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REVIEW OF PUBLISHED STUDIES TO CHARACTERIZE RELATIVE RISKS FROM DIFFERENT SOURCES OF FECAL CONTAMINATION IN RECREATIONAL WATER

U.S. Environmental Protection Agency
Office of Water
Health and Ecological Criteria Division

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ACRONYMS

AWQC	ambient water quality criteria
BEACH Act	Beaches Environmental Assessment and Coastal Health Act
CCDR	Canada Communicable Disease Report
CDC	U.S. Centers for Disease Control and Prevention
CFU	colony forming unit
CI	confidence interval
DNA	deoxyribonucleic acid
EC	European Commission
EEC	European Economic Community
EPA	U.S. Environmental Protection Agency
GI	Gastrointestinal
HUS	hemolytic uremic syndrome
MMWR	Morbidity and Mortality Weekly Report
MPN	most probably number
NM	nonmotile
NOAEL	no-observed-adverse-effect-level
NRC	National Research Council
PCR	polymerase chain reaction
QMRA	Quantitative microbial risk assessment
SCCWRP	Southern California Coastal Water Research Project
U.S.	United States
WHO	World Health Organization (United Nations)
WSAA	Water Services Association of Australia

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EXECUTIVE SUMMARY

Introduction

The overall goal of the current ambient water quality criteria for bacteria in the United States is to provide public health protection from gastroenteritis (gastrointestinal [GI] illness) associated with exposure to fecal contamination during water-contact recreation. Because fecal matter can be a major source of pathogens in ambient water, and because it is not practical or feasible to monitor for the full spectrum of all pathogens that may occur in water, water quality criteria are specified throughout the world in terms of fecal indicator organism densities. For decades, these fecal indicator organisms have served as surrogates for potential pathogens and subsequent health risks in both recreational and drinking waters.

The U.S. Environmental Protection Agency's (EPA) recommended recreational water quality criteria do not differentiate between fecal sources of pathogens. Thus, EPA's regulatory premise concerning recreational water quality has been that nonhuman-derived human pathogens in fecally contaminated waters are as hazardous as their human-derived counterparts. 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 microbiological indicators of fecal contamination can be "interpreted" using evidence of the presence or absence of human fecal contamination. This approach assumes that in general, sources other than human fecal contamination are less of a risk to human health. WHO indicated in their 2003 report *Health Based Monitoring of Recreational Waters: The Feasibility of a New Approach (The "Annapolis Protocol"*) 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."

Ultimately, the critical question is whether exposure to different fecal sources from recreational waters translates to significant differences in the risk of human infection or disease severity. The purpose of this white paper is to describe the existing knowledgebase available to characterize the relative risks of human illness from various sources of fecal contamination in recreational waters. Information related to human exposures to pathogens in fecally contaminated recreational and drinking waters was obtained by searching the scientific literature for epidemiological studies related to exposure to recreational waters and reports of outbreak investigations from both recreational and drinking waters.

Recreational Water Epidemiological Studies

Numerous epidemiological investigations have been conducted since the 1950s to evaluate the association between illness risk to recreational water users and the density of suitable fecal indicators. These studies have been conducted in Australia, Canada, Egypt, France, Hong Kong, Israel, the Netherlands, New Zealand, Spain, South Africa, United States, and the United Kingdom. Importantly, most of these studies investigated waters that were impacted or influenced by wastewater effluent.

Taken as a whole, the weight of evidence from these studies indicates that fecal indicator bacteria are able to predict GI and respiratory illnesses from exposure to recreational waters. However, as indicated above, most of these studies investigated waters that were impacted or influenced by wastewater effluent, and close inspection of this base of information reveals that few studies addressed sources of contamination other than wastewater effluent in the investigated waters. Prior to 1999, only three peer-reviewed publications addressed this topic substantially (Calderon et al., 1991; Cheung et al., 1990; McBride et al., 1998). In the last few years, researchers have conducted several additional epidemiological studies focusing on waters not predominately impacted by wastewater effluent. Additionally, the Southern California Coastal Water Research Project (SCCWRP) is also conducting a series of epidemiological studies that investigate recreational water with various contamination sources other than wastewater effluent.

