvum* | 13 | 477 | Australia | Yang et al. (2009) |
| *Cryptosporidium* spp. | 33-77 | 31 | USA | Santin et al. (2007) |
| *C. parvum* | 3.2 | 31 | | |
| *Giardia* spp. | 11.6 | 477 | Australia | Yang et al. (2009) |
| *Giardia* Assemblage A | 1.1 | 477 | | |
| *Giardia* | 6.5-12.9 | 31 | USA | Santin et al. (2007) |
| STEC *E. coli* | *stx*1 48<br>*stx*2 9<br>*eae* 13 | 46 | NZ | Cookson et al. (2006a) |
| non *E .coli* O157 STEC | 3.8 | 105 | NZ | Moriarty et al. (2011a) |
| *Salmonella* | 1.9 | 105 | | |

## 2.5 GOAT FAECES

While we found data from Spain and Belgium on the levels of *Giardia* and *Cryptosporidium* in goat faeces (TABLE 10), we saw no data on levels of faecal indicator bacteria and bacterial pathogens from this source.

**TABLE 10: Quantitative data on levels of pathogens in goat faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output/ goat | Country | References |
|---|---|---|---|---|---|---|
| *C. parvum* | 1.84E+02 | 7.7 | 116 | 2.13E+04 | Spain | Castro-Hermida et al. (2007) |
| *Cryptosporidium* spp. | 2.30E+05 | 9.5 | 148 | 3.28E+07 | Belgium | Geurden et al., (2008) |
| *Giardia duodenalis* | 1.13E+02 | 19.8 | 116 | 3.36E+04 | Spain | Castro-Hermida et al. (2007) |
| *Giardia* spp. | 1.80E+04 | 35.8 | 148 | 9.67E+06 | Belgium | Geurden et al., (2008) |

## 2.6 DOG FAECES

We were unable to find any data on *E. coli* levels in dog faeces. While there were some information on enterococci in dog faeces, the results are based on a total of 10 animals (TABLE 11), so again are limited. There are much more data on the presence or absence of pathogens in dog faeces, with a range of pathogenic *E. coli*, *Campylobacter*, *Cryptosporidium*, *Giardia*, *Clostridium* and *Salmonella* all reported in dog faeces (TABLE 12).

**TABLE 11: Quantitative data on levels of Enterococci in dog faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output/ dog | Country | References |
|---|---|---|---|---|---|---|
| Enterococci | 6.40E+07 | 100 | 6 medium-large dogs | 3.30E+09 | USA | Wright et al. (2009) |
| Enterococci | 5.90E+06 | 100 | 3 small dogs | 4.50E+06 | | |
| Enterococci | 2.30E+04 | 100 | 1 | 1.20E+06 | NZ | Anderson et al. (1997) |

**TABLE 12: Presence/absence data on pathogens in dog faeces**

| Microorganism | Prevalence % | Study size | Country | References |
|---|---|---|---|---|
| Pathogenic *E. coli* | 83 | 52 | USA | Holland et al. (1999) |
| Pathogenic *E. coli* (eae) | 7.3 | 153 | Germany | Krause et al. (2005) |
| ESC**E. coli* | 3 | 102 | Canada | Lefebvre et al. (2006) |
| ESBL** *E. coli* | 1 | | | |
| ESBL *E. coli* | 14 | 100 | Germany | Schaufler et al. (2015) |
| *C. jejuni* | 7 | 289 | Australia | Baker et al. (1999) |
| *C. coli* | 2 | | | |
| *C. upsaliensis* | 34 | | | |
| *C. jejuni* | 11 | 4 | Denmark | Damborg et al. (2004) |
| *C. jejuni* | 7 | 70 healthy dogs | Canada | Chaban et al. (2010) |
| *C. coli* | 0 | | | |
| *C. lari* | 0 | | | |
| *C. upsaliensis* | 43 | | | |
| *C. jejuni* | 46 | 65 diarrhoeic dogs | | |
| *C. coli* | 25 | | | |
| *C. lari* | 9 | | | |
| *C. upsaliensis* | 85 | | | |
| *Campylobacter* | 0 | 102 | Canada | Lefebvre et al. (2006) |
| *C. jejuni* | 1 | 249 | UK | Parsons et al. (2010) |
| *C. upsaliensis* | 37 | | | |

| Microorganism | Prevalence % | Study size | Country | References |
|---|---|---|---|---|
| *Staphylococcus aureus* (methicillin-resistant) | 0 | 102 | Canada | Lefebvre et al. (2006) |
| *Salmonella* (healthy dogs) | Range 0-3.5 | N/A | USA | Marks et al., (2011) |
| *Salmonella* (diarrhoeic dogs) | Range 0-3.6 | | | |
| *Salmonella* | 3 | 102 | Canada | Lefebvre et al. (2006) |
| Vancomycin-resistant enterococci | 0 | 102 | | |
| *Giardia* | 15.2 | Estimate of 4.3 million dogs | UK | Bouzid et al. (2015) |
| *Giardia* | 4 | 129 | USA | Wang et al. (2012) |
| *Giardia* | 2.6 | 77 | Japan | Yoshiuchi et al. (2010) |
| *Giardia duodenalis* | Pet store 39 | 69 | Canada | Uehlinger et al. (2013) |
| *Giardia duodenalis* | Vet clinic 38 | 78 | | |
| *Giardia duodenalis* | Shelter 6 | 62 | | |
| *Cryptosporidium* | 2 | 129 | USA | Wang et al. (2012) |
| *Cryptosporidium* | 3.9 | 77 | Japan | Yoshiuchi et al. (2010) |
| *Cryptosporidium* | Pet store 10 | 78 | Canada | Uehlinger et al., (2013) |
| *Cryptosporidium* | Vet clinic 8 | 62 | | |
| *Clostridium perfringens* | 34 | 95 | USA | Minamoto et al. (2014) |
| Enterotoxigenic *E. coli* | 48 | 104 | | |
| *Clostridium perfringens* | 84 healthy | 105 | Canada | Goldstein et al. (2012) |
| *Clostridium perfringens* | 91 diarrhoeic | 54 | | |
| *Clostridium difficile* | Range 10-21 | N/A | USA | Marks et al. (2011) |
| *Clostridium difficile* | 8 | 102 | Canada | Lefebvre et al. (2006) |

