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Simulated gastrointestinal risk from recreational exposure to Southern California stormwater and relationship to human-associated Bacteroidales marker HF183

Sarah A. Lowry[1], Joshua A. Steele[2], John F. Griffith[2], Kenneth C. Schiff[2], and Alexandria B. Boehm[1*]

[1] Department of Civil and Environmental Engineering, Stanford University, 473 Via Ortega, Stanford, CA 94305, USA
[2] Southern California Coastal Water Research Project, Costa Mesa, CA, 92626, USA

* Corresponding author: aboehm@stanford.edu

Environmental Significance Statement

The health of recreational swimmers may be at risk if they are exposed to stormwater containing pathogens. Health and water management authorities in Southern California can measure HF183, a human fecal indicator, but lack the context to translate HF183 concentrations to risk levels that can inform decision-making. This study uses quantitative microbial risk assessment to estimate the risk of stormwater in Southern California during wet weather using measured pathogen and HF183 concentrations, and derives a risk-based threshold for HF183. This model can be generalized to estimate risk and risk-based thresholds given inputs of pathogens and relevant indicators for a variety of geographical contexts.

Abstract

Stormwater may contain pathogens that pose a health risk to recreators. In this study, we use quantitative microbial risk assessment (QMRA) to simulate the human health risk associated with recreational exposure to stormwater using a regional dataset of pathogen concentrations measured over two wet seasons during wet weather events in Southern California, USA, a location where stormwater and sewage systems are separate. We model risk using a Monte Carlo simulation using *Salmonella*, *Campylobacter*, adenovirus, and norovirus concentrations in stormwater, the volume of water ingested during a recreational swimming event, and pathogen-specific dose-response functions. We estimated the median probability of illness from recreational exposure to stormwater to be approximately 190 illnesses per 1,000 swimmers (19%). However, stormwater sampling sites are not always designated for recreational use, so we simulated exposures to diluted stormwater, which may be encountered in downstream receiving waters designated for swimming. We determined that if stormwater is diluted 18% into receiving, pathogen-free, ambient waters, the median health risk meets the US EPA's threshold of 32 illnesses per 1,000 swimmers. At this dilution, the concentration of HF183, a human-associated fecal marker, is expected to be 100 copies per 100 milliliters. This study provides a risk-based threshold for HF183 concentrations in stormwater-impacted ambient waters from pathogen and indicator concentrations measured in stormwater. Implementing this risk-based threshold will require many policy considerations.

Introduction

In the United States, recreating in pathogen-contaminated surface waters incurs significant health and economic burdens every year, with an estimated 90 million illnesses resulting in costs (medication use, healthcare provider visits, emergency department visits, hospitalizations, lost productivity, long-term sequelae, and mortality) between 2.2 and 3.7 billion 2007 USD annually (1). Human pathogens enter recreational water through fecal pollution and can pose a risk to human health (2). Recreational water managers and health authorities typically rely on fecal indicator bacteria (FIB) to detect fecal pollution and determine the microbiological safety of water for recreators. FIB include enterococci and *Escherichia coli* bacteria which are usually not pathogenic and are found at high concentrations in feces (3). FIB are used for beach management since epidemiology studies show quantitative relationships between FIB concentrations in recreational waters and risk of gastrointestinal illness (4–8). These epidemiological studies were conducted in ambient waters impacted by discharges of treated wastewater.

FIB concentrations are usually high in stormwater from urban environments. For example, median enterococci concentrations of 5,000 CFU/100 mL (and as high as 80,000 CFU/100 mL) have been documented in stormwater and storm-influenced water around the world (9); these concentrations are one to two magnitudes of order lower than those typically observed in raw sewage (10,11). FIB can enter stormwater sewer systems that are separate from sanitary sewer systems through a variety of sources (e.g., septic leaks, urban runoff, agricultural runoff) (12), as well as leaking sewage lines (13). Because FIB can have many sources, including non-human (14) and non-fecal sources (15), there is concern that their high levels do not always indicate the presence of human pathogens, and therefore may not indicate a health risk (16,17). Only one epidemiology study to date has estimated incident illness rates for recreators in stormwater impacted recreational waters (18) and they found that illness risk was higher for exposures during wet weather when stormwater was present in the waterbody, and that risk correlated positively to FIB concentrations during wet weather. However, there is a need to better understand the potential risks associated with recreation stormwater exposures.

The goal of this study is to use quantitative microbial risk assessment (QMRA) to estimate the risk of illness from exposure to stormwater. In the QMRA framework, measurements of pathogens in the environment can be used to estimate the health risk to users interacting with the environment. To date, QMRA has been extensively used to estimate risk from exposure to recreational waters (19). However, relatively few QMRA models have estimated risk from exposure to pathogens measured in stormwater in wet weather (20–24).

Because recreational exposure to FIB in stormwater from human feces likely represents a greater risk than exposure to equivalent concentrations of FIB from other fecal and non-point sources (25), efforts to identify human fecal pollution specifically may provide better insights into risk associated with exposure to stormwater. The *Bacteroides* HF183 marker is a DNA sequence present in the genome of a human gut-associated microorganism (26,27). It is sensitive and specific to human feces based on studies completed using samples from throughout the world (28–30), and has been detected in separate stormwater systems that are

not connected to sanitary sewers (31–34). Measuring HF183 instead of traditional FIB may offer advantages for assessing health risks.