Review of the epidemiological studies that address recreational water predominantly impacted by sources other than wastewater effluent indicates that the results are equivocal. For example, Colford et al. (2007) found that the incidence of swimmer illness was not associated with any of the traditional fecal indicators at a marine beach with primarily avian contamination. This result is substantially different than those studies described above on wastewater impacted waterbodies. Whereas, a study from New Zealand (McBride et al., 1998) indicated that illness risks posed by animal versus human fecal material were not substantially different.

Outbreak Reports for Recreational and Drinking Waters

In the United States, formal surveillance data on the occurrence and causes of waterborne disease outbreaks are collected through collaboration between EPA, the Council of State and Territorial Epidemiologists, and the U.S. Centers for Disease Control and Prevention (CDC). The goals of the surveillance program include characterizing the epidemiology of outbreaks, identifying the agents causing outbreaks as well as trends and risk factors, identifying deficiencies in providing safe drinking water, encouraging health officials to investigate outbreaks, and fostering government and international agency collaboration on waterborne disease prevention. The number of outbreaks reported is a significant underestimate of the actual number of outbreaks that occur, as the actual numbers reported vary depending on issues such as a lack of laboratory capability. Thus, the extent of underestimation is unknown overall. Even with these problems, surveillance studies provide the best information available on waterborne disease outbreaks and such data are critical to adequately characterizing microbial hazards.

In reviewing outbreak information for recreational and drinking water waters, several overarching points emerged. One is that the pathogen source in the majority of drinking water-related outbreaks remains unknown. The source of pathogens in drinking water outbreaks in many cases could have been humans or animals. Most reports, however, offered little detail, leaving a critical information gap for the purposes of this review. Nevertheless, several outbreak investigation studies were able to link pathogens isolated from patients with water samples and/or animals using laboratory analysis. Other reports used circumstantial evidence to link animal waste to outbreaks; however, although compelling, laboratory results were not available to confirm the contamination source. The animal sources linked to outbreaks included beavers, cats/cougars, deer, elk, pigs, cattle, and chickens/poultry, and the corresponding animal-related human pathogens in these outbreaks were *Giardia intestinalis*, *Cryptosporidium* spp., *E. coli*

O157:H7, *Campylobacter* spp., *Toxoplasma gondii*, and *S. typhimurium*. Given that outbreaks are known to be a notoriously poor measure of the actual number of infections and illnesses caused by waterborne pathogens, those investigations that link pathogens isolated from patients and/or water samples with animals provide unequivocal evidence that human illnesses can and do occur from animal-based contamination. Unfortunately, the drinking water outbreak literature does not substantially enhance the current ability to quantitatively differentiate risks from animal- versus human-related pathogen sources for recreational water exposures.

The recreational water outbreak literature (Craun et al., 2005) indicates that of the 259 recreational water outbreaks that occurred in the United States between 1970 and 2000, only approximately half included any information about possible sources of the contamination or the sources contributing to it. Approximately 18 percent of the total outbreaks were associated with animals, likely etiologic agents included *E. coli* spp., *Schistosomes* spp., and *Leptospira* spp. *E. coli* was associated with cattle, deer, or duck feces; *Schistosomes* spp. were associated with snails; and *Leptospira* spp. were associated with rat urine. Similar to the drinking water outbreak compilation, the recreational water outbreak literature does not appear to substantially enhance the current state of knowledge on quantitatively characterizing risks from animal-related pathogen sources compared with human sources for recreational water exposures.

Interpretation of Results

Given that relatively few investigations worldwide have evaluated the risk to human health from recreational exposure to waters primarily impacted by sources of contamination other than wastewater effluent, and that the potential range of those sources is broad, the findings from this literature review are not surprising.

Although information on differentiating human versus animal sources of pathogens is lacking, several research organizations and countries have suggested novel approaches for addressing risks from nonhuman sources (e.g., Water Services Association of Australia). For example, New Zealand, where about 80 percent of total notified illnesses are zoonotic and potentially waterborne, has recently updated its recreational water quality criteria to address the issue of animal-source waterborne contamination by basing its freshwater guidelines principally on the risks associated with campylobacteriosis using *E. coli* concentrations as an indicator (Till and McBride, 2004).