*ESC = Cephalosporinase *E. coli*

**ESBL = Extended-spectrum beta-lactamase

## 2.7 CAT FAECES

We were unable to find any data on *E. coli* or enterococci levels in cat faeces. There are some data on pathogens found in cat faeces, with a range of pathogenic *E. coli*, *Campylobacter*, *Cryptosporidium*, *Giardia*, *Clostridium*, *Salmonella* and *Toxoplasma gondii* all reported in cat faeces (TABLE 13 and TABLE 14 ). We could not find an estimate of daily faecal outputs per cat.

**TABLE 13: Quantitative data on levels of pathogens in cat faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output | Country | References |
|---|---|---|---|---|---|---|
| *Giardia* | 2.00E+04 | 10.1 | 345 | N/A | Australia | Yang et al. (2015) |
| *Cryptosporidium* | 3.50E+03 | 10 | | N/A | | |

**TABLE 14: Presence/absence data on pathogens in cat faeces**

| Microorganism | Prevalence % | Study size | Country | References |
|---|---|---|---|---|
| *C. jejuni* | 4 | 195 | Australia | Baker et al., (1999) |
| *C. coli* | 0 | | | |
| *C. upsaliensis* | 11 | | | |
| *C. jejuni* | 33 | 4 | Denmark | Damborg et al. (2004) |
| Pathogenic *E. coli* | 6.5 | 62 | Germany | Krause et al. (2005) |
| *Clostridium perfringens* | > 80 healthy & diarrhoeic | N/A | USA | Marks et al., (2011), |
| *Salmonella* | Range 0-8.6 | N/A | | |
| *Giardia* | 12 | Estimate of 250, 000 | UK | Bouzid et al. (2015) |
| *Giardia* | 44 | 18 | USA | Fayer et al. (2006) |
| *Giardia* | 2 | 55 | | Yoshiuchi et al. (2010) |
| *Cryptosporidium* | 12 | 250 | USA | Ballweber et al. (2009) |
| *Cryptosporidium* | 100 | 18 | USA | Fayer et al. (2006) |
| *Cryptosporidium* | 13 | 55 | Japan | Yoshiuchi et al. (2010) |
| *Toxoplasma gondii* | 0.4 | 252 | Switzerland | Berger-Schoch et al. (2011) |
| *Toxoplasma gondii* | 30 | 123 | USA | Dabritz et al. (2007) |
| *Toxoplasma gondii* | 1 | 326 | USA | Dabritz (2006) |
| *Toxoplasma gondii* | 0 | 63 | NZ | Langham and Charleston, (1990). |

## 2.8 WILDFOWL FAECES

The faeces of a range of wildfowl have been examined for indicators and pathogens including black swans (TABLE 15), ducks (TABLE 16), Canada geese (TABLE 17), seagulls (TABLE 17), pigeons (TABLE 19), and some unspecified wildfowl (TABLE 20).

**TABLE 15: Quantitative data on levels of indicators and pathogens in black swan faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output/ swan | Country | References |
|---|---|---|---|---|---|---|
| *E. coli* | 1.91E+06 | 94 | 80 | 7.50E+08 | NZ | Moriarty et al. (2011b) |
| Enterococci | 1.10E+06 | 79 | | 3.63E+08 | | |
| *Campylobacter* | 2.04E+02 | 45 | | 3.84E+04 | | |

**TABLE 16: Quantitative data on levels of indicators and pathogens in duck faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output/ duck | Country | References |
|---|---|---|---|---|---|---|
| *E. coli* | 9.40E+07 | 95 | 80 | 3.00E+10 | NZ | Moriarty et al. (2011b) |
| *E. coli* | | 89 | 82 | | USA | Fallacara et al. (2001) |
| *E. coli* | 1.00E+06 | | 1 composite of 4 duck faeces | 3.36E+08 | NZ | Murphy et al. (2005) |
| *E. coli* | 1.40E+07 | | 16 | 4.70E+09 | USA | Haack et al. (2003) |
| Faecal coliforms | 3.30E+07 | | Not specified | 1.11E+10 | England | Gould and Fletcher (1978) |
| Faecal streptococci | 5.40E+07 | | | 1.81E+10 | | |
| Enterococci | 1.01E+08 | 100 | 80 | 3.39E+10 | NZ | Moriarty et al. (2011b) |
| Enterococci | 5.00E+07 | | 13 | 1.68E+10 | USA | Haack et al. (2003) |
| Enterococci | 3.40E+05 | | 2 | 1.14E+08 | NZ | Anderson et al. (1997) |
| *Campylobacter* | 5.92E+01 | 29 | 80 | 5.77E+03 | NZ | Moriarty et al. (2011b) |
| *C. jejuni* | | 40 | 82 | | USA | Fallacara et al. (2001) |
| *Salmonella* | | 1 | | | | |
| *Cryptosporidium* | 4.80E+01 | 49 | 69 | 7.90E+03 | USA | Kuhn et al. (2002) |
| *Giardia* | 4.36E+02 | 28 | | 4.10E+04 | | |