By measuring HF183 in parallel with pathogens in stormwater, we can use QMRA to connect human health risk to HF183 concentrations. This can allow the development of a risk-based threshold for HF183 concentrations. Risk-based thresholds have previously been developed for recreational waters contaminated with raw sewage or gull feces (35–40), but none have been developed for recreational waters impacted by stormwater. This study is the first to use pathogen and indicator measurements measured in stormwater to estimate risk to recreators and then develop a risk-based threshold for stormwater-impacted water.

This study estimates the risk from recreating in stormwater and derives a risk-based threshold for HF183 for stormwater-impacted recreational waters using a dataset of pathogen and HF183 concentrations quantified in stormwater from storm events in two wet seasons across Southern California (12). The risk-based threshold developed in this study can aid in the interpretation of HF183 concentrations across Southern California in wet weather, and the model framework employed here can be applied to locations worldwide.

Methods

This study follows the QMRA framework, which consists of five major components (41). In hazard identification, the pathogens important to the modeled scenario are identified. In exposure assessment, concentrations of the identified pathogens in the environment of interest are quantified, and the exposure is described. Dose-response assessment involves identifying equations that describe the mathematical relationship between the dose of pathogen and the probability of infection or illness in humans. Risk characterization consists of modeling the probability of illness using the pathogen concentrations and exposure route alongside the dose-response equations, and risk management involves the application and interpretation of results.

Hazard Identification

Pathogen concentrations were measured in municipal stormwater runoff samples for direct use in the QMRA model. Seventy (70) two-liter stormwater samples were collected from stormwater outfalls, flood control channels, and stormwater-dominated streams and rivers during wet weather events over two wet seasons (October 2021–March 2022; November 2022–March 2023). Rainfall totals for wet weather events ranged from 0.10 to 17.2 cm (12). Stormwater samples were collected from 31 sites across Southern California (including Los Angeles, Orange, Riverside, San Bernardino, San Diego, and Ventura counties), where each site was sampled during two or three wet weather events (Figure 1). Many of the locations downstream of the sites are designated for contact recreation in their respective Basin Plans (12). Detailed information about sample collection is described by Steele et al. (12).

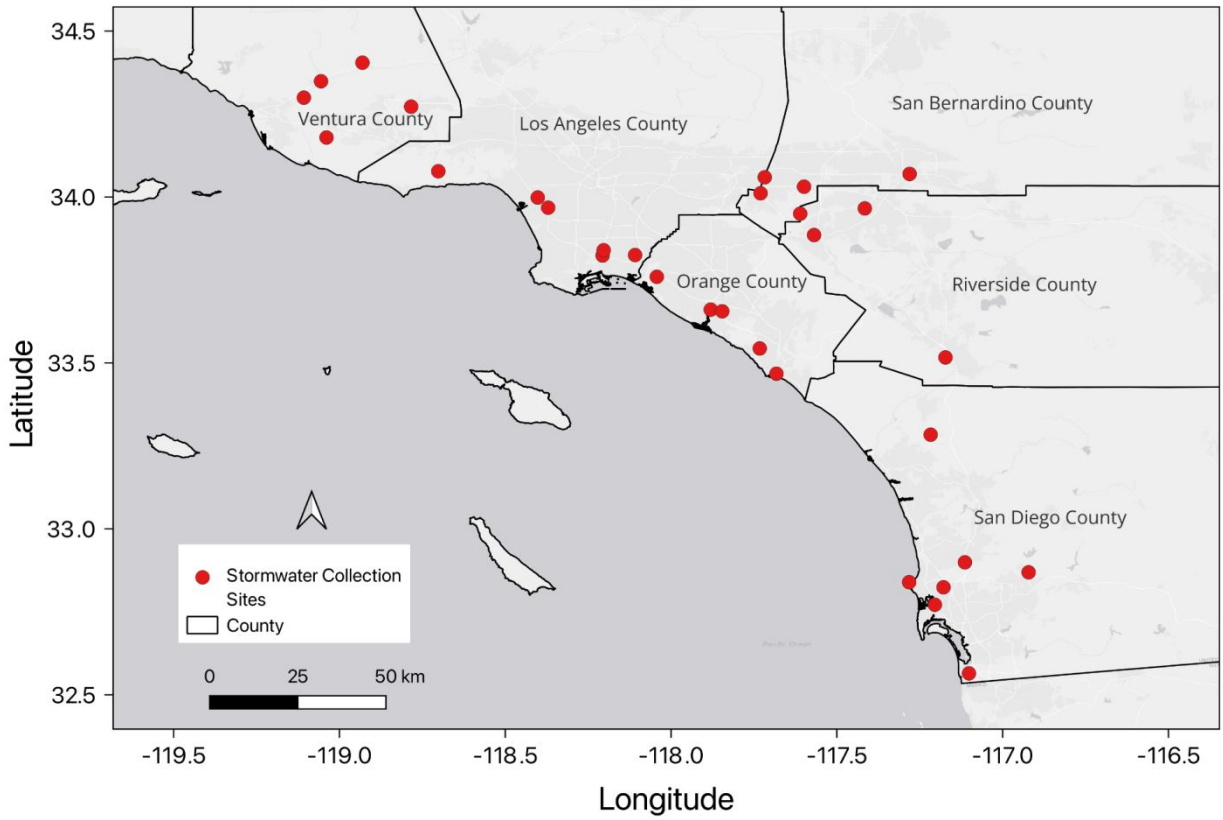

Figure 1. Sites in southern California where stormwater samples were collected during wet weather events. Made in QGIS with the Esri Light Gray Canvas basemap. Sources: Esri, TomTom, Garmin, FAO, NOAA, USGS, © OpenStreetMap contributors, and the GIS User Community.

