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Estimated human health risks from exposure to recreational waters impacted by human and non-human sources of faecal contamination☆

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ABSTRACT

This work was conducted to determine whether estimated risks following exposure to recreational waters impacted by gull, chicken, pig, or cattle faecal contamination are substantially different than those associated with waters impacted by human sources such as treated wastewater. Previously published Quantitative Microbial Risk Assessment (QMRA) methods were employed and extended to meet these objectives. Health outcomes used in the analyses were infection from reference waterborne pathogens via ingestion during recreation and subsequent gastrointestinal (GI) illness. Illness risks from these pathogens were calculated for exposure to faecally contaminated recreational water at the U.S. regulatory limits of 35 cfu 100 mL^{-1} enterococci and 126 cfu 100 mL^{-1} *Escherichia coli*. The probabilities of GI illness were calculated using pathogen dose-response relationships from the literature and Monte Carlo simulations. Three scenarios were simulated, representing a range of feasible interpretations of the available data. The primary findings are that: 1) GI illness risks associated with exposure to recreational waters impacted by fresh cattle faeces may not be substantially different from waters impacted by human sources; and 2) the risks associated with exposure to recreational waters impacted by fresh gull, chicken, or pig faeces appear substantially lower than waters impacted by human sources. These results suggest that careful consideration may be needed in the future for the management of recreational waters not impacted by human sources.

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1. Introduction

Since the 1950s, numerous epidemiology studies have been conducted worldwide to evaluate the association between recreational water quality and adverse health outcomes including gastrointestinal (GI) symptoms; eye infections; skin irritations; ear, nose, and throat infections; and respiratory illness (Prüss, 1998; Wade et al., 2006; Zmirou et al., 2003).

☆ The views expressed in this article are those of the authors and do not necessarily reflect the views or policies of the U.S. Environmental Protection Agency.

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Together these studies indicate that the rates of some adverse health outcomes are higher in swimmers compared with non-swimmers and that faecal indicator bacteria (faecal streptococci/enterococci and *Escherichia coli*, in particular) can be used to predict GI and in some cases, respiratory illnesses from exposure to recreational waters (Prüss, 1998; Wade et al., 2006; Zmirou et al., 2003).

Most bathing water epidemiology studies have investigated municipal wastewater effluent-impacted waters, and thus, the relative human health risks from exposure to recreational waters impacted by non-human sources are not as well understood. Sinton et al. (1998) reviewed available data to differentiate the relative health risks associated with human and animal faecal material and reported that reliable epidemiologic evidence was lacking for non-human impacted waters. More recently, the few studies undertaken provide mixed views. On one hand, Colford et al. (2007) reported that the incidence of swimmer illness was not associated with any of the traditional bacterial indicators at a marine beach with likely avian contamination. Fleisher et al. (2010) found no relationship between GI illness and increasing levels of enterococci at a subtropical marine water without known sources of sewage. Calderon et al. (1991) found no statistically significant association between swimmers' illness risk and animal faecal contamination in a freshwater pond. However, McBride (1993) suggested that if more swimmers had been included in the Calderon et al. (1991) study, achieving statistically significant results would have been possible. Finally, Dwight et al. (2004) demonstrated that surfers exposed to Southern California urban run-off had higher illness rates than surfers exposed to Northern California rural runoff, but detailed source characterizations were not provided. On the other hand, a marine bathing study in New Zealand (McBride et al., 1998) indicated that illness risks posed by animal versus human faecal material were not substantially different. In a study conducted in waters impacted by urban runoff, Haile et al. (1999) reported rates of illnesses in Southern California similar to those conducted in waters contaminated with municipal wastewater. However, the urban runoff source was known to have human sources of faecal contamination (Colford et al., 2007). The results from a marine water study in Hong Kong (Cheung et al., 1990) and a German freshwater study (Wiedenmann et al., 2006) are more difficult to interpret regarding risks from human versus non-human sources because in both studies, the analyses combined the results from sites with different predominant contamination sources. Taken together, these studies indicate that the health risks associated with swimming in non-sewage impacted waters remain equivocal.

The U.S. Environmental Protection Agency's (EPA) recreational water quality criteria do not differentiate between faecal sources (U.S. EPA, 1986). While new EPA recreational water criteria will be issued in 2012, the current situation is that waters impacted by non-human faecal contamination sources are considered as hazardous as human-derived sources. The World Health Organization's (WHO) recommended approach for classifying the water quality of recreational waters is based on the premise that the measure of bacterial (intestinal enterococci) indicators of faecal contamination should be interpreted in combination with evidence of the presence or absence of human faecal contamination (i.e. sanitary significance). The WHO approach assumes that in general, sources other than human faecal contamination are less of a risk to human health (WHO, 2003). In fact, WHO indicates that "due to the species barrier, the density of pathogens of public health importance is generally assumed to be less in aggregate in animal excreta than in human excreta and may therefore represent a significantly lower risk to human health" (WHO, 1999). From a regulatory and management perspective, it is important to understand whether exposure to recreational waters impacted by non-human sources corresponds to significantly different illness risks than human impacted waters.

In previous work, we presented a QMRA approach for comparing the potential health risk from exposure to recreational waters impacted by two sources of faecal contamination (Schoen and Ashbolt, 2010). Seagull faeces and primary sewage effluent were compared at the same density of faecal indicator bacteria (FIB) with the result of a lower predicted illness risk from seagull impacted waters (Schoen and Ashbolt, 2010). We also used QMRA to understand more fully the reported results from the 2003–2004 Great Lakes epidemiologic studies (Soller et al., submitted for publication). Those QMRA results indicate that human enteric viruses were the etiologic agents of primary concern during the epidemiologic studies and that using *Norovirus* as a reference pathogen likely accounted for the vast majority of gastrointestinal (GI) illness risk. The present study builds upon the previous work summarized above and was undertaken as an initial step to determine whether the relative risks from exposure to recreational waters impacted by gulls, chickens, pig, and/or cattle are substantially different than those associated with human impacted waters.

2. Methods

A QMRA-based approach was employed to predict estimated risks of infection and illness from ingestion of recreational water that is assumed to be contaminated with faeces from a range of human and non-human sources (secondary disinfected wastewater effluent, primary wastewater effluent, cattle, pig, chicken and gull faeces). The estimated risks were calculated for a hypothetical waterbody that contains sufficient contamination from each source so that the geometric mean FIB densities are at the U.S. recommended criteria for recreational marine and freshwaters (35 cfu 100 mL^{-1} enterococci and 126 cfu 100 mL^{-1} *E. coli* respectively). Epidemiology studies indicate that these indicator densities would result in highly credible gastrointestinal illness (HCGI) rates of approximately 0.01–0.02 (1–2 illnesses per hundred recreation events) for waters impacted by treated effluent (U.S. EPA, 1986). A recent redefinition of HCGI that excludes the need for fever (Colford et al., 2007; Wade et al., 2006, 2008) would result in an equivalent benchmark risk of approximately 0.03–0.04. Although undisinfected primary effluent is rarely discharged to recreational waters in the USA, this faecal contamination source was included here to evaluate the potential health implications of poorly treated effluent, leaking sewerage infrastructure, bather shedding, and/or poorly operating septic systems.

2.1. Pathogens included

The pathogens used in this study include *Norovirus*, *Cryptosporidium* spp., *Giardia lamblia*, *Campylobacter jejuni*, *Salmonella enterica* and *E. coli* O157:H7. Together these pathogens (reference pathogens) make up a large portion of all non-foodborne illnesses from known pathogens in the US (calculated based on data from Mead et al., 1999), are representative of the fate and transport of other pathogens potentially of concern from the waterborne route of exposure (Ferguson et al., 2009) and have corresponding dose-response relationships in the peer reviewed literature. The use of reference pathogens is an accepted practice in the field of QMRA (Roser et al., 2007; Soller et al., 2003, 2006) to represent the possible environmental fate and transport of members of each microbial group as well as the infectivity of known members of each group (WHO, 2004). In this study, only one reference virus, *Norovirus*, was selected for human sources of contamination as previous work indicated it represented the dominant GI illness risk for recreators swimming in waters impacted by secondary treated disinfected wastewater effluent (Soller et al., submitted for publication).

2.2. Population included

Individual level risks for recreators/swimmers from the general population were considered. However, the risks faced by children may be different than those faced by adults, due to potentially different contact times in water, different ingestion rates of water, and different susceptibility to infection for some pathogens (Gerba et al., 1996). Susceptibility to infection may also be substantially different for immunocompetent and immunocompromised populations. Given the lack of data on the differences between sensitive sub-populations and the general population (Parkin et al., 2003), and the limited number of dose-response relations for sensitive sub-populations, we did not specifically address children or other potentially sensitive sub-populations.

2.3. Health outcomes

Health outcomes were first estimated as infections for each reference pathogen following water ingestion during recreation and then, conditional on infection, as subsequent GI illness. Other potential health outcomes from primary contact recreation (inhalation, dermal, conjunctive exposures) were not included in this analysis, nor were more severe yet much rarer health outcomes that may result from exposure to enteric pathogens. For example, *E. coli* O157:H7 infections can lead to symptoms ranging from mild GI upset to bloody diarrhoea and haemolytic uremic syndrome (HUS). Young children and the immunocompromised are most at risk for HUS (Boyce et al., 1995).

2.4. Scenarios modeled

Reference pathogen doses were derived as a function of the density of the faecal indicator in the water from each of the specific sources as described by Schoen and Ashbolt (2010). The calculation of the pathogen dose is based on independent Monte Carlo samples from the observed ranges of pathogen and faecal indicator densities in faecal waste. This sampling scheme does not require a specific relationship between the indicator and pathogen in the faecal waste or in the receiving water. The dose, μ_{rp}^{S} of each reference pathogen from each source was calculated as follows (Equation (1)).

$$\mu_{rp}^{S} = \frac{C_{FIB}}{F_{FIB}^{S} \times 100} \times R_{rp}^{S} \times p_{rp}^{S} \times I_{rp}^{S} \times V \quad (1)$$

Where

S is the faecal contamination source (raw sewage, secondary disinfected effluent, fresh cattle, pig, chicken or gull faeces);
C_{FIB} is the waterbody density of enterococci or *E. coli* using a culture method (cfu 100 mL^{-1});
F_{FIB}^{S} is the density of bacterial indicators in faeces (wet mass) (cfu g^{-1}) or in sewage (cfu L^{-1});
R_{rp}^{S} is the density of pathogen species in faeces (wet mass) (number of pathogens or genomes g^{-1}) or in sewage (number of pathogens or genomes L^{-1});
p_{rp}^{S} is the fraction of human-infectious pathogenic strains from source S;
I_{rp}^{S} is the prevalence of infection in the non-human source[1] (proportion of animals shedding the pathogen); and
V is the volume of water ingested (mL).