**TABLE 17: Quantitative data on levels of indicators and pathogens in canada geese faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output/ bird | Country | References |
|---|---|---|---|---|---|---|
| *E. coli* | 3.62E+04 | 95 | 80 | 8.60E+06 | NZ | Moriarty et al. (2011b) |
| *E. coli* | | 63 | 357 | | USA | Fallacara et al. (2001) |
| *E. coli* | 3.60E+05 | | 63 | 6.71E+07 | USA | Middleton and Ambrose (2005) |
| *E. coli* | 4.20E+03 | | 16 | | USA | Haack et al. (2003) |
| Enterococci | 5.00E+02 | | 13 | | | |
| Enterococci | 2.51E+04 | | 80 | 6.15E+08 | NZ | Moriarty et al. (2011b) |
| Enterococci | 7.30E+05 | | 63 | 1.83E+08 | USA | Middleton and Ambrose (2005) |
| *Campylobacter* | 4.84E+03 | 40 | 80 | 4.84E+05 | NZ | Moriarty et al. (2011b) |
| *C. jejuni* | | 5 and 16 over two years | 318 | | USA | Rutledge et al. (2013) |
| *C. jejuni* | | 52 | 357 | | USA | Fallacara et al. (2001) |
| *Salmonella* | | 0 | | | | |
| *Cryptosporidium* spp. | | 5 | 80 | | NZ | Moriarty et al (2011b) |
| *Cryptosporidium* spp. | | 82 and 90 | 11 and 10 sites | | | Kassa et al. (2004) |
| *Cryptosporidium* spp. | | 23 | 209 | | USA | Zhou et al. (2004) |
| *C. parvum* | | 2 | | | | |
| *C. hominis* | | 1 | | | | |
| *Cryptosporidium* spp. (infectious *C. parvum* identified) | 3.7E+02 | 78% of sites | 9 sites | | USA | Graczyk et al. (1998) |
| *Giardia spp.* | 4.1E+02 | 100% of sites | | | | |

**TABLE 18: Presence/Absence and quantitative data on levels of indicators and pathogens in seagull faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output | Country | References |
|---|---|---|---|---|---|---|
| *E. coli* | 1.87E+07 | | 80 | 8.98E+08 | NZ | Moriarty et al. (2011b) |
| *E. coli* | 1.00E+07 | | Not specified | 5.00E+08 | USA | Fogarty et al. (2003) |
| Enterococci | 1.00E+06 | | | 5.00E+07 | | |
| Enterococci | 4.20E+03 | | 2 | 2.10E+05 | NZ | Anderson et al., (1997) |
| Enterococci | 8.96E+06 | 99 | 80 | 4.44E+08 | NZ | Moriarty et al. (2011b) |
| *Campylobacter* | 7.66E+02 | 59 | | 2.26E+04 | | |
| *C. lari* | | 2 | 205 | | Northern Ireland | Moore et al., (2002) |
| Urease-positive thermophilic *Campylobacter* | | 10 | 205 | | | |
| *C. jejuni* | | 1 | 205 | | | |
| *Cryptosporidium* spp. | | 0 | 205 | | | |

**TABLE 19: Presence/Absence data indicators and pathogens in pigeon faeces**

| Microorganism | Prevalence % | Study size | Country | References |
|---|---|---|---|---|
| *Salmonella enterica* | 3 | 277 | USA | Pedersen et al. (2006) |
| Shiga toxin *E. coli* (STEC) | 0 | 466 | | |
| Shiga toxin *E. coli* (STEC) virulence genes | 8 | 466 | | |

**TABLE 20: Data on levels and prevalence of indicators and pathogens in unspecified wildfowl faeces**

| Microorganism | Mean CFU/MPN /(oo)cysts $g^{-1}$ | Prevalence % | Study size | Daily Output | Country | References |
|---|---|---|---|---|---|---|
| Enterococci | 2.00E+04 | | 26 | | USA | Wright et al. (2009) |
| *C. jejuni* | | 26 | 180 | | UK | Brown et al. (2004) |
| *Campylobacter* spp. | | 50 | 449 | | USA | Fallacara et al. (2001) |

# 3. HUMAN SEWAGE

There are many studies of pathogens in both influent (raw sewage) and effluent (treated sewage) and only a sample of results from such studies are provided in this report in TABLE 21 and TABLE 22. Additional examples of concentration and prevalence are presented in TABLE 23, which looks at comparative studies between raw and treated sewage to assess removal rates of indicators and pathogens. We believe that there is additional unpublished data in New Zealand generated by district councils, regional councils and thesis dissertations, which future work should attempt to obtain.

## 3.1 STUDIES THAT DIRECTLY COMPARED REDUCTION VALUES FOR RAW AND TREATED WASTEWATER

Estimating the prevalence and abundance of pathogens in human sewage is complex and dependent on whether the sewage is raw or treated effluent and also the type of effluent treatment undertaken before discharge into the environment (Soller et al., 2010). TABLE 23 provides examples of the effect of treatment on various indicators and pathogens by presenting the $\log_{10}$ reduction in concentration of these microbes as they pass through the treatment process. In the study of Kitajima et al. (2014) the prevalence of viruses decreased with $\text{Log}_{10}$ reductions (<$\text{Log}_{10}$ 2.9). In addition, the potentially pathogenic viruses were still prevalent (range 25 to 92%) in effluent samples. Decrease in FIB levels ranged from removal rates of $\text{Log}_{10}$ 3.15 to 3.98 in a Canadian study by Shannon et al. (2007). A Swedish treatment site trialled three different types of treatment for removal of microbes from wastewater: T1) tertiary filtration, T2) membrane bioreactor (MBR), and T3) upflow anaerobic sludge blankets (UASB) (Ottoson et al., 2006). Treatment 2, the MBR, showed the highest log removal of indicators and viruses while Treatment 3, the USAB, showed the lowest removal rates for these organisms. The T2, MBR, produced an almost $\text{Log}_{10}$ 5.0 removal of *E. coli* and similar for enterococci. The removal rates for viruses were much lower, at less than $\text{Log}_{10}$ 2.0 removal even in the MBR system and viruses were still detected in effluent streams, ranging from 18 to 80% prevalence. Pathogenic protozoa showed the most effective removal rates for pathogens in the study with no *Giardia* or *Cryptosporidium* detected in the effluent from Treatments 2 and 3, even though *Giardia* was detected at 100% prevalence in influent.