After collection, samples were transported to the laboratory and processed within 12-24 hours. Concentrations of HF183, *Salmonella*, *Campylobacter jejuni*, *Campylobacter coli*, adenovirus, norovirus GI, and norovirus GII were measured in the stormwater samples (Table 1). The pathogen targets were selected for measurement because they have previously been detected in Southern California stormwater (34,42–46) and are recommended reference pathogens for QMRAs for recreational waters (47). HF183 was measured as an indicator of human fecal contamination and so that a risk-based threshold for HF183 could be derived.

A full description of the methods used to make these measurements and the results are reported elsewhere (12). In short, measurements of bacteria and DNA viruses were completed using droplet digital -polymerase chain reaction, ddPCR, and measurements of RNA viruses were completed using droplet digital reverse-transcription polymerase chain reaction, dd-(RT)PCR. Concentrations were reported by Steele et al. (12) in units of gene copies/100mL stormwater following dMIQE (48,49) and EMMI (50) guidelines.

Table 1. Targets ("Target") measured in stormwater, assays used for quantification ("Assay"), and their classification ("Type") as an indicator or pathogen.

Target	**Assay**	**Type**
Bacteroides 16S	HF183 (51,52)	Human fecal indicator
Salmonella	invA (53)	Bacterial pathogen
Campylobacter coli	glyA (54,55)	Bacterial pathogen
Campylobacter jejuni	hipO (54,56)	Bacterial pathogen
Adenovirus	JVTX (57)	Viral pathogen
Norovirus GI	NV1LC (58)	Viral pathogen
Norovirus GII	QNIFS (58)	Viral pathogen

Exposure Assessment

We modeled risk using a static microbial risk assessment via a Monte Carlo simulation. We first modeled risk for a scenario in which recreational swimmers are exposed to the pathogens in Table 1 as measured in stormwater via accidental ingestion during a recreational exposure event. Although not all sampling sites were designated for contact recreation, we assumed that recreators ingested undiluted stormwater in this scenario. We then modeled several scenarios where recreators are exposed to stormwater diluted with ambient water with no contamination. We did not consider decay of pathogens, and we assumed that recreators had no prior immunity to any pathogen. We assumed that pathogen concentrations are independent from one another, an assumption that is supported by the finding that pathogen concentrations are not significantly correlated (12).

Ingestion of stormwater in milliliters (mL) from recreational swimming activities was modeled using a truncated $\log_{10}$-normal distribution with a mean of 1.20, a standard deviation of 0.68, and a maximum of 2.45 (1). This distribution came from a study that used self-reported time spent in the water for over 68,000 children and adults to simulate the volume of water ingested per swimming event (1). The distribution was truncated at 2.45 (279 mL) because it was the maximum volume of water ingested observed in an ingestion study of recreational swimmers (59).

We used the values from Tables S8 and S9 of Steele et al.(12) as inputs to build a pathogen concentration distribution. Pathogen concentrations measured in stormwater in units of gene copies/100 mL were assumed to follow lognormal distributions (60). To account for highly left-censored concentration datasets, we used a maximum likelihood estimation-multiple imputation (MI-MLE) method that was found to minimize error when compared to five commonly used

methods (61). We chose this method because it performed best in predicting infection risks and minimizing bias as compared to substitution, maximum likelihood estimation, and Kaplan-Meier estimation methods (61), and it did not require the identification of a single LOQ value for a concentration dataset as is required in a Hurdle model. In this method, distribution parameters were estimated using maximum likelihood estimation assuming a lognormal distribution using the fitdistcens function from the R package fitdistrplus (62). Values below the LOQ were then imputed using the estimated parameters. With this complete dataset, we used the fitdist function from the R package fitdistrplus to estimate distribution parameters for each dataset (62). The MI-MLE process was repeated 1,000 times for each pathogen and the median parameters were selected for use to estimate final distribution parameters.

Dose Response Assessment

A literature review was performed to identify dose-response functions for the target pathogens (Table 2). Briefly, two types of searches were performed in PubMed: one using the terms “dose response” + “ingestion” and the name of the pathogen that was performed for each pathogen, and one using the terms “recreational water QMRA” to identify the dose-response functions used in previous recreational water QMRA studies. The dose-response functions identified from this review were examined to ensure that they modeled ingestion (and not some other exposure pathway like inhalation), represented the current state of the science, and had been previously used in recreational water QMRAs. Due to limited data on the norovirus GII and *Campylobacter coli* dose-response functions, norovirus GI and GII doses were summed and modeled using a norovirus GI dose-response function and *Campylobacter coli* and *Campylobacter jejuni* were summed and modeled using a *Campylobacter jejuni* dose-response function.