In summary, both human and animal feces in recreational waters continue to pose threats to human health. Although the public health importance of waterborne zoonotic pathogens is being increasingly recognized, it is still not well characterized. Policy makers and researchers have often assumed that the human health risk from pathogens associated with domestic and agricultural animal and wildlife feces is less than the risk from human feces, in large part because viruses are predominately host-specific. This literature review illustrates a lack of detailed and unequivocal information concerning the relative risks of human illness resulting from exposure to various sources of fecal contamination in recreational waters. Because of their retrospective nature, waterborne disease outbreak investigations rarely produce the data needed to draw conclusions about the impact of a pathogen source. Finally, the ability to measure how

the infectivity and virulence of known waterborne zoonotic pathogens are affected when passaged through animal hosts remains in its infancy.

I. BACKGROUND AND INTRODUCTION

I.1 Background: Context and Purpose

Since the U.S. Environmental Protection Agency (hereafter EPA or the Agency) last published recreational water quality criteria in 1986, significant advances have occurred, particularly in the areas of molecular biology, microbiology, and analytical chemistry. EPA believes that these new scientific and technical advances need to be considered and evaluated for feasibility and applicability in the development of new or revised Clean Water Act (CWA) Section 304(a) criteria for recreation. The Beaches Environmental Assessment and Coastal Health (BEACH) Act of 2000 (which amended the CWA) required EPA to conduct new studies and issue new or revised criteria, specifically for Great Lakes States and coastal marine waters. To this end, EPA has been conducting research and assessing relevant information to provide the scientific foundation for new or revised criteria.

To address the BEACH Act requirement, EPA has engaged a range of stakeholders representing the general public; public interest groups; state, local, and municipal governments; industry; and wastewater treatment professionals. In March 2007, EPA convened 43 national and international technical, scientific, and implementation experts from academia, numerous states, public interest groups, EPA, and other federal agencies at a formal workshop to discuss the state of the science on recreational water quality research and implementation (USEPA, 2007). The workshop outcome included a suggestion to incorporate the ability to differentiate sources of fecal contamination and determine the relative human health risk from these sources into the new or revised criteria.

Based on the feedback from the larger group of stakeholders and detailed input and recommendations from the scientific community, the Agency developed a *Critical Path Science Plan for Development of New or Revised Recreational Water Quality Criteria*. A key question the science plan asks is what is the risk to human health from swimming in water contaminated with human fecal matter as compared to swimming in water contaminated with nonhuman fecal matter? Human and animal feces both can potentially contain pathogens that cause human illness. However, while some human pathogens are host-specific (i.e., human enteric viruses), other human pathogens can be shed by both humans and animals. In other words, all enteric pathogens of humans are infectious to other humans, while relatively few of the enteric pathogens of animals are infectious to humans (zoonotic pathogens). Understanding which human pathogens are associated with which source of fecal contamination would allow the Agency to recommend better (more appropriate) water quality criteria in those situations.

EPA's current recommended criteria treat the human health risk from various sources of fecal contamination as equivalent based on health risks from fecal contamination originating from publicly owned treatment works, which represent the highest relative risks to swimmers. Because health risks from other sources (e.g., poorly treated or untreated human waste, nonhuman fecal matter, and mixed sources, such as urban stormwater runoff) were not well understood at the time the 1986 criteria were developed, the approach was to protect human health regardless of the source. However, EPA recognizes that the health risk from fecal sources other than publicly owned treatment works may be different and that recent scientific advances

may now allow the Agency to better characterize the relative risks to human health from different sources of fecal contamination. Specifically, the Agency is interested in understanding what human illnesses are caused by swimming in waters contaminated with human fecal matter from various sources, including human sources (wastewater effluent and untreated human fecal matter), nonhuman fecal matter (ranging from wildlife sources to agricultural inputs), and mixed sources, such as urban stormwater runoff.

The purpose of this white paper is to describe the existing knowledge base available to characterize the relative risks of human illness from various sources of fecal contamination in recreational waters. An overview of recreational water epidemiology studies and results from illness outbreak investigations are discussed in an attempt to demonstrate any differential risk to human health that may exist from various sources of fecal contamination.