As is evident from the removal rates outlined in TABLE 23, secondary treated effluent has the potential for a higher risk of illness than raw sewage due to the higher removal of indicator organisms during treatment compared with the greater resistance of pathogens such as viruses and protozoa. Therefore, the concentration of microbial indicators may be

within water quality guidelines but there is still the potential for infection by pathogens when treated wastewater is identified as the source of contamination (MfE and MoH 2003).

**TABLE 21: Quantitative data on levels of indicators and pathogens in untreated human sewage**

| Microorganism | Mean CFU/MPN/PFU 100 $mL^{-1}$ virus/(oo)cyst $L^{-1}$ | Range CFU/MPN/PFU 100 $mL^{-1}$ (oo)cyst $L^{-1}$ | Pre-Valence % | Study size | Country | References |
|---|---|---|---|---|---|---|
| *E. coli* | 1.00E+08 | | 100 | 13 | Honolulu | Yang et al. (2014) |
| *E. coli* | 3.60E+06 | | | 1 | Spain | Marín et al. (2015) |
| *Salmonella* spp | 0.00E+00 | | | 1 | | |
| *Cryptosporidium* spp. | 0.00E+00 | | | 1 | | |
| Faecal coliforms (Baseflow) | 1.70E+07 | | 100 | 252 | UK | Kay et al. (2008) |
| Faecal coliforms (Highflow) | 2.80E+06 | | 100 | 279 | | |
| Enterococci | 5.00E+06 | 1.0E+06-1.0E+07 | 100 | | NZ | Anderson et al. (1997) |
| F-specific coliphage | 1.58E+05 | | 100 | | Japan | Haramoto et al. (2015) |
| Enteropathogenic *E. coli* (EPEC) eae: | 3.99E+02 | | 100 | 13 | Honolulu | Yang et al. (2014) |
| Enterohemorraghic *E. coli* (EHEC) $stx_1$: | 1.5E+00 | | 15.40 | | | |
| *E. coli* (EHEC) $stx_2$: | 2.1E+00 | | 23.1 | | | |
| *ESBL *Enterobacteriaceae* | 2.40E+08 | 1.9-2.9E+08 | 100 | 21 | Poland | Korzeniewska and Harnisz (2013) |
| *Cryptosporidium* spp. | 8.7E+01 | 7.4E+01 – 1.0E+02 | 92 | 24 | USA | Kitajima et al. (2014a) |
| *Giardia* spp. | 5.60E+03 | 4.8 E+03 – 6.4 E+03 | 100 *G. intestinalis*** 17 Plant A; 67 Plant B | | | |
| *Cyclospora* spp. | 1.20E+04 copies | | 25 | | | |
| Human adenovirus | 5.01E+05 | | 100 | 10 | Japan | Haramoto et al. (2015) |
| Norovirus Genogroup I: | 1.45E+05 | | 90 | | | |
| Norovirus Genogroup II: | 7.94E+06 | | 60 | | | |

*ESBL = Extended spectrum beta-lactamase

**TABLE 22: Quantitative data on levels of indicators and pathogens in treated sewage**

| Treatment type | Microorganism | Mean CFU/PFU/MPN 100 $mL^{-1}$ (oo)cyst $L^{-1}$ virus GC $L^{-1}$ | Prevalence % | Study size | Country | References |
|---|---|---|---|---|---|---|
| Final effluent | *E. coli* | 1.70E+04 | | 1 | Spain | Marín et al. (2015) |
| Primary settled sewage | Faecal coliforms | 1.80E+07 | 100 | 60 | UK | Kay et al. (2008) |
| Settled septic tank | Faecal coliforms | 7.20E+06 | 100 | 42 | | |
| Trickling filter | Faecal coliforms | 4.30E+05 | 100 | 477 | | |
| Tricking/sand filter | Faecal coliforms | 2.10E+05 | 100 | 11 | | |
| Activated sludge | Faecal coliforms | 2.80E+05 | 100 | 261 | | |
| oxidation ditch | Faecal coliforms | 2.00E+05 | 100 | 35 | | |
| UV disinfection | Faecal coliforms | 2.80E+02 | 100 | 108 | | |
| Reedbed /grass plot | Faecal coliforms | 1.30E+04 | 100 | 71 | | |
| Treated wastewater | *E. coli* | 1.84E+02 | 75 | 24 | USA | Kitajima et al. (2014b) |
| oxidation pond effluent | Enterococci | 1.00E+03 | | | NZ | Anderson et al. (1997) |
| Treated wastewater | F-specific coliphage | 3.2E+02 | | 10 | Japan | Haramoto et al. (2015) |
| effluent (clarifiers) | *E. coli* O157 | - | 7 | 44 | France | Bertrand and Roig (2007) |
| Treated Sewage | ESBL isolates of *Enterobacteriaceae* | Range 6.0E+01-3.5E+06 | 100 | 21 | Poland | Korzeniewska and Harnisz (2013) |
| Activated sludge or biological trickling filter | *Cryptosporidium* spp. | 1.25E+01 | 83 | 24 | USA | Kitajima et al. (2014a) (oo)cyst $L^{-1}$ |
| | *Giardia* | 1.12E+02 | 100 | | | |
| | *Cyclospora* spp. | - | 13 | | | |
| Treated Sewage Virus concentrations reported in $\log_{10}$ gene copies $L^{-1}$ | Human adenovirus | 1.29E+04 | 100 | 10 | Japan | Haramoto et al. (2015) |
| | Norovirus Genogroup I | 1.82E+03 | 70 | | | |
| | Norovirus Genogroup II | 5.25E+04 | 30 | | | |