A detailed literature search was conducted to find appropriate values for each of the model parameters shown in Equation (1). The literature search strategy entailed searches in multiple databases for studies reporting the prevalence (as % of manure samples) and abundance (as organisms per g wet weight of manure) of the reference pathogens, enterococci (ENT) and *E. coli* (EC) in solid, fresh cattle manure, pig manure, chicken litter, and sewage.

Since many of the parameters used in estimating dose have natural variability, the QMRA process accounted for variability using a Monte Carlo simulation approach, with each run consisting of 10,000 trials. Log-uniform distributions were employed to characterize parameter value ranges to capture the substantial natural variability in the model parameters. The use of log-uniform distributions for highly variable or uncertain parameter ranges is consistent with previous QMRA work (Eisenberg et al., 1996, 1998). The ingestion of water was modeled as a lognormal distribution (Dufour et al., 2006). The relative fraction of human-infectious strains of each of the reference pathogens in the non-human sources is a highly uncertain model parameter. Insufficient data were available to confidently assign quantitative values to this model parameter. Thus, a qualitative assessment of this parameter was used where categorical values of low (L), medium (M) or high (H), were assigned to each pathogen for each non-human source. The qualitative potential for human infection was based upon the prevalence of known human-infectious species/strains/serotypes/isolates in animal faeces and our best professional judgment. The mid-points of the ranges of 0–33% for L, 33–66% for M, and 67–100% for H were then used as point estimates in the analysis and the impact of these assignments was explored through sensitivity analysis (Runs 1–3, as described below).

[1] For human sources I_{rp}^{S} is assumed to be 1.0 because the indicator and pathogen data are from sewage not individual faecal samples, and therefore already accounts for the pathogen prevalence 25.

The output from each trial is a distribution of the pathogen dose for the selected faecal indicator in the waterbody (C_{FIB}). Separate analyses were conducted to predict the risk from pathogens for waterbodies at the specified levels of ENT or EC. Three scenarios (runs) were simulated, representing a range of feasible interpretations of the available data. Each run was unique in relation to two uncertain parameters, the prevalence of infection among individuals or samples I^S_{rp} and the fraction of human-infectious strains of each of the reference pathogens in the non-human sources p^S_{rp}. Run 1 represents the most conservative health-protective assumptions, followed subsequently by Runs 2 and 3 as follows:

Run 1 The prevalence of infection in each of the sources and the proportion of human-infectious pathogenic strains from each source was assumed to be 100%;

Run 2 The prevalence of infection in each of the sources was assumed to be as shown in Table 2 and the proportion of human-infectious pathogenic strains from each source was assumed to be 100%; and

Run 3 The prevalence of infection in each of the sources and the proportion of human-infectious pathogenic strains from each source was assumed to be as shown in Table 2.

2.5. QMRA model selection

The probabilities of infection (P_{inf}) and subsequent illness (P_{ill}) (Teunis et al., 1996) for individuals were calculated using dose-response relationships (Haas et al., 1999; Medema et al., 1996; Teunis et al., 1996; 2008a,b; U.S. EPA, 2006) and morbidity data from the literature. The dose-response relationship for *Salmonella* yields illness risks whereas all others yield infection risks. *Norovirus* was assumed to be non-aggregated and with a ratio of total to infectious virions consistent with that in the inoculum used for the dose-response parameterization (Teunis et al., 2008a). The morbidity data were used in conjunction with the output from the dose-response relationship to compute the probability of illness for each pathogen.

Statistical analysis and simulations were implemented in R and Mathematica® v. 5.2. The risk associated from each source was characterized as the total probability of GI illness, P^S_{ill} which was calculated using the probability of illness from each source-specific pathogen in a manner that is parallel to computing annual risks of infection by combining daily risks (Regli et al., 1991) $P^S_{ill} = 1 - \prod_{rp}(1 - P^S_{ill_{rp}})$.

Those source-specific results were then compared to each other and to a revised illness benchmark (0.03 per swim), as most of the pathogens investigated do not result in fever, which was previously included as a necessary component of GI illness (U.S. EPA, 1986).

3. Results

3.1. Literature review and summary of model parameters

A summary of the results from the literature review is provided below for 1) reference pathogen levels in cattle, pig, and chicken sources as well as for chlorinated secondary effluent, and 2) relative fraction of human-infectious strains of each of the reference pathogens in the non-human sources. A complete description of the comprehensive review is available under separate cover (U.S. EPA, in press). The data used for gulls were reported previously by Schoen and Ashbolt (2010). The studies identified during the review differed in the study size (as number of animals, number of farms), the degree to which potential human pathogen species were identified, and the duration of the study. The criteria used to select data and assign ranges for prevalence and abundance were as follows: data from studies conducted in the United States were preferred to studies conducted elsewhere (since the ultimate use of the study is for US waters), data from studies with large-scale and long duration were preferred, and data based on individual (not composite) samples were preferred.

Large-scale studies of *Salmonella* prevalence in pigs exhibited high year-to-year and herd-to-herd variability, with reported prevalence generally falling in the range 7.9–15% (Foley et al., 2008; Hutchinson et al., 2004). However, *Salmonella* prevalence among pigs appears to increase with age (Dorr et al., 2009). *Salmonella* infection in cattle differed between dairy and beef cattle, with cattle age, season and herd size (Callaway et al., 2005; Edrington et al., 2004; Huston et al., 2002; Kunze et al., 2008; Warnick et al., 2003; Wells et al., 2001). Large-scale studies of *Salmonella* infection in cattle (both dairy and beef) (Fossler et al., 2005; Hutchinson et al., 2004) indicate prevalence in the range 5–18%, with higher prevalence reported for some individual herds. Prevalence in chicken flocks (both layers and broilers) was found to be highly variable and dependent on age of the chickens (Byrd, 1998; Martin et al., 1998) and possibly on geographic region (Ebel et al., 1992; Garber et al., 2003). Based on the high variability of *Salmonella* observed in these studies, a prevalence range of 0–95% was selected as representative of *Salmonella* shedding among chickens. Few studies were found reporting *Salmonella* densities in fresh pig manure, because most pig wastes are stored as slurries. Among pig manure samples positive for *Salmonella*, two studies (Boes et al., 2005; Hutchinson et al., 2004) indicate a range of *Salmonella* faecal abundance from $10^{2.8}$–$10^{4.9}$ organisms g^{-1} faeces. *Salmonella* abundance in cattle faeces was reported to be in the range $10^{3.0}$–$10^{5.8}$ organisms g^{-1} faeces, with the lower end of the range set equal to the reported geometric mean of the densities, since minimum density was not reported (Hutchinson et al., 2004). Abundance of *salmonellae* in faeces of chickens appears to be independent of bird age and inoculation/ingestion dose (Byrd, 1998), with representative densities in the range 10^{-1}–$10^{4.5}$ organisms g^{-1} of fresh chicken excrement (Kraft et al., 1969).

Campylobacters are frequently found in pig slurry lagoons (McLaughlin et al., 2009) and pig faeces (Dorner et al., 2004; Weijtens et al., 1997), with prevalence generally increasing with age of the animal. Given the high prevalence observed and the trend toward increasing prevalence with animal age, the pig *Campylobacter* prevalence is estimated to be in the range 46–98%. Cattle *Campylobacter* prevalence differs between beef and dairy cattle, with feedlot cattle generally

exhibiting higher prevalence than cattle on pasture and with prevalence increasing with duration of cattle occupancy in feedlots (Besser et al., 2005). Considering the different prevalence among operations and between age cohorts, a representative range of prevalence for *Campylobacter* among all cattle is 5–38% (Hoar et al., 2001; Wesley et al., 2000). Chicken shedding prevalence for *Campylobacter* also tends to increase with age (Luangtongkum et al., 2006) and flocks frequently approach 100% infection rates (Cox et al., 2002). *Campylobacter* shedding is nearly universal among chicken houses and within-house rates are high and increase with bird age. A representative range of *Campylobacter* prevalence in chickens is 57–69% (Cox et al., 2002; El-Shibiny et al., 2005). Studies reporting *Campylobacter* abundance in pig faecal samples (Hutchison et al., 2005; Weijtens et al., 1999) suggest a representative density range of $10^{2.0}$–$10^{5.7}$ organisms g^{-1} faeces. Studies of cattle *Campylobacter* abundance (Hutchison et al., 2005; Inglis et al., 2004; Moriarty et al., 2008; Stanley et al., 1998) reported diverse results. The range of abundance selected for use here was the widest range reported in a single study ($10^{1.2}$–$10^{7.3}$ organisms g^{-1} faeces). Studies on *Campylobacter* abundance in chicken faeces (Bull et al., 2006; Cox et al., 2002; Hutchison et al., 2005; Whyte et al., 2001) were in general agreement, with a representative range of $10^{2.8}$–$10^{6.5}$ organisms g^{-1} faeces.

E. coli O157:H7 infection and shedding occurs frequently among cattle and pigs, and is highly uncommon in chickens (Doane et al., 2007). Several studies report relatively low infection rates among pigs (Chapman et al., 1997; Cornick and Helgerson, 2004; Feder et al., 2003; Hutchinson et al., 2004) with prevalence differing among types of operations and ages of animals, typically in the range 0.1–12%. Pig shedding of *E. coli* O157:H7 is highly variable and a representative range appears to be none detected – 10^7 organisms g^{-1} faeces (Cornick and Helgerson, 2004), with animals shedding more intensely during the early time period of infection and shedding at lower levels with chronic infection. Cattle *E. coli* O157:H7 prevalence and shedding are difficult to characterize, given wide differences among age cohorts and animals on different types of operations. *E. coli* O157 prevalence appears to differ between calves and adult cattle and between cattle before and after their arrival on feedlots. *E. coli* O157 infection peaks in young cattle between 3 and 18 months of age, and declines thereafter (Ellis-Iversen et al., 2009). In a large study of feedlot beef cattle, LeJeune et al. (2004) observed a general trend of increasing prevalence of *E. coli* O157:H7 among animals with their duration in the feedlot. Assessment of the available studies on *E. coli* O157:H7 in cattle (key studies were Berry et al., 2007; Hutchinson et al., 2004) led to estimates of prevalence and abundance ranges of 9.7–28% and $10^{3.1}$–$10^{8.4}$ organisms g^{-1}, respectively. The high end of the cattle *E. coli* O157:H7 abundance range is very high, but because it was taken from a large, systematic study that did not account for animal age or super shedding, it is considered part of the representative range of abundance in the general cattle population.