I.2 Introduction

The overall goal of the current ambient water quality criteria (AWQC) for bacteria in the United States (USEPA, 1986) is to provide public health protection from gastroenteritis (GI illness) associated with exposure to fecal contamination during water contact recreation. Because fecal matter can be a major source of pathogens in ambient water, and because it is not practical or feasible to monitor for the full spectrum of all pathogens that may occur in water, water quality criteria are specified throughout the world in terms fecal indicator organism densities (USEPA, 1986; WHO, 2003). These fecal indicator organisms have been used for decades as surrogates for potential pathogens and subsequent health risks in both recreational and drinking waters (NRC, 2004).

The Agency used prospective cohort epidemiological studies of wastewater effluent-impacted recreational waters (USEPA, 1986) to develop the 1986 AWQC for recreational water. The results of those epidemiological studies provide quantitative relationships between fecal indicator density in recreational waters (*E. coli* and enterococci for freshwaters and enterococci for marine waters) and GI illness levels for those individuals exposed to recreational waters (USEPA, 1986). Using randomized controlled trials, Kay et al. (1994) showed fecal streptococci (enterococci[1]) to be predictive of gastroenteritis (GI illness) among bathers in the United Kingdom. Fleischer et al. (1996) reported that exposure to fecal streptococci also could predict acute febrile respiratory illnesses. These studies formed the basis of the guidelines recommended by the World Health Organization (WHO, 2003). In addition, detailed reviews conducted by Wade et al. (2003) and Prüss (1998) strongly support the findings that increased enterococci (or fecal streptococci) concentrations predict GI illness risk (Lepesteur et al., 2006).

Internationally, many countries still rely on fecal and total coliforms as a basis for their recreational water quality criteria, standards, or guidelines (see WHO, 1999). Other countries rely on measurements of enterococci, *E. coli*, or both for their recreational waters, most based on criteria recommendations provided by WHO (2003) and/or on EPA's epidemiological studies that led to the 1986 criteria.

[1] The terms fecal streptococci, enterococci, intestinal enterococci, and *Enterococcus* are often used to refer to essentially the same environmental and fecal species of bacteria (see NRC, 2004 for further information). Thus, for convenience, this white paper uses the term enterococci unless otherwise noted.

EPA's recommended recreational water quality criteria do not differentiate between fecal sources of pathogens. Thus, EPA's regulatory premise concerning water quality has been that nonhuman derived human pathogens when present in fecally contaminated waters are as hazardous as their human-derived counterparts (Schaub, 2004). This presumption is supported by scientific literature that confirms that there are many waterborne zoonotic bacteria and protozoa common to both humans and to various types of animal populations (especially mammalian species). The literature also suggests, however, that in certain instances there may be attenuation of the infectivity, virulence, and disease severity to humans from animal-derived ("passaged") human pathogens. Moreover, "[w]e must also recognize that more than half the bacteria in the human intestine and more than 99 percent of environmental bacteria have not been cultured or characterized…This is almost certainly also true for the broad array of domestic and wild animals in our environment" (Bolin et al., 2004).

WHO's recommended approach for classifying the water quality of recreational waters relies on combining microbiological monitoring results for fecal indicators and a sanitary inspection (WHO, 2003). This approach is based on the premise that the measure of microbiological indicator of fecal contamination can be "interpreted" using evidence of the presence or absence of human fecal contamination; the approach also presumes that in general, sources other than human fecal contamination present a lesser risk to human health (WSSA, 2003). WHO indicated 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).

WHO highlighted this issue of potential differential risks between human and nonhuman sources in its recent report on zoonoses (WHO, 2004), which noted that the inability to distinguish human from animal fecal contamination has led resource managers and regulators to treat all fecal contamination as equally hazardous to human health. The report further indicated the following (Till and McBride, 2004):

- This approach frequently results in the closure of beaches and shellfish harvesting areas that are affected by stormwater runoff that carries fecal indicator bacteria of nonhuman origin.
- The true risk of exposure to waters contaminated by animals is not well characterized.
- Studies that have attempted to define the risk associated with swimming in animal-contaminated water have not given a clear indication that there is an excess illness rate related to this type of exposure.
- These equivocal results do not lead to the conclusion that all fecally contaminated waters should be treated alike.
- New research to define the risk posed by animal fecal wastes to users of water resources and indicator systems that identify animal contamination of surface waters are needed.