We made several assumptions in applying the dose-response functions in this study. First, we assumed one gene copy detected via ddPCR or ddRT-PCR translated to one viable and infectious pathogen (i.e., no dose harmonization), despite the units for *Salmonella, Campylobacter,* and adenovirus shown in Table 2 not matching the units used in this study. This was done because we lacked site-specific information on the translation of gene copies to other units (e.g., colony-forming units, plaque-forming units, tissue culture infectious dose). This assumption has been made in previous work (23). For the norovirus dose-response function, we assumed that norovirus is disaggregated in stormwater (i.e. the dose-response function “a” parameter is 0), which is supported by previous findings that the pH of environmental waters is typically higher than the isoelectric point of norovirus (63–65). We also assumed that the recreator population was composed only of Se+ individuals (i.e., those susceptible to norovirus infection) (65). These dose response functions have been used by others in QMRA models for recreational exposure (19,23,35,37,66–70) and when data for comparison was available outcomes of these models have agreed with results from concurrently conducted epidemiology studies (23).

Table 2. Dose-response functions for target organisms (“Target Organism”) with the unit of measurement used to derive the function (“Unit”), the probability of infection function (“P_{inf}”) ,the probability of illness given infection function (“$P_{ill|inf}$”) , and the reference for each dose-response function (“Ref.”).

Target Organism	Unit	P_{inf}	$P_{ill\|inf}$	Ref.
Salmonella	CFU	$1-(1+\mu/2884)^{-0.3126}$	$U(0.17-0.4)$	(41)
Campylobacter	CFU	$1-\ {}_1F_1(0.44, 0.44\ +\ 0.51, -\mu)$	$1-(1+\mu/0.88)^{-0.06}$	(71)
Adenovirus	$TCID_{50}$	$1-\ {}_1F_1(5.11, 5.11\ +\ 2.8, -\mu)$	$1-(1+\mu/6.53)^{-0.41}$	(72)
Norovirus	Genome copies	$1-\ {}_1F_1(0.393, 0.393\ +\ 0.767, -\mu)$	$1-(1+\mu/0.801)^{-3.19}$	(73)

${}_1F_1$ indicates a hypergeometric function, U() indicates a uniform distribution, μ is dose. Details on hypergeometric function computation can be found in the Supplementary Information.

Risk Characterization

A static QMRA model was used to characterize the probability of illness resulting from ingestion of stormwater using R version 4.1.2 (74). The static model does not consider secondary transmission or immunity (75), so the risk is that for recreators who have direct contact with the contaminated water.

The probability of illness was estimated for recreational exposure to different simulated waters: one that was 100% stormwater, and then waters that were 10^{-2}, $10^{-1.5}$, 10^{-1}, and $10^{-0.5}$ parts stormwater mixed with pathogen-free ambient water. Hereafter, these waters are described by their "stormwater fraction" (F_{sw}) which represents the fraction of the water sample that is composed of stormwater in Equation 1:

$$F_{sw} = stormwater/(stormwater\ + uncontaminated\ water) \quad \text{(Eqn 1)}$$

Pathogen concentrations measured in stormwater were multiplied by F_{sw} to obtain pathogen concentrations representative of the simulated dilution (undiluted stormwater F_{sw} = 1). We used the following equation (Eqn 2) to calculate the probability of illness for any single pathogen (P_{ill_i}) from exposure to water with a specific F_{sw}:

$$P_{ill_i} = DR\{VC * F_{sw}\} * P_{ill|inf} \quad \text{(Eqn 2)}$$

$DR\{\}$ is the dose-response function for the probability of infection, V is the volume of stormwater ingested in mL, C is the concentration of the pathogen in stormwater in gene copies/mL, F_{sw} is the fraction of stormwater, and $P_{ill|inf}$ is the conditional probability of illness given infection. The cumulative probability of illness from exposure to any pathogen in the model (P_{ill}) was given by the following equation (Equation 3):

$$P_{ill} = 1 - \Pi_i(1 - P_{ill_i}) \quad \text{(Eqn 3)}$$

The model was run 10,000 times for each simulated concentration using a Monte Carlo simulation in which model parameters V, $C_{Salmonella}$, $C_{C.\ coli}$, $C_{C.\ jejuni}$, $C_{adenovirus}$, $C_{norovirus\ GI}$, and $C_{norovirus\ GI}$ were randomly drawn from their respective distributions each iteration for a total of 50,000 model runs. For *Campylobacter,* C in Equation 2 was found by summing $C_{C.\ coli}$ and $C_{C.\ jejuni}$. For norovirus, C in Equation 2 was found by summing $C_{norovirus\ GI}$ and $C_{norovirus\ GII}$. We repeated this process with a narrower range of stormwater fractions to determine the stormwater fraction that most closely approximated the risk threshold. We calculated the median and interquartile range of the probability of illness for the 10,000 model runs for each simulated concentration.