**TABLE 23: Comparison studies of influent and effluent at sewage treatment plants**

| Microorganisms | Mean $log_{10}$ gene copies 100 $mL^{-1}$ Or comparable prevalence | Reference and Comments |
|---|---|---|
| Viruses | Norovirus<br>**Influent** Genogroup (G) I and II ~100% in both Plant A and B<br>**effluent** Genogroup (G) I and II, 75% in both A and B,<br>G IV: 67% Plant A; 25% Plant B | **Kitajima et al. (2014)**<br>US study of two wastewater treatment plants with influent (n = 12 from each plant) and effluent samples (n = 12 from each plant) collected monthly over a one year period. Plant A used activated sludge process and Plant B a biological trickling filter tower, and both used chlorination for disinfection. Isolation was by an electronegative filter method and identification by quantitative PCR for 11 viruses which included rotavirus, adenovirus, enterovirus, polyomaviruses and the genogroups of norovirus.<br>Norovirus had the highest viral reduction during treatment at both plants (GII $log_{10}$ reduction 2.1 at A and 2.9 at B; followed by GIV norovirus, $log_{10}$ reduction 1.7 at A and 2.7 at B) compared with all other viruses. Both treatment plants had statistically similar reduction rates for all viruses, with the exception of enterovirus which had greater reduction at Plant B (bio trickling filter). |
| | Enterovirus<br>**Influent** 100% Plants A and B;<br>**Effluent** 58% (A); 92% (B) | |
| | Adenovirus<br>**Influent** 100% (A) and 83% (B);<br>**Effluent** 58% (A); 92% (B) | |
| *E. coli* | **Influent**: 7.18<br>**Final Effluent**: 3.20<br>$Log_{10}$ reduction range: 3.52-3.98 | **Shannon et al. (2007)**<br>Canadian study of five stages of one wastewater treatment plant using real-time qPCR methods. Concentrations measured in $log_{10}$ gene copies (GC)/100 mL.<br>*Listeria monocytogenes* (no data given) and *Aeromonas hydrophila* (4.32 $log_{10}$ GC/100 mL) were only detected in influent, thereafter, not detected at any stage in treatment process.<br>*E. coli* O157:H7, *Salmonella* spp., *Staphylococcus aureus*, *Legionella monocytogenes* and *Helicobacter pylori* were not detected in influent or at any stage of treatment |
| *C. perfringens* | **Influent**: 5.85<br>**Final Effluent**: 2.70<br>$Log_{10}$ reduction range: 3.15-3.39 | |
| *Enterococcus faecalis* | **Influent**: 4.66<br>**Final Effluent**: 1.42<br>$Log_{10}$ reduction: 3.24 | |
| *Pseudomonas aeruginosa* | **Influent**: 4.38;<br>**Primary effluent**: 2.22. Thereafter, not detected during treatment process or final effluent | |

**TABLE 23 continued: Comparison studies of influent and effluent at sewage treatment plants**

| Microorganisms | Mean MPN $L^{-1}$ | Prevalence | Reference and Comments |
|---|---|---|---|
| *Giardia* | **Influent** 1.3E+03 cysts<br>**Effluent** T1: 0.4 cysts | **Influent** 100%;<br>**Effluent** T1 11%; T2 0%; T3 0% | **Ottoson et al. (2006)**<br>Swedish study of the inlet wastewater and effluent from an experimental treatment plant using one of three treatment (T1, T2, T3) regimes<br>T1) tertiary filtration,<br>T2) membrane bioreactor (MBR)<br>T3) upflow anaerobic sludge blankets (UASB).<br>Protozoan were analysed using immunofluorescence detection and viruses by RT-PCR. There was no speciation of protozoa to attribute sources to humans or to animals. The PCR method for enterovirus assayed all types of enteroviruses and is therefore an index of enterovirus removal rather than direct risk evaluator of pathogenic enteroviruses. Removal quantified as a $\log_{10}$ reduction. Norovirus more frequently detected in winter samples (86%, n =7).<br><br><u>Mean removal rates for indicators for each treatment regime</u><br><br>*E. coli*: $\log_{10}$ removal T1, 3.23; T2, 4.97; T3 1.97<br><br>Enterococci: $\log_{10}$ removal T1,3.17; T2, 4.52; T3 1.75<br><br>*C. perfringens*: $\log_{10}$ removal T1, 2.38; T2, 3.04; T3 0.66<br><br>Somatic coliphages: $\log_{10}$ removal T1, 2.32; T2, 3.08; T3 0.76<br><br>F-RNA phage: $\log_{10}$ removal T1, 3.47; T2, 3.78; T3 2.38 |
| *Cryptosporidium* | **Influent** 5.0E+01 oocysts<br>**Effluent** T1: 0.13 oocysts | **Influent** 21%;<br>**Effluent** T1 6%; T2 0%; T3 0%<br>T1 and T2 showed 97.4% and >96.4% removal (resp.) | |
| Enteroviruses | **Influent** 1E+04<br>**Effluent**<br>T1 and T2 <2.1E+02<br>T3: 3.5E+03 | **Influent** 78%, n =23<br>**Effluent** T1 36%; T2 29%; T3 80%<br>T1 and T2 showed 98.0% and 98.4% removal whereas T3 showed 65% removal of viruses after treatment | |
| Norovirus | **Influent** 3.0E+02<br>**Effluent**<br>T1 and T2 <3.5E+01<br>T3: 3.0E+02 | **Influent** 36.4%, n = 22<br>**Effluent** T1 18%; T2 18%; T3 40%<br>T1 and T2 showed 89 % and 93% (resp.) removal whereas T3 showed zero removal of viruses after treatment | |

# 4. COMPARISON BETWEEN SOURCES

There are extensive data on microorganisms in livestock, particularly beef cattle and dairy cows, of which we have provided a subset. The data provided in sections 2 and 3, however, does highlight the limited knowledge we have on the presence and levels of microorganisms in many of the other animal sources of interest such as avian species. A full QMRA comparing sources is beyond the scope of this report and is, based on the data available, potentially misleading. However, we have in TABLE 24, presented a comparison of the levels of microorganisms from a range of sources expressed as microorganisms per animal per day. Young livestock (lambs and calves) have the highest daily shedding potential for *Campylobacter* and *Cryptosporidium* and sheep have greater daily shedding of these two pathogens compared with the larger dairy cows, although not in comparison with *Cryptosporidium* shed by beef cattle. In general, avian species have two to three orders of magnitude reduced daily outputs of *E. coli* and *Campylobacter* compared with livestock.