Ultimately, the merit of an AWQC approach that treats animal and human sources of pathogens and indicators differently or somehow discounts animal sources depends on both the characterization of relevant differences between human and animal fecal material and the availability of technology that can accurately and reliably differentiate between their sources. These issues are of concern to the development of new or revised recreational AWQC because

the normal microbial composition of animal feces is different from human feces and can change dramatically over time and space, especially in recreational waters (Table I.2.1) (Boehm et al., 2002; Dorner et al., 2007; NRC, 2004). Moreover, the critical question is whether these differences in fecal sources resulting from recreational water-related exposures translate to significant differences in the risk of human infection or disease severity.

Table I.2.1. Representative Fecal Indicator Bacteria and Zoonotic Pathogen Densities in Human and Animal Feces and Sewage*

Indicator	Observed Density	Source
Bacteria		
Fecal coliforms	10^8-10^9/gram feces	Infected person
Fecal coliforms	4.9×10^{10} organisms/day	Canada geese
Fecal coliforms	1.4×10^8-2.4×10^9 organisms/day	Chicken
Fecal coliforms	5.4×10^9 organisms/day	Cows
Fecal coliforms	4.2×10^8 organisms/day	Horses
Fecal coliforms	8.9×10^9-1.1×10^{10} organisms/day	Pigs
Fecal coliforms	1.8×10^9-1.2×10^{10} organisms/day	Sheep
Fecal coliforms	5×10^9 organisms/day	Dogs and cats
Fecal coliforms	10^5-10^7 most probable number (MPN)/100 mL	Sewage
Fecal coliforms	4.2×10^6 organisms/100 mL	Combined sewer overflow
Fecal coliforms	9.6×10^2-4.3×10^6 organisms/100 mL	Urban runoff
Fecal coliforms	1.2×10^2-1.3×10^6 organisms/100 mL	Grazed pasture runoff
Fecal coliforms	1.35×10^6-2.4×10^8 organisms/100 mL	Feedlot runoff
Fecal coliforms	1.2×10^1-1.43×10^4 organisms/100 mL	Cropland runoff
E. coli	1.7×10^8 E. *coli*/gram	Pigeons
E. coli	10^3-10^4 colony forming units (CFU)/100 mL	Stormwater
Enterococci	4.0×10^5 enterococci/day	Pigeons
Enterococci	10^2-10^3 enterococci/mL; 5.4×10^5 enterococci/100 mL	Sewage

Indicator	Observed Density	Source
Pathogens		
Bacteria		
E. coli O157:H7	10^2-10^6 per gram feces	Calves
Pseudomonas aeruginosa	2.3 x 10^5 MPN/100 mL	Sewage
Salmonella	0.2-11,000 MPN/100 mL	Sewage
Protozoa		
Protozoan parasites	10^6-10^7/gram feces	Infected person
Cryptosporidium oocysts	370 ± 197 oocysts/gram feces 1.2 x 10^5-3.9 x 10^5 organisms/ day	Canada geese
Cryptosporidium oocysts	13.7/mL	Slaughterhouse (cattle)
Cryptosporidium oocysts	1.4 x 10^4-3.96 x 10^4/L	Treated effluent (activated sludge)
Cryptosporidium oocysts	4.0 x 10^0-1.6 x 10^1/L	Treated effluent (activated sludge and sand filtration)
Cryptosporidium oocysts	3-13,700/1,000 mL	Sewage
Giardia cysts	450 cysts/gram feces; 3.1 x 10^5 cysts/day	Canada geese
Giardia cysts	2-200,000/1000 mL	Sewage
Viruses		
Enteric viruses	10^3-10^{12}/gram feces	Infected person
Adenovirus	1-10,000/100 mL	Sewage
Enterovirus	0.05-100,000/100 mL	Sewage
Reovirus	0.1-125/100 mL	Sewage
Rotavirus	0.1-85,000/100 mL	Sewage
Caliciviruses	10^6/gram (10^{13}/day)	Gray whales**

* Although marine mammal caliciviruses are not known to be human pathogens, they have been being investigated as potential sources of emerging zoonoses (Smith et al., 1998).