Derivation of risk-based threshold for HF183

Given the measured distribution of HF183 in stormwater (12), we derived the concentration of HF183 that corresponds to the US EPA's recommended target illness threshold of 32 illnesses per 1000 recreators (6); hereafter, this is referred to as the risk-based threshold of HF183 in stormwater. To derive a risk-based threshold for HF183, we calculated a simulated HF183 concentration for each stormwater fraction F_{sw} described above. The simulated HF183 concentration was calculated by multiplying the median HF183 concentration measured in 100% stormwater by the stormwater fraction F_{sw}. The risk-based threshold for HF183 is the concentration of HF183 for the F_{sw} where median risk of illness is 32/1000.

Sensitivity Analysis

A sensitivity analysis was performed following the method outlined in Xue et al.(76)) and followed by similar QMRA analyses (35,38,67). In this method, the 25th, 50th, and 75th percentiles for each model input variable (V, $C_{Salmonella}$, $C_{C.\ coli}$, $C_{C.\ jejuni}$, $C_{adenovirus}$, $C_{norovirus\ GI}$, and $C_{norovirus\ GI}$) were tabulated. Then, holding all other variables at their 50th percentile value, the QMRA model was run for each variable twice: once at its 25th percentile value and once at its 75th percentile value. The P_{ill} value was recorded for each variable, and a ratio of $P_{ill,75}$ to $P_{ill,25}$ was calculated for each variable. A ratio of $P_{ill,75}$:$P_{ill,25}$ < 1 shows that the probability of illness decreases as the variable increases, a ratio of $P_{ill,75}$:$P_{ill,25}$ = 1 shows that the probability of illness is not affected as the variable increases, and a ratio of $P_{ill,75}$:$P_{ill,25}$ > 1 shows that the probability of illness increases as the variable increases.

Results

Exposure Assessment

The concentrations of pathogens used to build lognormal distributions for the model are described in detail in Tables S8 and S9 in Steele et al. (12). Table 3 shows the parameters of the lognormal distribution built for each pathogen that served as an input to the Monte Carlo simulation.

Table 3. Estimated Lognormal Parameters for Pathogen Distribution Inputs

Pathogen	Log Mean	Log Standard Deviation
Salmonella	1.71	2.91
Campylobacter coli	-8.6	6.66
Campylobacter jejuni	2.54	2.02
Adenovirus	0.45	2.00
Norovirus GI	-0.36	2.75
Norovirus GII	-4.49	5.08

QMRA for Exposure to Undiluted Stormwater

We calculated the probability of illness (P_{ill}) from recreational exposure to undiluted stormwater using a QMRA (Figure 2). Median P_{ill} associated with exposure to norovirus and *Campylobacter* (median P_{ill} = 0.037 and 0.036, respectively) are higher than those associated with exposure to adenovirus and *Salmonella* (median P_{ill} = 0.0018 and 2.2 x 10^{-5}, respectively). The median P_{ill} from exposure to all pathogens is approximately 19%, or 190 illnesses per 1000 swimmers. This is higher than the US EPA illness guideline of 32 illnesses per 1000 swimmers for a recreational swimming event (6). The median P_{ill} from norovirus or *Campylobacter* alone exceeds the US EPA's threshold of 32/1000 illnesses. Norovirus and *Campylobacter* exposures drive P_{ill} from exposure to stormwater, given that setting the concentrations of adenovirus and Salmonella to 0 yields a similar risk from exposure to stormwater (15% as compared to 19%) as shown in Figure S1 (see Supplementary Information). The cumulative median P_{ill} of 19% is higher than the sum of the individual median pathogen risks because the upper half of individual pathogen distributions are right skewed, causing an increase in the median P_{ill} as calculated using Equation 3.