It should be noted that many studies report prevalence and quantitative data at the genus level, for example, *Cryptosporidium*, and then test a limited subset of the isolates for specific pathogens known to be zoonoses, such as *C. parvum*. Therefore, the data for prevalence and concentration of microorganism will include a proportion that are not zoonotic and potentially do not represent a human health risk. Another example is infections of cattle by *Giardia duodenalis*. Most of the *G. duodenalis*belong to the non-zoonotic Assemblage E compared with the human infective Assemblage A (Atwill et al., 2012).

In TABLE 25, the numbers from TABLE 24 have been normalised to represent the concentration of indicators and pathogens that could be associated with 1000 CFU/g *E. coli* identified in the faeces of a particular animal species. There are approximately ten to 200-fold higher *E. coli* levels in livestock, black swans and gulls compared with enterococci. In contrast, Canada geese have tenfold higher enterococci levels compared with *E. coli*. In general, there are 1000-fold lower levels of *Campylobacter* in livestock compared with *E. coli* and even lower levels in most avian species, except for Canada geese, which have approximately 100-fold lower *Campylobacter* than *E. coli*. The potential for faecal pollution from Canada geese to cause illness in humans has been highlighted in a recent review (Gorham and Lee,2015). Levels of *Cryptosporidium* are at least $10^4$ fold less than *E. coli* in livestock, and even less in ducks. *Giardia* are present in very low levels compared with *E. coli* ($10^5$ and$10^6$ fold less concentration in ducks and livestock, respectively).

The prevalence of indicators and pathogens in mammals and birds is presented in TABLE 26. *E. coli* and enterococci are identified in >93% of the faeces of all livestock, with a lower

overall prevalence in avian species ranging from 63 to 95% for *E. coli* and 79 to 100% for enterococci.

The highest prevalence of *Campylobacter* was seen in lambs at 84% with the next highest in dairy cows, which ranged from 7- 64%. Calves and lambs are born *Campylobacter*-free but are rapidly colonised from the farm environment after birth (Gannon et al., 2002; Stanley and Jones, 2003). In general, calves and lambs are identified as shedding higher concentrations of *Campylobacter* compared with adult animals.

*Campylobacter* in avian species ranged from 29-52%, except for gulls at 1% prevalence.

*Cryptosporidium* was identified in 12-100% of cat faeces, however the 100% prevalence was from a smaller study size compared with the lower prevalence (12 and 13%) from a larger study size. The next highest prevalence of *Cryptosporidium* was seen in young livestock with ranges of 0.9-77% in lambs and 10-80% in calves.

*Giardia* was identified in up to 37% of sheep and lambs, only 5% in calves and 0-26% in beef cattle with no data on dairy cows. Maximum prevalence of *Giardia* in dogs and cats was 39 and 44%, respectively. Ducks were identified as carriers of *Giardia* at 28% prevalence, but there were no data on other avian species.

Pathogenic *E. coli* were identified at a maximum prevalence of 83% in dogs and 46% in beef cattle and 21% in dairy cows but at less than 8% prevalence in individual sheep and lambs. *E. coli* O157:H7 has been identified in widely varying concentrations in the faeces of sheep and cattle and tends to be sporadic with levels fluctuating between <100 to $10^6$ CFU/g of faeces (Atwill et al., 2012; Chase-Topping et al., 2007; Matthews et al., 2006). These fluctuations have led to the term “super-shedders” for those livestock that carry >$10^3$-$10^4$ CFU/g of *E. coli* O157:H7. In addition, the duration of shedding varies widely, with individual animals shedding for a few days or weeks and others up to six months. It has been estimated that 80% of transmission of *E. coli* O157:H7 is from 20% of the most infectious livestock (Matthews et al., 2006). A study of shiga-toxin producing *E. coli* (STEC) identified that 58.3% of 319 sheep and cattle faeces in New Zealand carried an STEC gene (Cookson et al., 2006b). Another study identified the same genetic isolates of STEC from cattle and human clinical samples using molecular subtyping techniques, illustrating that livestock in NZ can be a reservoir of disease-causing STEC in the human population (Cookson et al., 2006a).

Data for pathogenic *E. coli* in avian species were limited to 8% prevalence in pigeons from one study. *Salmonella* prevalence in all mammals and birds was generally low for both NZ

and international studies, ranging from 0 - 9%, except for dairy cows, which ranged between 10 and 56% prevalence.

*Toxoplasma gondii* is a protozoan that completes its sexual life cycle phase in the intestinal tract of cats and other felines, resulting in the excretion of oocysts in faeces. Infections by *Toxoplasm*a are usually asymptomatic but the immunocompromised can become seriously unwell. Pregnant women are particularly susceptible to toxoplasmosis and infection of the foetus may result in foetal death. *Toxoplasm*a was identified in cats in four studies at a prevalence ranging between 0 - 30%. However, three of these studies with samples sizes ranging between 63 and 252 had a prevalence of ≤1%.