Source: Adapted from: *Protocol for Developing Pathogen TMDLs* (USEPA, 2001), *Impacts and Control of CSOs and SSOs: Report to Congress* (USEPA, 2004), and *Waterborne Zoonoses: Identification, Causes, and Control* (WHO, 2004).

II. METHODS

As indicated previously, the primary purpose of this white paper is to describe the existing knowledge base available to characterize the relative risks of human illness from various sources of fecal contamination in recreational waters. The paper specifically covers human illness resulting from water-related exposures to fecal material from human and nonhuman sources. Although the ultimate use of this information is to support the development of new or revised water quality criteria and/or standards for recreational waters, this review encompasses both recreational and drinking water exposures.

Information related to human exposures to pathogens in fecally contaminated recreational and drinking waters was obtained by searching the scientific literature for epidemiological studies related to exposure to recreational waters and reports of outbreak investigations from both recreational and drinking waters. Appendix A details the specific search strategies and databases employed.

This literature review combines disparate information to infer potential differences in risk between various sources of contamination and the limitations in each class of study are important. For example, in epidemiological studies conducted on recreational waters, the amount of water ingested during recreation is often not known. In many cases, studies also include multiple potential sources of waterbody contamination. Furthermore, outbreaks are a notoriously poor measure of the actual number of infections and illnesses caused by waterborne pathogens (Craun, 2004). The timely investigation of a waterborne outbreak by the appropriate professionals who have access to adequate laboratory resources can provide information about an outbreak's mode of transmission, the etiologic agent, and sources of contamination. However, many outbreaks are not recognized or investigated, and even in recognized outbreaks, not all cases of disease are reported; the likelihood of reporting is dependent on many factors, including pubic awareness of waterborne illnesses (such as media coverage of an event), the local requirements for reporting, and the availability of laboratory facilities (Craun et al., 2005).

Often, gaps in data result in an incomplete picture of the incident—especially in recreational water, where water testing may occur days or weeks after the outbreak started. In many recreational water outbreaks, by the time an outbreak is discovered and the etiologic agent is isolated, it is too late to provide meaningful data regarding the source of disease.

An example of the difficulty of linking illness with its source in recreational water occurred during an outbreak of *E. coli*-related illness in the Netherlands (Cransberg et al., 1996). Four children hospitalized in one town with hemolytic uremic syndrome prompted a search for a single source of illness. The results of the investigation pointed to a shallow lake where the ill children had all been swimming during a 5-day period. Pulsed-field electrophoresis of *E. coli* isolates of patients and family members showed an identical pattern: no *E. coli* DNA could be detected in filter-concentrated lake water samples using polymerase chain reaction (PCR). However, water samples were not taken until two weeks after the exposures.

The principal methodology employed for this white paper is provided below:

- Summarize the recreational water epidemiology studies that have been published worldwide.
- Provide a brief description of the most influential recreational water epidemiology studies as well as those that investigated waters impacted with sources other than wastewater and wastewater effluent.
- Summarize waterborne disease outbreaks associated with drinking water in the United States reported by the CDC from 1999 to 2004.
- Summarize select waterborne disease outbreaks associated with drinking water from other countries and from the United States before 1999.
- Summarize waterborne disease outbreaks associated with recreational water in the United States reported by the CDC from 1999 to 2004.
- Summarize select waterborne disease outbreaks associated with recreational water from other countries and from the United States before 1999.
- Provide brief descriptions of reported drinking water and recreational outbreaks with animal-related pathogen sources.
- Compile the information described above and determine the weight of evidence available to demonstrate any differential risk to human infection, disease severity, or both resulting from exposure to fecal material from human or animal sources in recreational water.