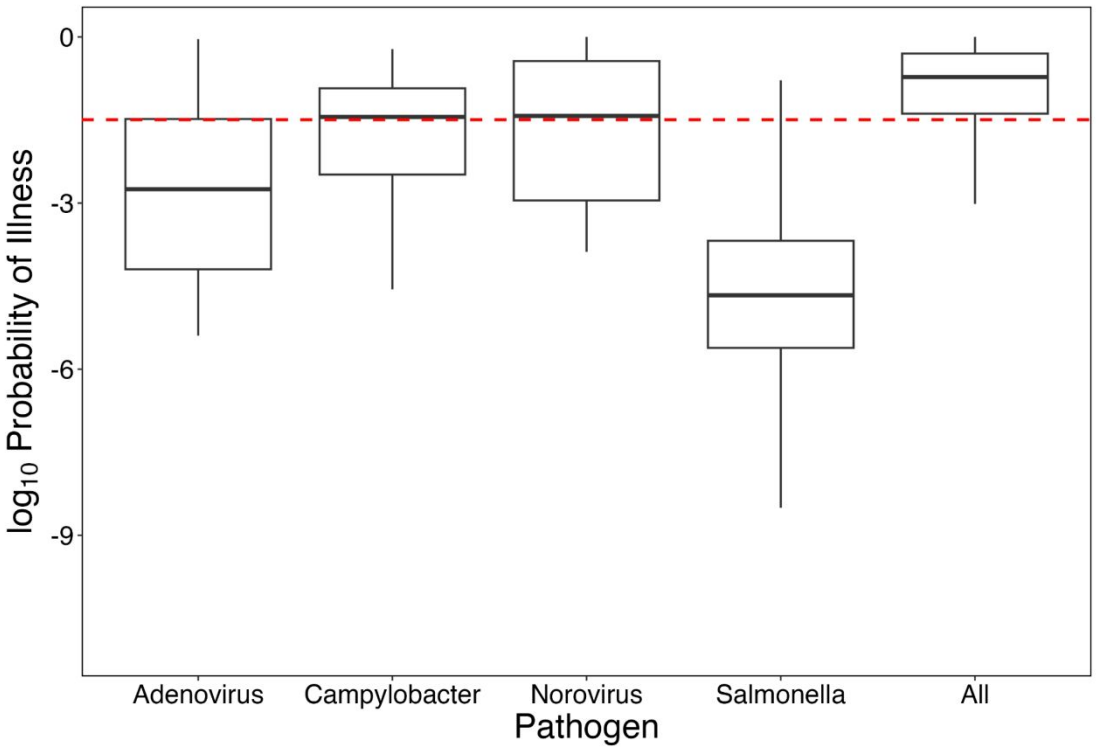

Figure 2. Box plot of log_{10}-transformed probability of illness by pathogen type, where "all" represents the probability that a recreational swimmer becomes ill from exposure to all pathogens included in the model. The horizontal line in the box represents the median, the bottom and top of the box represent the 25th and 75th percentile, and the top and bottom whiskers represent Q3 + 1.5*IQR (interquartile range) and Q1 - 1.5*IQR, respectively, from the 10,000 model runs in the Monte Carlo simulation. The red dashed line represents a log_{10}-transformed risk threshold of 32/1000 illnesses.

We next used the QMRA model to estimate the probability of illness from exposure to stormwater diluted with ambient water free of any pathogens (Figure 3). We modeled five stormwater fractions ranging from 0.01 to 1 and found that when stormwater is diluted between 10% and 30%, exposure to it is predicted to result in a median P_{ill} of 32 illnesses per 1000 recreators. We then modeled a smaller subset of stormwater fractions from 0.16 to 0.2 to identify the stormwater fraction that most closely corresponded to the US EPA threshold (Figure S2). We found that at a stormwater fraction of 0.18, the median P_{ill} of 33 illnesses per 1000 recreators was closest to the US EPA threshold of 32 illnesses per 1000 recreators.

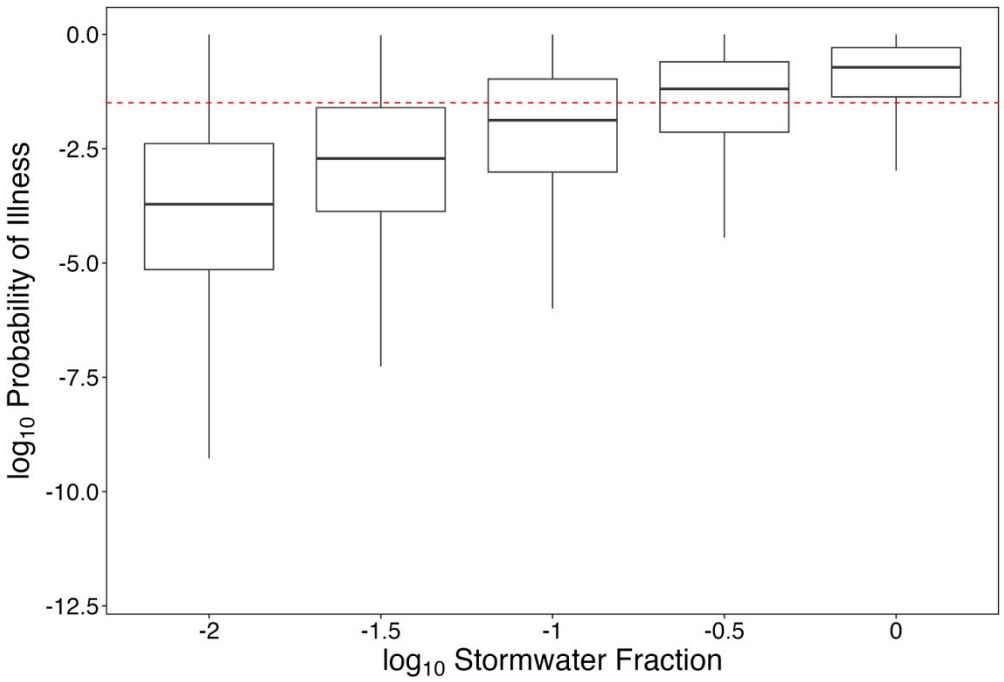

Figure 3. Box plots of log_{10}-transformed probability of illness versus log_{10}-transformed stormwater fraction. The horizontal line in the box represents the median, the bottom and top of the box represent the 25th and 75th percentile, and the top and bottom whiskers represent Q3 + 1.5*IQR and Q1 - 1.5*IQR, respectively. The red dashed line represents the log_{10}-transformed risk threshold of 32/1000 illnesses.