Estimates of ranges of prevalence and abundance of *Giardia* and *Cryptosporidium* in livestock and other wastes are based on a comprehensive review provided by Ferguson et al. (2009), supplemented with additional studies. *Cryptosporidium* shedding is sporadic among pigs and individual herd prevalence may be low (Heitman et al., 2002; Hutchison et al., 2005; Xiao et al., 2006), with a characteristic range of 0–45%. Like *E. coli* O157:H7, *Cryptosporidium* prevalence in cattle varies dramatically with age, with young cattle (<3 months) exhibiting much higher prevalence than older cattle (Wade et al., 2000) along with genotypes that are more infectious to humans (Chalmers and Giles, 2010). A representative range for *Cryptosporidium* prevalence, inclusive of all age groups, in cattle is estimated as 0.6–23%. *Cryptosporidium* shedding is observed among chickens, though the species detected are generally not those implicated in human infections. Ley et al. (1988) report *Cryptosporidium* prevalence among chickens between 6 and 27%. A representative range of *Cryptosporidium* shedding rates among pigs is $10^{1.7}$–$10^{3.6}$ oocysts g^{-1} (Hutchinson et al., 2004). Cattle shedding rates for *Cryptosporidium* vary widely for calves and adults, with a representative range of $10^{2.3}$–$10^{3.9}$ oocysts g^{-1} (Atwill et al., 2006). No studies were identified to allow an estimate of abundances of *Cryptosporidium* in chicken faeces, although Hutchinson et al. (2004) searched for *Cryptosporidium* in fresh chicken manure as part of a large-scale study of pathogens in livestock manures.

Estimates for the prevalence of *Giardia* in pig faeces are primarily drawn from Heitman et al. (2002), Xiao et al. (2006) and Hutchinson et al. (2004). Xiao et al. indicate the potential for dependence of prevalence on age, although that study was relatively small and conducted within a limited geographic region. The range of *Giardia* prevalence in pig manures is estimated to be 3.3–18%. In cattle, *Giardia* prevalence varies with animal age, with infection peaking when calves are relatively young and the probability of infection of an individual within its lifetime approaching 100% in some operations (Olson et al., 1997; Ralston et al., 2003; Wade et al., 2000). Two large-scale studies (Fayer et al., 2000; Wade et al., 2000) indicate a prevalence range for *Giardia* among cattle of 0.2–37%. Wide ranges of shedding intensities of *Giardia* among both pigs and cattle were observed, with pig faeces abundance in the range 10^0–$10^{6.8}$ cysts g^{-1} (data presented graphically, Maddox-Hyttel et al., 2006) and for cattle faeces in the range 10^0–$10^{4.9}$ cysts g^{-1} (Wade et al., 2000).

The Centers for Disease Control and Prevention (CDC, 2006) identified the serotypes from human *S. enterica* isolates for the period 1996–2006 and the United States Department of Agriculture Food Safety and Inspection Service (USDA FSIS, 2009) identified the serotypes for *Salmonella* isolates from broilers, market hogs, steer and heifers, and cows and bulls for the period 1998–2007. These data indicate that the prevalence of serotypes within a given host changes significantly from year-to-year, although for humans, the serotypes Typhimurium and Enteriditis were consistently among the top three serotypes isolated. The overlap between serotypes prevalent in humans and those present in livestock can be used to develop an estimate of the potential loading of human-infectious *Salmonella* from livestock. The 24 serotypes most commonly isolated from humans account for 79.5% of all positive isolates. The prevalence of the 24 most common human serotypes among livestock ranges from 52.5% to 59.8% of isolates. Because *S. enterica* infections are sporadic (Callaway et al., 2008) and

serotype prevalence may change dramatically from year to year (USDA FSIS, 2009), there exists the possibility for an animal-associated outbreak (among humans) for a relatively uncommon or an unknown serotype. Based on the overlap of livestock *Salmonella* serotypes with the serotypes most commonly implicated in human illness, the faecal pollution for chickens, cattle and swine were all assigned a qualitative level of "Medium" human-infectious potential.

The ability of *Campylobacter* isolates to infect humans also varies among species and isolates and the prevalence of strains differs in animals and humans. Although other species may play smaller roles in human health effects, *Campylobacter jejuni* and *Campylobacter coli* are the most important human-disease-causing species of *Campylobacter* commensal in livestock (Wesley et al., 2000). Ketley (1997) designated *C. jejuni* and *C. coli* as the species playing a major role in human infections (80%–90% of *Campylobacter* infections) and notes that other species have the potential for initiating human infections. For all livestock hosts, the prevalence of *Campylobacter* species or subtypes of species varies between farms and regions, with age of animal, with season, between isolates from rectal faecal samples and isolates from other environmental reservoirs (e.g., trough water) and probably with other factors (El-Shibiny et al., 2005; Hakkinen and Hänninen, 2009; Minihan et al., 2004; Weijtens et al., 1999; Wesley et al., 2000). *C. jejuni* and *C. coli* are prevalent among cattle, pigs and chickens, with chickens exhibiting higher incidence of *C. coli* shedding (as a percentage of all *Campylobacter* positive samples) than cattle and pigs (El-Shibiny et al., 2005). Based on these observations, cattle and swine *Campylobacter* were assessed as high infectious potential for humans and chicken *Campylobacter* are assessed to be of medium human-infectious potential.

Assessing the potential for cattle and other wildlife to generate virulent *E. coli* O157 is difficult given the apparent ability of Shiga-toxin-negative *E. coli* O157 to acquire *stx* virulence gene in a variety of hosts and settings (Wetzel and LeJeune, 2007), and the potential for differences in virulence between isolates from humans and other sources, though these differences were not observed in a recent study (Lenahan et al., 2009). Given the variability even among *E. coli* O157:H7 originating from the same source, we adopt a conservative approach and assume that *E. coli* O157:H7 from any source pose the same hazard to humans, and thus, is assigned a value of high human-infectious potential.

Among the more than 16 species of *Cryptosporidium* identified to date, *Cryptosporidium parvum* and *Cryptosporidium hominis* are believed to cause the majority of human infections among immunocompetent hosts. Other animals considered major hosts for *C. parvum* and *C. hominis* include cattle, sheep, goats, and monkeys (Xiao et al., 2004, 2006). Humans are considered minor hosts for other *Cryptosporidium* species, including *Cryptosporidium muris*, *Cryptosporidium meleagridis*, *Cryptosporidium felis*, and *Cryptosporidium canis*. Among livestock species, cattle prevalence of *Cryptosporidium* species aligns closely with species infecting humans, whereas swine *Cryptosporidium* are more seldom isolated in human infections and poultry *Cryptosporidium* appear not to overlap with species causing human infections (Xiao et al., 2006). Consequently, the human-infectious potential of cattle and swine *Cryptosporidium* is assessed as high (given the occurrence of human-infectious *Cryptosporidium* in swine, but not the occurrence of *Cryptosporidium suis* in humans), and the human-infectious potential of chickens is assessed as low.

The species of *Giardia* causing the majority of human illnesses are called *G. lamblia*, *Giardia duodenalis*, and *Giardia intestinalis* by different researchers (Adam, 2001; Thompson, 2004) as the taxonomy for *Giardia* remains unsettled. Thompson (2004) notes that *Giardia* isolates from humans fall into one of two major genotype assemblages and that some *Giardia* genotypic groupings are confined to specific animal hosts. Based on a listing of the most important *Giardia* species and genotypes and their associated hosts (Adam, 2001), cattle and pigs appear to have the potential for shedding *Giardia* that pose risks to humans, and chicken do not appear to be a significant source of human-infectious *Giardia* cysts. Thus, cattle and swine *Giardia* are assigned a high human-infectious potential and chicken *Giardia* are assessed as low human-infectious potential.

Estimating ranges of pathogen abundance in human faecal pollution is complicated by the episodic occurrence of pathogens in sewage, wide differences in removal of the pathogens for different wastewater treatment processes, and differences in disinfection doses and contact times. None of the bacterial pathogens (*E. coli* O157:H7, *Campylobacter, Salmonella* spp.) are reported to appear in significant densities in chlorinated secondary effluent (Garcia-Aljaro et al., 2005; Lemarchand and Lebaron, 2003; Stampi et al., 2003). Reported densities of *Cryptosporidium* in secondary effluent are relatively low, even in the absence of disinfection (Bonadonna et al., 2002; Bukhari et al., 1997; Castro-Hermida et al., 2008; Payment et al., 2001; Scott et al., 2003). A representative range of *Cryptosporidium* densities in secondary effluent accounting for episodes of natural variability in raw sewage and treatment process performance is $10^{-1.0}$–$10^{1.5}$ oocysts L^{-1} (Rose et al., 2004). *Giardia* densities in wastewater treatment (WWTP) plant effluent are, in general, somewhat higher than *Cryptosporidium* densities, although *Giardia* is also subject to episodic loading and variations in removal among treatment processes (Bukhari et al., 1997; Carraro et al., 2000; Castro-Hermida et al., 2008; Payment et al., 2001; Scott et al., 2003). Similar to the approach used for *Cryptosporidium*, the range of *Giardia* abundance in chlorinated secondary effluent was selected based on the widest reported range and estimated to be 10^{-1}–$10^{2.1}$ cysts L^{-1} (Rose et al., 2004) noting densities have not been corrected for method recovery. *Giardia* cysts in chlorinated secondary effluent are only slightly higher than those for *Cryptosporidium* because the inactivation of *Giardia* with chlorine (U.S. EPA, 2005) is greater than that for *Cryptosporidium*, despite higher densities in raw sewage and secondary undisinfected effluent. A wide range of *Norovirus* density in secondary effluent has been reported in the literature (Haramoto et al., 2006; Katayama et al., 2008; Laverick et al., 2004). Because relatively few publications describing *Norovirus* occurrence in WWTP were identified, the range of *Norovirus* abundance in chlorinated secondary effluent was estimated based on the reported raw sewage *Norovirus* densities in the range 10^3–10^6 genomic copies L^{-1} (Haramoto et al., 2006; Katayama et al., 2008) and an estimated range of removal in treatment of 2.2–3.0 logs (Haramoto et al., 2006).