In general, viruses in animals are not considered to be zoonotic because it is believed that there are strong barriers to prevent viruses crossing between animal species. A brief search of zoonotic viruses in the literature revealed some concerns about the potential for zoonotic viruses (Cavirani, 2008; Kallio-Kokko et al., 2005). Many of these viruses are not of common concern in the New Zealand environment but have been shown to cause disease in Africa and other continents. The West Nile virus (WNV) is an example of a virus introduced to North America in the late 1990s, which had rapid dissemination via mosquito vectors from various animal hosts. The introduction of WNV led to 14,000 cases of illness and 586 deaths being recorded in the USA up till 2005 (Kallio-Kokko et al., 2005).

There has been a recent research focus on the zoonotic potential of several viruses that cause gastroenteritis illness (GI) in humans such as norovirus and rotavirus and whether the specific viral types found in animal reservoirs including dogs, cattle, pigs and sheep, have the potential to cross the host-species barrier and cause illness in humans (Medici et al., 2015; Widdowson et al., 2005; Wolf et al., 2009). Some studies have highlighted that these viral species with animal reservoirs have caused infection in humans (but not necessarily illness) as evidenced by the detection of antibodies against bovine norovirus in humans (Bank-Wolf et al., 2010; Widdowson et al., 2005).

**TABLE 24: Quantitative data on average daily output per animal of indicators and pathogens in mammals and birds**

| | *E. coli* | Enterococci | *Campylobacter* | *Cryptosporidium* | *Giardia* |
|---|---|---|---|---|---|
| Sheep | 2.51E+10 | 1.02E+09 | 1.80E+07 | 9.48E+05 | 4.21E+03 |
| Lambs | 4.53E+11 | 1.08E+10 | 2.02E+08 | 4.61E+06 | 1.00E+05 |
| Dairy cows | 2.01E+09 | 1.04E+07 | 6.81E+06 | 5.95E+04 | |
| Calves | | | | 1.50E+10 | 1.00E+03 |
| Beef cattle | | 2.66E+06 | | 1.11E+06 | 6.63E+05 |
| Dogs | | 3.30E+09 | | | |
| Cats | | | | 2.00E+04 | 3.50E+03 |
| Black swans | 7.50E+08 | 3.63E+08 | 3.84E+04 | | |
| Ducks | 1.17E+10 | 1.70E+10 | 5.77E+03 | 7.90E+03 | 4.10E+04 |
| Canada geese | 3.80E+07 | 3.99E+08 | 4.84E+05 | | |
| Gulls | 7.00E+08 | 1.65E+08 | 2.26E+04 | | |

**TABLE 25: Levels of indicators and pathogens relative to *E. coli* concentration normalised to 1000 CFU/g**

| | *E. coli* | Enterococci | *Campylobacter* | *Cryptosporidium* | *Giardia* |
|---|---|---|---|---|---|
| **Sheep** | 1000 | 41 | 0.72 | 0.038 | 0.0002 |
| **Lambs** | 1000 | 24 | 0.45 | 0.010 | 0.0002 |
| **Dairy cows** | 1000 | 5 | 3.39 | 0.030 | 0.0000 |
| **Black swans** | 1000 | 484 | 0.0511 | | |
| **Ducks** | 1000 | 1453 | 0.0005 | 0.0007 | 0.0035 |
| **Canada geese** | 1000 | 10500 | 12.7 | | |
| **Gulls** | 1000 | 236 | 0.0323 | | |

**TABLE 26: Comparison of prevalence of indicators and pathogens in animal faeces**

| | Sheep | Lambs | Dairy Cows | Calves | Beef cattle | Dogs | Cats | Goats | Black Swans | Ducks | Canada Geese | Gulls | Pigeons |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| ***E. coli*** | 100% | 100% | 99.5% | 100% | | | | | 94% | 89-95% | 63-95% | | |
| **Enterococci** | 100% | 100% | 93.3% | 100% | | | | | 79% | 100% | | | |
| ***Campylobacter*** | 25-30% | 81% | 7-64% | | | 0-46% | 0-33% | | 45% | 29-40% | 40-52% | 1% | |
| ***Cryptosporidium*** | 3-25% | 0.9-77% | 0.6-7.3% | 10-80% | 8% | 2-10% | 12-100% | 8-9.5% | | 49% | 1-23% | 0% | |
| ***Giardia*** | 19%-37% | 1-37% | | 4.5% | 0-26% | 2.6-39% | 2-44% | 20-36% | | 28% | | | |
| **Pathogenic *E. coli*** | 1-7% | 4% | 1-21% | | 1.6-46% | 1-83% | | | | | | | 8% |
| ***Salmonella*** | 0% | 1.9% | 9.6-56% | | 0.3- 6% | 0-3.6% | 0-8.6% | | | 1% | 0% | | 3% |
| ***Toxoplasma gondii*** | | | | | | | 0-30% | | | | | | |

# 5. CONCLUSIONS

Epidemiological studies exploring the relationship between infectious illness and the microbial quality of recreational waters impacted by non-human faecal sources have produced ambiguous results. This uncertainty associated with non-human faecal pollution and its impact on human health has raised concern when epidemiological studies tried to assess health impacts based on the knowledge gained from the effects of human faecal contamination. This knowledge gap led to a seminal paper by Jeffrey Soller and colleagues on using quantitative microbial risk assessment (QMRA) to explore the human health risks from recreational water impacted by pollution from either human, gull, chicken, pig or cattle faeces (Soller et al. 2010). The figure reproduced below from that paper shows that in water containing the same level of faecal indicator from each source there is potentially a lower risk of illness when the water is impacted by chicken, gull and pig faecal material, than human faecal matter. In contrast, there are similar risks from illness between faecal contamination derived from cattle (beef and dairy) and that from humans.

Soller et al. (2010) noted that a key limitation in their study was the limited amount of data on the levels of indicator and pathogens in the sources they examined. This USA study (Soller et al., 2010) did not consider all of the sources important in New Zealand such as sheep, domestic pets, and a range of other wildfowl beyond seagulls. As this current report illustrates there are a lack of quality data on the levels of indicator organisms and pathogens in many of the faecal sourcesto New Zealand waters.