HF183 Risk-Based Threshold

The median concentration of HF183 measured in stormwater was 557 gene copies/100 mL (25th percentile 139 gene copies/100 mL, 75th percentile 3441 gene copies/100 mL, 90th percentile 10389 gene copies/100 mL). Using this median HF183 concentration and the stormwater fraction of 0.18, the median HF183 concentration at which risk is equal to the US EPA 32/1000 illnesses threshold is 100 gene copies/100 mL (25th percentile 25 gene copies/100 mL, 75th percentile 619 gene copies/100 mL, 90th percentile 1870 gene copies/100 mL).

Sensitivity Analysis

The sensitivity analysis found that the model is the most sensitive to the volume of water ingested, the concentration of *Campylobacter jejuni*, and the concentration of norovirus GI. Increases in these three parameters result in an increase in P_{ill}. *Campylobacter coli* concentration, *Salmonella* concentration, adenovirus concentration, and norovirus GII concentration had little to no effect on model output. The results of the sensitivity analysis can be found in Table 4.

Table 4. Results from Sensitivity Analysis

Parameter	P75:P25 Ratio
Salmonella concentration	1.004
Campylobacter jejuni concentration	6.231
Campylobacter coli concentration	1.001
Adenovirus concentration	1.522
Norovirus GI concentration	5.387
Norovirus GII concentration	1.235
Volume of water ingested	13.800

Discussion

While it is well established that fecal indicator bacteria in stormwater can be high (9,77), there are uncertainties associated with the human health risk from recreational exposure to stormwater. A previous literature review documented pathogen concentrations in stormwater, suggesting that microbial contamination of stormwater may represent a health risk (77). Particularly in highly urbanized environments, stormwater conveyances or locations adjacent to stormwater conveyances (e.g., beaches) may represent sites of recreational exposures particularly for groups like children (78) or surfers (23). In the present study, we used measurements of pathogens in stormwater runoff to model the risk from recreational exposure to the stormwater. We found that direct recreational exposure to stormwater results in a simulated risk of 190 illnesses per 1000 swimmers, higher than the USEPA recreational risk guideline of 32 gastrointestinal illnesses out of 1000 primary contact recreators. While this comparison assumes that swimmers are recreating in stormwater, it is important to note that not all sampling sites in this study were designated for recreational use and thus not all sites are subject to this risk guideline. Additionally, the USEPA recreational risk guideline represents an average of 30 days of both dry and wet weather.

Previous recreational water exposure QMRA studies have found that norovirus typically drives simulated gastrointestinal risk, and *Campylobacter* has been found to be a secondary driver (23,35–37,68). Similarly, in this study exposure to norovirus was the most important contributor to simulated risk, followed closely by *Campylobacter*. The pathogen concentration inputs to this QMRA model were empirical as compared to other QMRAs that relied on reference pathogen doses measured in raw sewage or feces in the literature from a variety of locations (35–37,68) or came from their own site-specific measurements (23). A previous study conducted at two beaches in San Diego, California measured the same pathogen targets as were measured here

with the addition of enterovirus and found that *Campylobacter* and norovirus were consistently detected in stormwater (79), in agreement with our observations.

Our model simulated risks that are consistent with those measured in an epidemiology study of surfer exposure to stormwater-impacted recreational water in our study region (18). This surfer study measured site-specific stormwater fractions that we could input in our model to estimate risk and compare to the epidemiology study's risk estimates. The epidemiology study observed a gastrointestinal illness incidence rate of 10.2 illnesses per 1000 surfers during wet weather conditions (18) and, in that study, it was estimated that during surfer exposures, stormwater was diluted in the ambient ocean water such that it represented 0.6% - 4% of the water (23). Dilution of stormwater in the ocean can occur due to mixing causes by tides, waves, and wind (80). We ran our QMRA model using F_{sw} = 0.6% - 4% and found that at a stormwater fraction of 0.6%, our model predicts a median probability of illness of 0.5 illnesses per 1000 recreators, and at a stormwater fraction of 4%, our model predicts a median probability of illness of 2.9 illnesses per 1000 recreators, lower than, but within a factor of ~3 of 10.2 illnesses per 1000 surfers. It is important to note that our model does not estimate risk to surfers specifically, which may make direct comparisons between model output and the epidemiology study results challenging. On the one hand, surfers might be more susceptible to pathogen contamination because they may ingest larger quantities of water during surfing than a general recreator (81). On the other hand, the surfers are typically healthy adults, and may have increased immunity to some illnesses due to high levels of exposures (82), and therefore may be a less susceptible population than the general public, especially children or immunocompromised individuals. Regardless, the similar illness rates produced by the studies is encouraging.

This study derived a risk-based threshold of 100 HF183 gene copies per 100 mL (25^{th} percentile 25 gene copies/100 mL, 75^{th} percentile 619 gene copies/100 mL) for ambient waters impacted by stormwater runoff. By treating stormwater as the source of pathogens and HF183 in a recreational water body, we simulated dilution of the source to estimate the fraction of stormwater at which the US EPA swimmer risk threshold of 32 illnesses per 1000 exposures is met, finding a stormwater fraction of 18%. This means that stormwater-impacted ambient waters with dilutions of stormwater lower than 18% would meet the risk threshold for recreation. Using the characterization of HF183 concentrations in the stormwater during wet weather events, we calculated a risk-based threshold for stormwater-impacted recreational water. This approach presumes that the predominant source of fecal contamination in stormwater is human; a presumption that is supported by the detection of HF183 at 90% of sites and the generally positive correlation between HF183 and pathogen concentrations in this study (12).