Table 1 – Parameter values used in QMRA model.

Source	Primary sewage			Secondary chlorinated effluent			Gulls			Cattle[f]					Pigs				Chickens						
Density of:	Log_{10} range[a,b]		Ref	Log_{10} range		Ref	Log_{10} range		Ref	Log_{10} range		Basis[c]	Type[d]	Ref	Log_{10} range		Type	Ref	Log_{10} range		Basis	Type	Manure[e]	Ref
	a	b		a	b		a	b		a	b				a	b			a	b				
Enterococci in faecal waste	5.8	8.0	Lemarchand and Lebaron (2003); Metcalf and Eddy (2003)	0.5	2.7	Rose et al. (2004)	6.0	8.0	Fogarty et al. (2003); Haack et al. (2003)	2.0	5.1	D	C	Sinton et al. (2007)	5.3	7.2	D	Peu et al. (2006)	5	7	W	C	L	Brooks et al. (2009)
E. coli in faecal waste	6.7	8.0	Rose et al. (2004)	0.5	4.0	Rose et al. (2004)	5.0	9.0	Fogarty et al. (2003)	5.0	6.7	W	C	Berry et al. (2006)	6.1	7.3	C	Peu et al. (2006)	5.1[k]	10.9	W	C	L	Terzich et al. (2000)
E. coli O157:H7 in faecal waste	ND	3.3	Garcia-Aljaro et al. (2005)	ND[h]		Garcia-Aljaro et al. (2005)	NR[i]			3.1[j]	8.4	W	C	Hutchinson et al. (2004)	ND	7.0	D	Cornick and Helgerson (2004)	NR					Doane et al. (2007)
Campylobacter in faecal waste	ND	2.3	Lemarchand and Lebaron (2003) and Stampi et al. (2003)	ND		Stampi et al. (2003)	3.3	6.0	Lévesque et al. (2000)	1.2	7.3	W	D	Moriarty et al. (2008)	2.0	5.7	D	Weijtens et al. (1999)	2.8	6.5	W	D	F	Cox et al. (2002)
Salmonella in faecal waste	0.5	3.0	Lemarchand and Lebaron (2003)	ND		Lemarchand and Lebaron (2003)	2.3	9.0	Lévesque et al. (2000)	3.0[l]	5.8	W	C	Hutchinson et al. (2004)	2.8[j]	4.9	C	Hutchinson et al. (2004)	−1	4.5	D	D[m]	F	Kraft et al. (1969)
Cryptosporidium in faecal waste	−0.3	2.6	Rose et al. (2004)	−1	1.5	Rose et al. (2004)	NR		Schoen and Ashbolt (2010)	2.3	3.9	W	C	Atwill et al. (2006)	1.7[j]	3.6	C	Hutchinson et al. (2004)	ND					Hutchinson et al. (2004)

Giardia in faecal waste	0.8	4.0	Rose et al. (2004)	−1	2.1	Rose et al. (2004)	NR	Schoen and Ashbolt (2010)	0.0	4.9	W	D	Wade et al. (2000)	0	6.8[n]	D	Maddox-Hyttel et al. (2006)	NR
Norovirus in faecal waste[o]	3.0	6.0	Haramoto et al. (2006) and Katayama et al. (2008)	2.2–3.0 log removal[p]		Haramoto et al. (2006)	NA[g]		NA				NA	NA				NA

Note values shown are min and max of log-uniform distributions unless otherwise specified (e.g. 2.0 corresponds to a density of $10^{2.0}$).

a For primary sewage and secondary chlorinated effluent, units of minimum and maximum observations are $\log_{10}$ (cfu L^{-1} or oocysts L^{-1} or cysts L^{-1}); for livestock wastes units are $\log_{10}$(cfu g^{-1} or oocysts g^{-1} or cysts g^{-1}).

b "a" denotes the minimum observed value and "b" denotes the maximum observed value.

c Basis refers to weight basis for manure. D denotes dry weight and W denotes wet weight.

d Sample type is either composite (C) or direct (D).

e Chicken manure type is litter (L) or fresh (F).

f All cattle and pig faecal abundances reported are for solid, fresh faecal samples (not slurries or treated manure).

g Not applicable, generally thought not to be present in this source.

h Not detected.

i None reported, no data were found in the literature to quantify densities in this source.

j Geometric mean (minimum observed density not reported).

k Reported as mean per State. Range presented is range from State with lowest mean concentration to State with highest mean concentration.

l Low end of range of values "typically measured in cattle manure." Actual minimum not presented.

m Samples were taken at random from the top of the litter pile. Since the droppings were fresh, it is presumed they were derived from a single bird.

n Estimated from data presented graphically.

o Units of genomes L^{-1}.

p Removal range (rather than range of density). Attenuation in treatment (in log units) is assumed to be uniformly distributed.

Table 2 – Prevalence and relative occurrence of human-infectious species of pathogens.

Pathogen	Gulls			Cattle (beef & dairy)				Pigs				Chickens			
	Prevalence (%)[a]	Human infection potential[b]	Reference (s)	Minimum observed prevalence (%)	Max observed prevalence (%)	Human infection potential	Reference (s)	Minimum observed prevalence (%)	Max observed prevalence (%)	Human infection potential	Reference (s)	Minimum observed prevalence (%)	Max observed prevalence (%)	Human infection potential	References
E. coli O157:H7	None reported			9.7	28	H	Berry et al. (2007)	0.1	12	H	Hutchinson et al. (2004) and Cornick and Helgerson (2004)	0	0	NA	Chapman et al. (1997) and Doane et al. (2007)
Campylobacter spp.	100	L	Lévesque et al. (2000)	5	38	H	Hoar et al. (2001) and Wesley et al. (2000)	46	98	H	Dorner et al. (2004)	57	69	M	Cox et al. (2002) and El-Shibiny et al. (2005)
Salmonella enterica	100	L	Lévesque et al. (2000)	5	18	M	Hutchinson et al. (2004) and Fossler et al. (2005)	7.9	15	M	Hutchinson et al. (2004) and Dorr et al. (2009)	0	95	M	Byrd (1998) and Martin et al. (1998)
Cryptosporidium spp.	None reported			0.6	23	H	Atwill et al. (2006) and Sturdee et al. (2003)	0	45	L	Heitman et al. (2002) and Xiao et al. (2006)	6	27	L	Ley et al. (1988)
Giardia lamblia	None reported			0.2	37	H	Fayer et al. (2000) and Wade et al. (2000)	3.3	18	H	Heitman et al. (2002) and Xiao et al. (2006)	None reported			

Potential for human infection was based upon the prevalence of known human-infectious species/strains/serotypes/isolates in animal faeces.

NA – not applicable.

a For gulls, faecal prevalence and abundance data were based on observations from composite samples. All samples yielded campylobacters and salmonellae, so a conservative estimate of 100% prevalence was used.

b A qualitative approach was taken to characterize this parameter; low (L), medium (M), or high (H) assignments were made based on the results of a literature review, and point estimates were used to characterize the fractions of human-infectious strains based on the mid-point of the ranges of 0–33% for L, 33–66% for M, and 67–100% for H.

Table 3 – Dose-response models.

Reference Pathogen	Published dose-response model	Model parameters	ID50	Morbidity (% of infections resulting in illness)	Health Endpoint
Norovirus	Hypergeometric (Teunis et al., 2008a)	0.04,0.055	1018 genome copies	60%	Infection
Cryptosporidium spp.	Exponential (U.S. EPA, 2006)	0.09	8 oocysts	50%	Infection
Giardia lamblia	Exponential (Haas et al., 1999)	0.0199	35 cysts	45%	Infection
Campylobacter jejuni	Beta-Poisson (Medema et al., 1996; Teunis et al., 1996)	0.145,7.59	800 cfu	28%	Infection
E. coli O157:H7	Beta-Poisson (Teunis et al., 2008b)	0.4,45.9	207 cfu	28%	Infection
Salmonella enterica	Beta-Poisson (Haas et al., 1999)	0.3126,2884	23,600 cfu	20%	Illness

Based on the data obtained during the literature review and reported previously (Schoen and Ashbolt, 2010), the ranges used to characterize the densities of the indicators (EC and ENT) and reference pathogens in the faecal sources are provided in Table 1.

The prevalence of infection for the reference pathogens in each of the non-human sources and the relative proportion of human-infectious strains/types in the non-human sources are summarized in Table 2. The dose-response models that were used along with morbidity estimates are summarized in Table 3.

3.2. Scenarios

The predicted probabilities of GI illness under the most conservative assumptions (Run 1) for FIB densities of 35 cfu 100 mL^{-1} enterococci (Fig. 1A) and 126 cfu 100 mL^{-1} *E. coli* (Fig. 1B) demonstrate that disinfected municipal effluent and fresh cattle manure could present similarly high risk compared to the other faecal sources. The associated contributions of each pathogen to those risks are presented in Fig. 2A and B, noting that all pathogen strains are assumed to be human-infectious.