In a 2014 paper, Soller et al. extended their initial QMRA work. The starting point was that 35 enterococci /100ml provided an acceptable level of risk, and was based on the source of those enterococci being human faecal matter. The risk of illness was defined as 36 gastrointestinal illnesses (GI) per 1000 swimmers. Using QMRA modelling they estimated the level of enterococci that would provide an equivalent level of protection if those enterococci were from non-human sources. Their analysis suggested that if the enterococci are entirely from chicken, pig or gull sources, the equivalent level of enterococci that would provide the same protection, ranged from threefold to 50 times higher (TABLE 27).

Another key finding from the Soller et al. (2014) study was that where there are mixed sources of contamination identified, then the risk is dependent on the most potent source of faecal contamination. The risk of illness decreased as the contribution from human sources reduced from 100%, so that by 30% human attribution to FIB levels, the predicted risk of infection had lowered by 50% compared with the risk if all detected FIB were derived from human sources. Thereafter, the risk declined more rapidly, so that at ≤20% human

contribution to the mixed faecal source, the predicted risk was five times lower compared with a pure human source. These predictions were based on the faecal source being from recent faecal events and did not account for the differential die-off between FIB and pathogens. This preferential decay of FIB was seen in the treated wastewater data (TABLE 23), which illustrated higher log removal of FIB in comparison to pathogenic protozoa and viruses. The fact that the most potent faecal source (human or cattle, Soller et al. 2010) was the driver of predicted risk is of particular relevance to rural areas where ruminant agricultural sources are detected often in conjunction with avian sources. Therefore, unless the ruminant signal accounts for less than 30% of the mixed contamination, then the health risk is 50 to 100% of the risk associated with a solely ruminant faecal source.

Monitoring the research into the emergence of viral zoonoses in livestock and other animal reservoirs in the New Zealand environment, particularly for the GI causing norovirus and rotavirus species is required for the future.

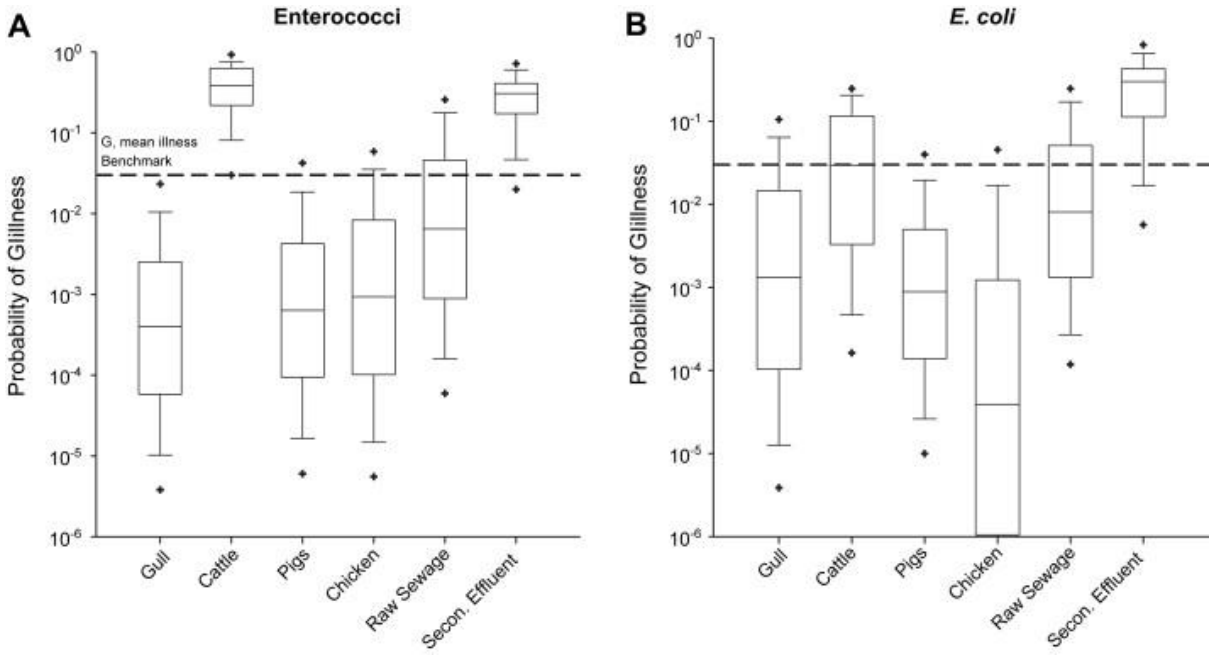


**Figure 1. Probability of GI illness from ingestion of water containing fresh faecal pollution at densities of 35 cfu 100mL$^{-1}$ ENT (3A) and 126 cfu 100mL$^{-1}$ *E. coli* from a range of sources. Figure reproduced from Soller et al. (2010).**

**TABLE 27: Predicted median enterococci densities that correspond to GI illness levels of 0.036, analogous to 36 people out of 1000 becoming ill if they ingest recreational water containing these levels of enterococci (reproduced from Soller et al., 2010)**

| Human contribution | 0% | 10% | 20% | 30% | 50% | 70% | 100% |
|---|---|---|---|---|---|---|---|
| **Non-human contribution** | **100%** | **90%** | **80%** | **70%** | **50%** | **30%** | **0%** |
| Pig | 607 | 278 | 164 | 114 | 70 | 50 | 35 |
| Chicken | 103 | 95 | 87 | 79 | 62 | 49 | 35 |
| Gull | 1947 | 339 | 174 | 116 | 70 | 50 | 35 |
| *Non-pathogenic source | - | 350 | 175 | 117 | 70 | 50 | 35 |

*For example, environmental or naturalised enterococci

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