The regional wet-weather HF183 risk-based threshold developed in this study could be used in risk management by stormwater managers to prioritize sites when HF183 data are available for water quality improvement projects. While this study provides a risk-based threshold for recreational exposure to stormwater-impacted waters, there are still many factors for policymakers and regulators to consider before implementing a water quality application such as the number of samples necessary for confidently estimating a median HF183 concentration, frequency of sampling, duration of threshold exceedance, method limit of quantification, and

spatial representativeness in fast-flowing storm flows. Comparable to EPA water quality objective development(6), decisions whether median risk in combined dry and wet weather flows over 30 days or whether single sample thresholds at the 75th or 90th percentile during a single storm event are issues to be considered. All of these issues are particularly challenging in watersheds where storms are infrequent and/or intense.

As a number of studies have derived risk-based HF183 thresholds for different contamination scenarios, it is useful to place the one derived herein in context. Previous studies have relied on reference concentration distributions of raw sewage found in the literature, and explicitly considered sewage-contaminated recreational waters. It is important to note that these previous studies may have used different dose-response functions than we used; as newer data become available, risk models can be updated, and we updated our approach in the present study to the best available data. The risk-based threshold reported by Boehm et al.(35) for exposure to ambient water contaminated by sewage of an unknown age is several orders of magnitude higher than the one derived in this study (4100 copies/100 mL versus 100 copies/100 mL); the high value is influenced by the presence of fresh sewage in the contamination mixture which has a relatively low norovirus to HF183 concentration ratio compared to aged sewage as norovirus has a smaller decay rate than HF183 (35). Interestingly, the risk-based threshold for exposure to 4-day old, aged sewage (37) is comparable to the threshold we derived (171 copies/100 mL vs 100 copies/100 mL). It is not unreasonable to suspect that stormwater may contain aged sewage as a source of contamination.

There are several important limitations to this analysis. While we had wide geographic breadth, we only measured two to three storms per site. Hydrographs and pollutographs for each site and each storm may vary significantly, and sampling at different points of a hydrograph and pollutograph may cause significant variations in microbial concentrations. We measured certain pathogens and therefore could be missing contributions to risk from exposure to pathogens (e.g., rotavirus, *Cryptosporidium, Giardia*) not included in the model. The pathogen concentrations we used could be overestimated as we assumed that a gene copy detected via ddPCR was equivalent to an infectious pathogen. This could have resulted in gene copies from non-viable pathogens being classified as infectious pathogens. At the same time, a fraction of measurements were below the method LOQ, and we had to use a maximum likelihood estimation-multiple imputation method to impute concentrations that were below the LOQ. Stormwater is a highly complex matrix containing compounds that inhibit PCR, as observed in previous work in Southern California stormwater (34), meaning that the gene copies measured in this study may be underestimated. Regardless of these limitations, we have used the best available science and approaches to provide simulated risks.

Future work should focus on the characterization of pathogen and indicator concentrations in stormwater to better understand the underlying distributions of these targets and the development of methods for improved quantification of targets in stormwater. While pathogen quantification in stormwater is associated with methodological challenges (12), larger datasets of pathogen and human indicator concentrations can help improve the input distributions for a QMRA model. Comparing culturable and digital PCR measurements of pathogens would be

useful to determine an infectivity ratio for modeling, although this task may not be feasible for norovirus for which infectivity measurements are challenging (83). Additionally, determining the age and source (e.g., leaking sewer line, leaky septic, open defecation) of human fecal pollution in stormwater would be useful in addressing the source of pollution and validation of risk-based thresholds as compared to previously suggested values. As this model simulates dilution, future work that measures site-specific dilution of stormwater across inland and coastal sites could serve as a useful comparison to model outputs.

Conclusion

The risk estimates and HF183 threshold derived in this study provide an important understanding of stormwater risk and the relationship with HF183 concentrations in wet weather events in Southern California. The proposed risk-based threshold offers an interpretation of measured HF183 concentrations, setting a foundation for potential policy development with further research and consideration of external factors. The ability to connect a human-specific fecal indicator with human health risk in recreational waters provides an advantage over the usage of more general fecal indicators that may come from many sources. The model employed here can be widely applicable to any dataset containing pathogen and indicator concentrations from a specific source and can be used to set risk-based thresholds for different geographic settings or indicators.

Author Contributions

Sarah A. Lowry: methodology, formal analysis, data curation, writing - original draft, writing - review & editing, visualization; Joshua Steele: conceptualization, methodology, investigation, writing - review and editing; John Griffith: conceptualization, writing - review & editing; Ken Schiff: conceptualization, writing - review & editing; Alexandria B. Boehm: conceptualization, writing - original draft, writing - review & editing.

Conflicts of interest

There are no conflicts to declare.

Data Availability

Datasets and code for this manuscript can be found in the Stanford Digital Repository (https://purl.stanford.edu/zy346wk0898). Additional information on the pathogen and indicator concentrations used in this manuscript can be found in Steele et al.(12)

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Data Availability

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