Inspection of Figs. 1 and 2 reveals several interesting observations. First, the results for primary effluent yield consistently lower risks than those for secondary disinfected effluent. These results stem from the approach that was used to normalize the faecal contamination across sources: sufficient contamination was assumed to be present so that the hypothetical waterbody contained the specified indicator densities. Although environmental waters impacted with primary effluent often have FIB levels higher than waters impacted by secondary disinfected effluent, if sufficient contamination were present to achieve the specified levels of FIB, the corresponding risks for primary effluent would be less than those for disinfected secondary effluent. This higher risk from more treated wastewater simply results from a higher

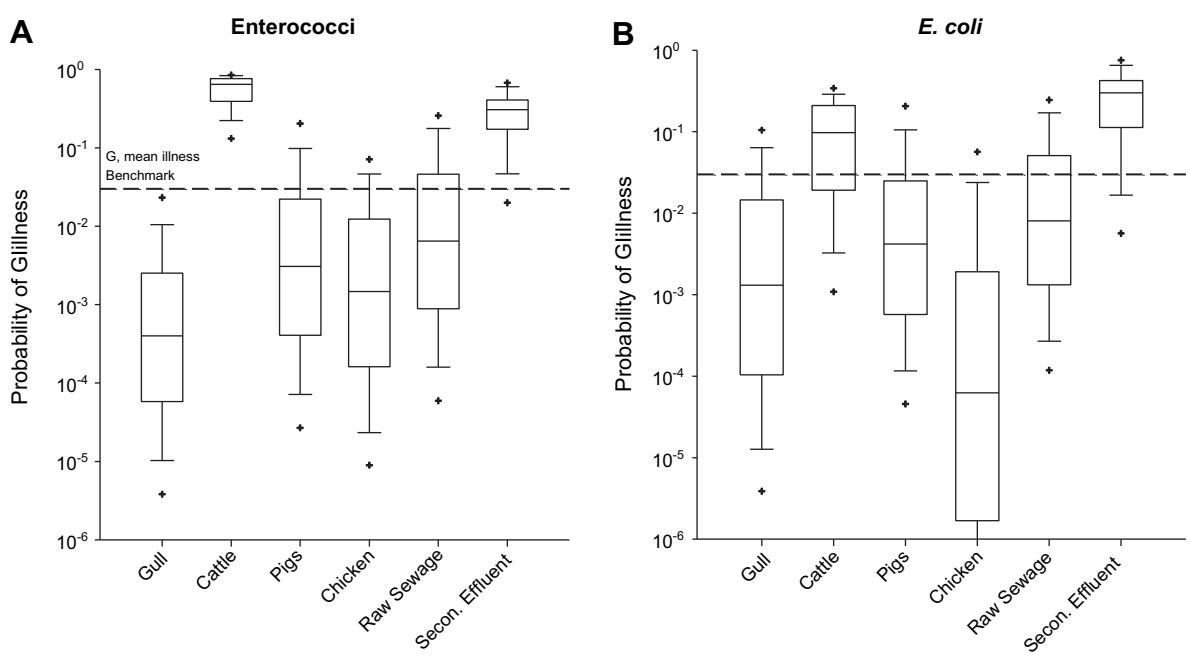

Fig. 1 – Run 1 probability of GI illness. Run 1 probability of GI illness from ingestion of water containing fresh faecal pollution at densities of 35 cfu 100 mL^{-1} ENT (1A) and 126 cfu 100 mL^{-1} *E. coli* (1B). Predicted risk (median, interquartile range, 10th and 90th percentiles, and 5th and 95th percentiles) for fresh gull, cattle and pig faeces, and chicken litter. Human impacts are presented for primary sewage (Human 1) and secondary disinfected effluent (Human 2). The illness benchmark represents a geometric mean probability of illness of 0.03.

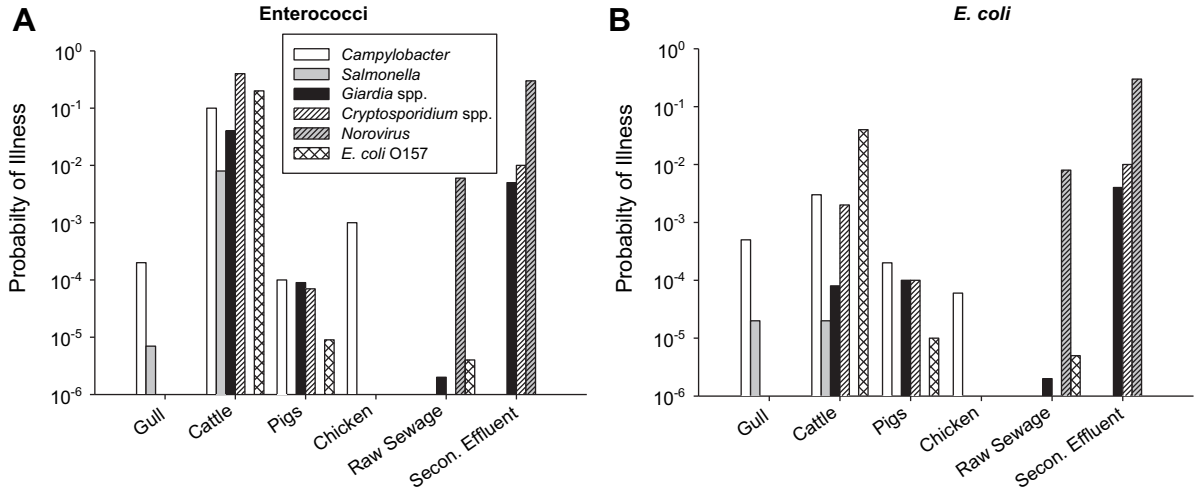

Fig. 2 – Run 1 contribution of each pathogen. Run 1 contribution of each pathogen to the probability of GI illness from ingestion of water containing fresh faecal pollution from animals or sewage at faecal indicator densities of 35 cfu 100 mL^{-1} ENT (2A) and 126 cfu 100 mL^{-1} *E. coli* (2B).

proportion of FIB being removed than viral and parasitic protozoan pathogens by wastewater treatment and disinfection (Metcalf and Eddy, 2003).

Second, the results for gull, pig, and human impacted waters are relatively consistent for enterococci and *E. coli*, whereas those for cattle and chickens are less consistent between the two indicators. The estimates for enterococci densities in cattle faeces differ from those for *E. coli* because the former were derived from dry weights whereas the rest of the cattle densities were based on wet weight. The study that provided the best estimates for enterococci densities in typical solid cattle manure (Sinton et al., 2007) reported enterococci density as organisms per dry mass of faeces. To use those data, the solids fraction of the manure was estimated using typical solids values for cattle manure (Lorimer et al., 2004). Conversion of enterococci densities in cattle manure from dry to wet weight is a potential source of uncertainty or bias and may explain some of the differences in the distributions of risk between FIB indicators for cattle-impacted waters. Consequently, the risk estimates for cattle based on *E. coli* may be more accurate.

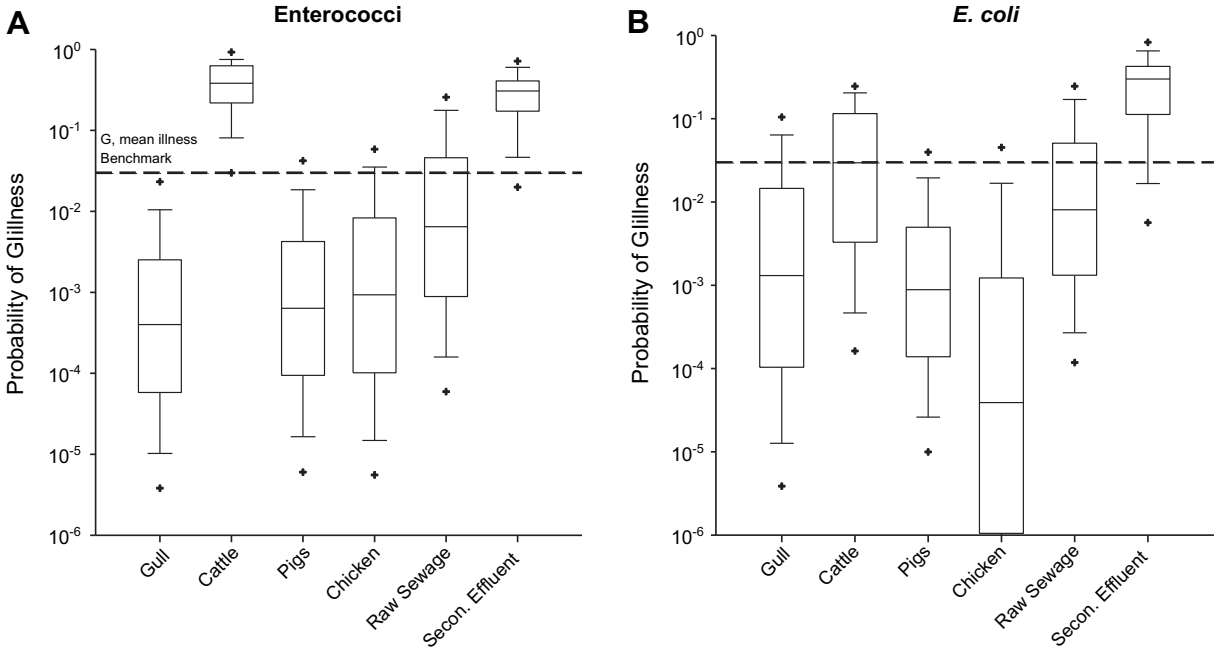

Fig. 3 – Run 2 probability of GI illness. Run 2 probability of GI illness from ingestion of water containing fresh faecal pollution at densities of 35 cfu 100 mL^{-1} ENT (3A) and 126 cfu 100 mL^{-1} *E. coli* (3B). Predicted risk (median, interquartile range, 10th and 90th percentiles, and 5th and 95th percentiles) for fresh gull, cattle and pig faeces, and chicken litter. Human impacts are presented for primary sewage (Human 1) and secondary disinfected effluent (Human 2). The illness benchmark represents a geometric mean probability of illness of 0.03.

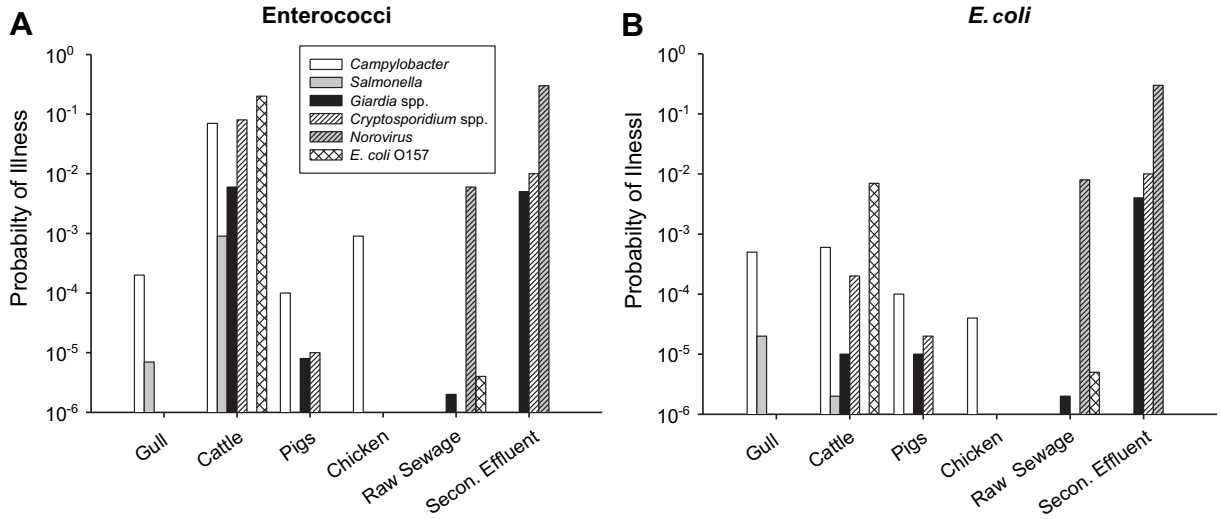

Fig. 4 – Run 2 contribution of each pathogen. Run 2 contribution of each pathogen to the probability of GI illness from ingestion of water containing fresh faecal pollution from animals or sewage at faecal indicator densities of 35 cfu 100 mL^{-1} ENT (4A) and 126 cfu 100 mL^{-1} *E. coli* (4B).

Third, for gull and chicken-impacted waters, *C. jejuni* emerged as the pathogen of primary concern. For cattle-impacted waters, the risks from *C. jejuni*, *Giardia* spp., *Cryptosporidium* spp. and *E. coli* O157:H7 were all of similar magnitude, whereas for pig-impacted waters, the risks from *C. jejuni*, *Giardia* and *Cryptosporidium* spp. were of similar magnitude. *Norovirus* dominated the GI illness risk for waters impacted by secondary disinfected municipal effluent (Soller et al., submitted for publication) and primary effluent (Schoen and Ashbolt, 2010).

The results from Run 2 were generally similar to Run 1 (Figs. 3 and 4). Most notably, however, was that the GI illness risks associated with cattle and pig-impacted waters were substantially lower (~1–2 log units) in Run 2 compared to Run 1. This observation is due to the fact that the prevalence of infection for the pathogens of concern in cattle and pig-impacted waters is substantially below 100% (Table 2). By contrast, the risks associated with chicken-impacted waters did not decrease as much because *C. jejuni* prevalence levels (the pathogen of concern in

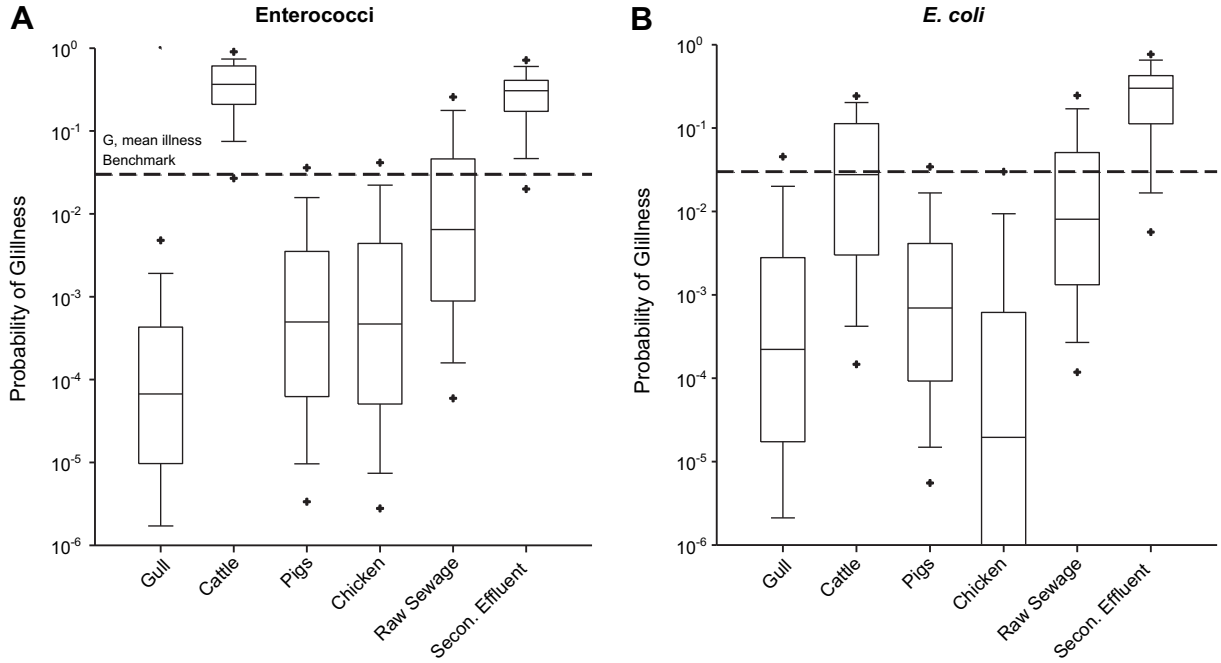

Fig. 5 – Run 3 probability of GI illness. Run 3 probability of GI illness from ingestion of water containing fresh faecal pollution at densities of 35 cfu 100 mL^{-1} ENT (5A) and 126 cfu 100 mL^{-1} *E. coli* (5B). Predicted risk (median, interquartile range, 10th and 90th percentiles, and 5th and 95th percentiles) for fresh gull, cattle and pig faeces, and chicken litter. Human impacts are presented for primary sewage (Human 1) and secondary disinfected effluent (Human 2). The Illness Benchmark represents a geometric mean probability of illness of 0.03.

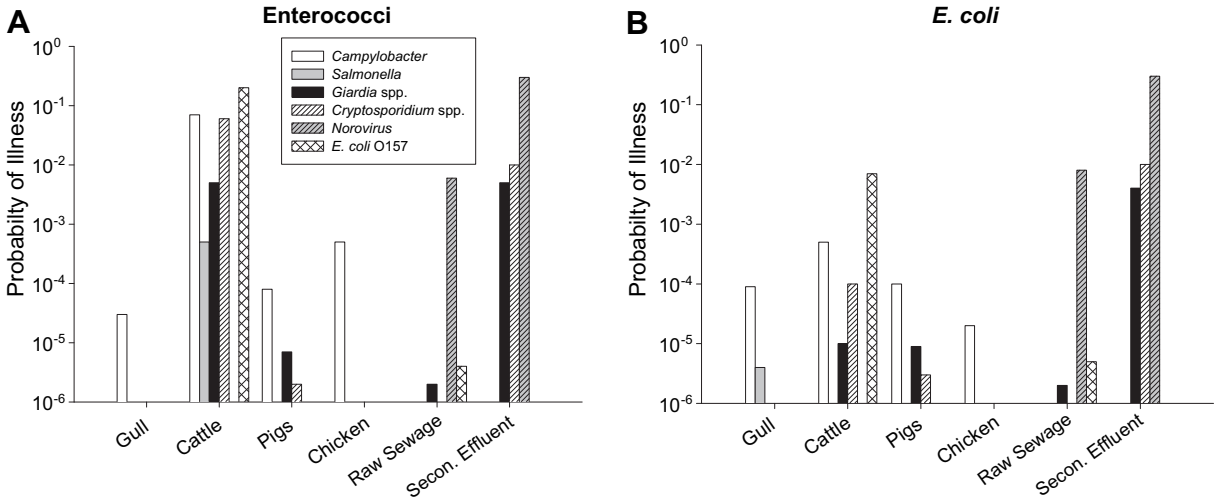

Fig. 6 – Run 3 contribution of each pathogen. Run 3 contribution of each reference pathogen to the probability of GI illness from ingestion of water containing fresh faecal pollution from animals or sewage at faecal indicator densities of 35 cfu 100 mL^{-1} ENT (6A) and 126 cfu 100 mL^{-1} *E. coli* (6B).

chicken-impacted waters) were relatively high by comparison (57–68%). Moreover, the illness risk associated with *E. coli* O157:H7 illness in cattle-impacted waters emerged in Run 2 as the dominant risk across FIB classes, and the illness risk associated with *C. jejuni* infection became the dominant pathogen risk in pig-impacted waters.

The Run 3 results (Figs. 5 and 6) revealed lower risks than predicted in Run 1 for gulls, cattle, pigs, and chickens. Gull and chicken risk reductions are due to the lower occurrence of known human-infectious strains of *Campylobacter* spp. compared to cattle and pig manures (Table 2). Cattle and pig risk reductions in Run 3 seem to be due to a combination of the prevalence of infection in these sources and the proportion of human-infectious pathogenic strains from each source.

4. Discussion

The probability of GI illness was estimated for exposure to recreational water with likely human and non-human fresh faecal sources. We evaluated three scenarios which were intended to bracket the uncertainty surrounding the prevalence of infection in each of the sources and the proportion of human-infectious pathogenic strains from each source (Runs 1–3). Based on our detailed review of the literature, we believe that results from Runs 2 and 3 represent credible interpretations of the available data. In the three scenarios, the numerical method relied on an implicit assumption that sufficient fresh faecal contamination was present in a hypothetical waterbody to achieve the specified levels of FIB. In reality, environmental waters will contain FIB from other sources. The extent to which those FIB levels could impact this assessment was not investigated.

For human sources of contamination it is likely that the illnesses measured in the epidemiologic studies that supported the 1986 AWQC and the 2003/2004 recreational water epidemiology studies conducted on the Great Lakes (Cabelli et al., 1982; Dufour, 1984; Wade et al., 2006, 2008) resulted from a combination of both well-treated, disinfected wastewater and less well-treated or untreated sewage contamination (directly from swimmers, poorly operating septic systems, sewage bypassing treatment etc.). The two sets of results presented here for human contamination, bracket possible conditions of human contamination and taken together represent an average risk that is consistent with the findings from the epidemiologic studies in the US (Cabelli et al., 1982; Dufour, 1984; Wade et al., 2006, 2008).

Our analysis indicates that the GI illness risks associated with human exposure to recreational waters impacted by fresh cattle manure may not be substantially different from those impacted by human sources: the distributions of risk effectively span the same range. This finding is in part due to the unknown proportion of human-infectious species/strains in cattle manure-impacted waters. In the absence of effective management practices that would significantly reduce these risks or new knowledge on infectivity, less stringent or alternative water quality standards for cattle-impacted waters do not seem appropriate at this time. Moreover, a suite of pathogens appears to be present in cattle-impacted waters (*C. jejuni*, *Cryptosporidium* and *Giardia* spp., in addition to *E. coli* O157:H7), any one of which may be present at a level that could be of concern. Within this context, *E. coli* O157:H7 and similar Shiga-toxin-producing strains are of particular concern because several are known to cause adverse health outcomes that are substantially more serious than self limiting GI illness (Bettelheim, 2007). Furthermore, this situation is more complex than presented here, as some strains of *E. coli* O157:H7 are not human pathogens (Bettelheim, 2007) and adult cattle largely excrete oocysts (of *Cryptosporidium brevis* and *Cryptosporidium andersoni*) that are much less likely to be human pathogens than from calves (excreting *C. parvum*) (Chalmers and Giles, 2010).

In contrast, the water-related risks associated with gull, chicken, and pig faeces are estimated to be substantially lower than those impacted from human faecal sources at the indicator densities assessed; median risks from these sources are at least two orders of magnitude lower than the human-based benchmark. Based on these results, the potential for developing alternative water quality standards (or guidelines) for gull, chicken, and pig-impacted waters should not be ruled out. One caveat however, is the emerging risk from pig hepatitis E virus genogroup C in human disease (Rutjes et al., 2009).

There are a number of important considerations to the work presented here. First, the analysis relied on a review of the readily available scientific literature. Additional data may refine the relative risk estimates presented here. Second, super shedding exposure scenarios were not considered in this analysis (Arthur et al., 2009; Chase-Topping et al., 2008). Risks to human health would increase if super shedding cattle (or calves) were present due to the increased levels of pathogens in faeces (Bryan et al., 2009; Chase-Topping et al., 2008).

Third, chicken pathogen data are based on fresh faeces, whereas the FIB data are from chicken litter. This causes an additional level of uncertainty which could result in over or underestimated levels of risk due to potential differential die-off of indicator bacteria as compared to pathogens. Fourth, the analyses presented here are based on the assumption that the contamination is recent and from relatively fresh faeces. As the contamination becomes less fresh, both FIB and pathogens will decay, however they may not decay at the same rate (Anderson et al., 2005). Thus, differential persistence over time could yield results that differ from those summarized here because many pathogens are more persistent than FIB while others are less persistent. These effects could be particularly important for chicken litter and pig faecal slurries, both of which are subject to widely variable storage times and handling practices. Our future work includes investigating the impacts of differential persistence on recreational water risks. Fifth, the analyses for pig-impacted waters were based on FIB and pathogens in pig manure. However, pig manure is commonly land-applied as slurry, yet, the literature review indicated that sufficient data were not available to conduct this analysis directly for pig manure slurry. The potential impact of differential persistence of FIB and pathogens in pig slurry relative to pig manure was not identified.

Finally, the occurrence of pathogens in recreational waters is a function of both spatial and temporal variability. Thus, the actual risks to human health present in any specific location at a particular time could vary substantially from the estimates presented here. This finding is particularly relevant to cattle, for which there are known and significant seasonal variations in shedding rates for all of the representative pathogens, as well as in rain-induced run-off that may drive manure-related pathogens into waterbodies.

5. Conclusions

The analysis presented here is an initial step toward understanding whether or not the relative risks from exposure to recreational waters impacted by gulls, chickens, pigs, and/or cattle are substantially different from those associated with human (sewage)-impacted waters. The QMRA results are consistent with the findings from epidemiology studies. In particular, illness risk associated with non-sewage impacted beaches appears to depend on the source of contamination, i.e. some animals show relatively lower risks than others, which could account for the conflicting epidemiology findings (Till et al., 2008).

The principal findings from this work are that the GI illness risks associated with exposure to recreational waters impacted by fresh cattle faeces may not be substantially different from those impacted by human sources, whereas the risks associated with exposure to recreational waters impacted by gull, chicken, and pig wastes appear to be substantially lower than those impacted by human sources. There are a number of important limitations to the work presented here. Nevertheless, these results suggest that careful consideration may be needed in the future for the management of recreational waters not impacted by human sources.

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Weather History for KSBA - October, 2012

Wednesday, October 3, 2012

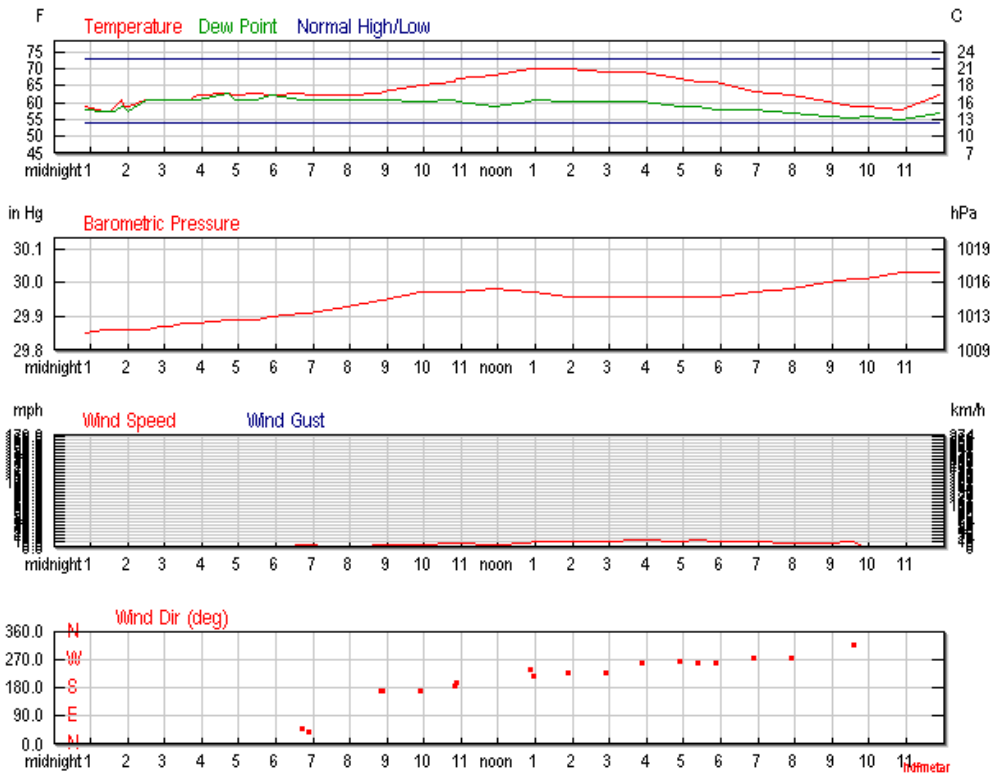
Daily | Weekly | Monthly | Custom

	Actual	Average	Record

T = Trace of Precipitation, MM = Missing Value

Source: NWS Daily Summary

Daily Weather History Graph



Search for Another Location

Airport or City:

KSBA

Submit

Trip Planner

Search our weather history database for the weather conditions in past years. The results will help you decide how hot, cold, wet, or windy it might be!

Date:

Astronomy

Oct. 03, 2012	Rise	Set
Actual Time	6:55 AM PDT	6:39 PM PDT

Oct. 03, 2012	Rise	Set
Civil Twilight	6:30 AM PDT	7:05 PM PDT
Nautical Twilight	6:01 AM PDT	7:34 PM PDT
Astronomical Twilight	5:32 AM PDT	8:03 PM PDT
Moon	8:46 PM PDT (10/3)	10:13 AM PDT (10/3)
Length of Visible Light	12h 34m	
Length of Day	11h 44m	

Waning Gibbous, 88% of the Moon is Illuminated

Oct 3	Oct 8	Oct 15	Oct 21	Oct 29
Waning Gibbous	Last Quarter	New	First Quarter	Full

Hourly Weather History & Observations

Time (PDT)	Temp.	Dew Point	Humidity	Pressure	Visibility	Wind Dir	Wind Speed	Gust Speed	Precip	Events	Conditions
12:53 AM	**59.0** °F	**57.9** °F	96%	**29.85** in	**8.0** mi	Calm	Calm	-	N/A		Clear
1:22 AM	**57.2** °F	**57.2** °F	100%	**29.86** in	**7.0** mi	Calm	Calm	-	N/A		Scattered Clouds
1:31 AM	**57.2** °F	**57.2** °F	100%	**29.86** in	**8.0** mi	Calm	Calm	-	N/A		Mostly Cloudy
1:40 AM	**59.0** °F	**57.2** °F	94%	**29.86** in	**1.8** mi	Calm	Calm	-	N/A		Overcast
1:51 AM	**60.8** °F	**59.0** °F	94%	**29.86** in	**0.5** mi	Calm	Calm	-	N/A	Fog	Fog
1:53 AM	**59.0** °F	**59.0** °F	100%	**29.86** in	**0.5** mi	Calm	Calm	-	N/A	Fog	Fog
2:00 AM	**59.0** °F	**57.2** °F	94%	**29.86** in	**0.2** mi	Calm	Calm	-	N/A	Fog	Fog
2:30 AM	**60.8** °F	**60.8** °F	100%	**29.86** in	**1.2** mi	Calm	Calm	-	N/A		Overcast
2:33 AM	**60.8** °F	**60.8** °F	100%	**29.86** in	**3.0** mi	Calm	Calm	-	N/A		Overcast
2:53 AM	**61.0** °F	**61.0** °F	100%	**29.87** in	**2.0** mi	Calm	Calm	-	N/A		Overcast
3:06 AM	**60.8** °F	**60.8** °F	100%	**29.87** in	**0.8** mi	Calm	Calm	-	N/A		Overcast
3:30 AM	**60.8** °F	**60.8** °F	100%	**29.88** in	**1.0** mi	Calm	Calm	-	N/A		Mist
3:42 AM	**60.8** °F	**60.8** °F	100%	**29.88** in	**0.8** mi	Calm	Calm	-	N/A		Mist

Time (PDT)	Temp.	Dew Point	Humidity	Pressure	Visibility	Wind Dir	Wind Speed	Gust Speed	Precip	Events	Conditions
3:53 AM	62.1 °F	61.0 °F	96%	29.88 in	0.8 mi	Calm	Calm	-	N/A		Mist
4:44 AM	62.6 °F	62.6 °F	100%	29.89 in	1.0 mi	Calm	Calm	-	N/A		Mist
4:53 AM	62.1 °F	61.0 °F	96%	29.89 in	1.2 mi	Calm	Calm	-	N/A		Overcast
5:29 AM	62.6 °F	60.8 °F	94%	29.89 in	0.8 mi	Calm	Calm	-	N/A		Overcast
5:53 AM	62.1 °F	62.1 °F	100%	29.90 in	0.8 mi	Calm	Calm	-	N/A		Overcast
6:43 AM	62.6 °F	60.8 °F	94%	29.91 in	2.5 mi	NE	4.6 mph	-	0.01 in		Overcast
6:53 AM	62.1 °F	61.0 °F	96%	29.91 in	3.0 mi	NE	3.5 mph	-	0.01 in		Overcast
7:53 AM	62.1 °F	61.0 °F	96%	29.93 in	2.5 mi	Calm	Calm	-	N/A		Overcast
8:51 AM	62.6 °F	60.8 °F	94%	29.95 in	5.0 mi	South	3.5 mph	-	N/A		Overcast
8:53 AM	63.0 °F	61.0 °F	93%	29.95 in	5.0 mi	South	3.5 mph	-	N/A		Overcast
9:53 AM	64.9 °F	60.1 °F	84%	29.97 in	6.0 mi	South	3.5 mph	-	N/A		Haze
10:50 AM	66.2 °F	60.8 °F	83%	29.97 in	6.0 mi	South	6.9 mph	-	N/A		Haze
10:53 AM	66.9 °F	60.1 °F	79%	29.97 in	6.0 mi	SSW	5.8 mph	-	N/A		Haze
11:53 AM	68.0 °F	59.0 °F	73%	29.98 in	6.0 mi	Variable	3.5 mph	-	N/A		Haze
12:53 PM	70.0 °F	60.1 °F	71%	29.97 in	8.0 mi	WSW	6.9 mph	-	N/A		Haze
12:56 PM	69.8 °F	60.8 °F	73%	29.97 in	8.0 mi	SW	9.2 mph	-	N/A		Clear
1:53 PM	70.0 °F	60.1 °F	71%	29.96 in	10.0 mi	SW	9.2 mph	-	N/A		Clear
2:53 PM	69.1 °F	60.1 °F	73%	29.96 in	10.0 mi	SW	8.1 mph	-	N/A		Clear
3:53 PM	69.1 °F	60.1 °F	73%	29.96 in	10.0 mi	West	10.4 mph	-	N/A		Clear
4:53 PM	66.9 °F	59.0 °F	76%	29.96 in	10.0 mi	West	8.1 mph	-	N/A		Clear
5:24 PM	66.2 °F	59.0 °F	78%	29.96 in	10.0 mi	West	10.4 mph	-	N/A		Clear
5:53 PM	66.0 °F	57.9 °F	75%	29.96 in	10.0 mi	West	8.1 mph	-	N/A		Clear

Time (PDT)	Temp.	Dew Point	Humidity	Pressure	Visibility	Wind Dir	Wind Speed	Gust Speed	Precip	Events	Conditions
6:53 PM	**63.0** °F	**57.9** °F	84%	**29.97** in	**10.0** mi	West	**8.1** mph	-	N/A		Clear
7:53 PM	**62.1** °F	**57.0** °F	84%	**29.98** in	**10.0** mi	West	**5.8** mph	-	N/A		Clear
8:53 PM	**60.1** °F	**55.9** °F	86%	**30.00** in	**10.0** mi	Variable	**5.8** mph	-	N/A		Clear
9:34 PM	**59.0** °F	**55.4** °F	88%	**30.01** in	**10.0** mi	NW	**9.2** mph	**161.1** mph	N/A		Clear
9:53 PM	**59.0** °F	**55.9** °F	90%	**30.01** in	**10.0** mi	Calm	Calm	-	N/A		Clear
10:53 PM	**57.9** °F	**55.0** °F	90%	**30.03** in	**10.0** mi	Calm	Calm	-	N/A		Mostly Cloudy
11:53 PM	**62.1** °F	**57.0** °F	84%	**30.03** in	**10.0** mi	Calm	Calm	-	N/A		Overcast

| |

Dilution Calculations for Fecal coliform (MPN/100mL)

Secondary treated sewage estimate	
1,000,000	MPN

Carpinteria loss disinfection information

281,250 gallons

Approved shellfish standard	
14	MPN

Step 1

Calculate load

Effluent Concentration x Flow = Load

Flow	281,250	gallons
convert flow to ft^3	37,600.27	ft^3
Load	3.76E+10	FC/7.5 hours

Step 2

Calculate volume needed to dilute to 14 MPN

Effluent Load/Target Concentration =Volume for Dilution

Volume	2.69E+09	ft^3

Step 3

Calculate surface area

dilution volume/ depth = surface area

Depth approx 25 feet

Surface Area	1.07E+08	ft^2

Step 4

Calculate radius of impact

Square root ((2 x Area)/ Pi)= Radius

Radius	8,270	ft	
Convert to miles	1.57	miles	Zone of Impact

Disinfection Requirements for
Region 3 facilites with Ocean Outfalls

Facility Name	NPDES Permit #	Order No.	Facility Information	Discharge Location
Monterey Regional Water Pollution Control Agency WWTP (Major)	CA0048551	R3-2014-0013	29.6 MGD; undisinfected secondary effluent & brine to ocean; tertiary treated effluent to land.	Depth To Water (DTW): 100 ft.; 11,260 ft. outfall/diffuser; seawater to efflution dilution rato (dilution) 145:1.
City of Watsonville WWTP (Major)	CA0048216	R3-2014-0006	12 MGD (dry)/36 MGD (wet) undisinfected secondary, brine, & landfill leachate to ocean; tertiary treated effluent to land.	DTW: 64 ft.; 7,350 ft. outfall/diffuser; dilution 84:1.
Morro Bay/Cayucos WWTP (Major)	CA0047881	R3-2008-0065	2.06 MGD (dry); up to 1 MGD secondary treated effluent blended with primary treated effluent prior to disinfection & discharge to ocean.	DTW: 50 ft.; 4,400 ft. outfall/diffuser; dilution 133:1.
Ragged Point Inn (Minor)	CA0049417	R3-2009-0020	0.015 MGD Disinfected treated secondary effluent to ocean; tertiary treated effluent to land	Discharge to ocean is via the cliff face.
South San Luis Obispo SD WWTP (Major)	CA0048003	R3-2009-0046	5 MGD; disinfectd secondary effluent & brine to ocean.	DTW: 55 ft.; 4,400 ft. outfall/diffuser shared City of Pismo; dilution 165:1.
Pismo Beach WWTP, City of (Major)	CA0048151	R3-2009-0047	up to 1.9 MGD disinfected secondary effluent to ocean.	DTW: 55 ft.; 4,400 ft. outfall/difuser shared with So. San Luis; dilution 165:1.
Avila Beach CSD WWTP (Minor)	CA0047830	R3-2009-0055	up to 0.2 MGD; equal to secondary treated effluent is disinfected then discharged to ocean.	DTW: 34.5 ft.; 2,700 ft. outfall/diffuser; dilution 151:1
Goleta SD WWTP (Major)	CA0048160	R3-2010-0012	9 MGD (dry)/9.7 MGD (peak dry flow); flow > 4.38MGD primary treatment only & blended with treated secondary prior to disinfection & discharge to ocean.	DTW: 87 ft.; 5,912 ft. outfall/diffuser; dilution 122:1.
Santa Barbara City PWD, El Estero WWTP (Major)	CA0048143	R3-2010-0011	11 MGD disinfected secondary effluent & 12.5 MGD desal brine to ocean.	DTW 70 ft.; 8,700 ft. outfall/diffuser; dilution 120:1.
Santa Cruz DPW WWTP (Major)	CA0048194	R3-2010-0043	17 MGD (dry)/81 MGD (wet); dry weather flows from Neary Lagoon, septage, etc. and secondary treated effluent is disinfected prior to dicsharge to the ocean.	DTW: 110 ft.; 12,500 ft. outfall/diffuser shared with City of Scotts Valley; dilution 114:1.
Carpinteria SD WWTP (Major)	CA0047364	R3-2011-0003	2.5 MGD; disinfected secondary effluent to ocean.	DTW: 25 ft.; 1,000 ft. outfall/diffuser; dilution 93:1.
Montecito SD WWTP (Major)	CA0047899	R3-2012-0016	1.5 MGD; disinfected secondary treatment to ocean.	DTW: 35 ft.; 1,500 ft. outfall/diffuser; dilution 89:1.
City of Scotts Valley WWTP (Major)	CA0048828	R3-2013-0001	1.5 MGD; disinfected secondary treatment to ocean.	DTW: 110 ft.; 12,500 ft. outfall/diffuser shared with City of Santa Cruz; dilution 114:1.
San Simeon WWTP (Minor)	CA0047961	R3-2013-0021	0.2 MGD disinfected secondary effluent to ocean; tertiarty treated effluent to land.	DTW: 20; 900 ft. outfall/diffuser; dilution 115:1
Summerland SD WWTP (Minor)	CA0048054	R3-2013-0042	0.3 MGD; stormwater & disinfected secondary effluent to ocean.	DTW: 20 ft.; 740 ft. outfall/diffuser; dilution 60:1.
Carmel Area Wastewater District WWTP (Major)	CA0047996	R3-2014-0012	3 MGD; disinfected secondary & brine to ocean in winter; tertiary treated effluent to land in summer.	DTW: 35 ft.; 650 ft. outfall/diffuser; dilution 121:1.
	Facilities are not required to disinfect before discharge to the ocean.			