III. RESULTS: RECREATIONAL WATER EPIDEMIOLOGICAL STUDIES

III.1 Overview of Recreational Water Epidemiological Studies

Since the 1950s, numerous epidemiological studies have been conducted throughout the world to evaluate the association between recreational water quality and adverse health outcomes, including GI symptoms; eye infections; skin complaints; ear, nose, and throat infections; and respiratory illness (Prüss, 1998; Sinton et al., 1998; Wade et al., 2003; Zmirou et al., 2003). Although most of these studies investigated wastewater effluent-impacted marine and estuarine waters alone or in combination with freshwater, several investigated freshwater recreational environments or nonwastewater effluent-impacted waters. These studies indicate that the rates of some adverse health outcomes are higher in swimmers compared with nonswimmers (Prüss, 1998).

Several groups of researchers have compiled information and generated broad and wide-ranging inferences from these epidemiological studies. Below is a brief overview of the meta-analyses conducted by Prüss (1998), Wade et al. (2003), and Zmirou et al. (2003); Table III.1.1 provides an overview of the epidemiological studies included in these reviews.

- Prüss (1998) conducted a systematic review following discussions between the WHO Regional Office for Europe and WHO Headquarters to initiate development of new guidelines for recreational use of the water environment. The comprehensive review of 22 published studies on sewage pollution of recreational water and health outcomes concluded that the epidemiological basis had been laid to develop WHO guidelines on fecal pollution based on a causal association between GI illness symptoms and increased bacterial indicator density (i.e., enterococci for marine, enterococci and *E. coli* for fresh) in recreational waters.
- A meta-analysis of 18 published studies (Zmirou et al., 2003) was conducted by researchers at the National Institute for Public Health Surveillance at the request of the French Ministry of Health to help provide a scientific basis for establishing new standards for the microbial quality of marine and fresh recreational waters to replace the 30 year-old European Union bathing water quality guidelines (EEC, 1976). The researchers provided four major results: (1) increased concentrations of fecal coliforms or *E. coli* and enterococci in both fresh and marine recreational waters are associated with increased risks of acute GI illness, with enterococci eliciting four to eight times greater excess risks than fecal coliforms or *E. coli* at the same concentrations; (2) GI illness risks associated with enterococci occur at lower concentration in marine versus fresh recreational waters; (3) increased concentrations of total coliforms have little or no association with GI illness risk; and (4) no evidence exists of a threshold of indicator density below which there would be no GI illness risk to bathers.
- Wade et al. (2003) conducted a systematic review and meta-analysis of 27 published studies was to evaluate the evidence linking specific microbial indicators of recreational water quality to specific health outcomes under nonoutbreak (endemic) conditions. Secondary goals included identifying and describing critical study design issues and

Table III.1.1. Recreational Water Epidemiology Studies Included in Reviews by Prüss (1998), Wade et al. (2003), and Zmirou et al. (2003)

First Author	Year	Location	Water Type	Study Design	Review Article
Alexander et al.	1992	UK	Marine	Cohort	Wade, Zmirou
Bandaranayake	1995	New Zealand	Marine	Cohort	Prüss
Brown	1987	UK	Marine	Cohort	Zmirou
Cabelli	1983	USA	Marine	Cohort	Wade, Prüss, Zmirou
Cabelli	1983	Egypt	Marine	Cohort	Wade, Prüss
Calderon et al.	1991	USA	Fresh	Cohort	Wade
Cheung et al.	1990	Hong Kong	Marine	Cohort	Wade, Prüss, Zmirou
Corbett et al.	1993	Australia	Marine	Cohort	Wade, Prüss, Zmirou
Dufour	1984	USA	Fresh	Cohort	Wade, Prüss, Zmirou
Fattal et al.	1986	Israel	Marine	Cohort	Wade, Prüss, Zmirou
Ferley et al.	1989	France	Fresh	Cohort	Wade, Prüss, Zmirou
Fewtrell et al.	1992	UK	Fresh	Event	Wade, Zmirou
Fewtrell	1994	UK	Marine	Cohort	Wade, Zmirou
Fleisher et al.	1993	UK	Marine	Randomized trial	Wade
Fleisher et al.	1996	UK	Marine	Randomized trial	Prüss
Foulon et al.	1983	France	Marine	Cross-sectional	Wade
Haile et al.	1996, 1999	USA	Marine	Cohort	Wade, Prüss, Zmirou
Kay et al.	1994	UK	Marine	Randomized trial	Wade, Prüss, Zmirou
Kueh et al.	1995	Hong Kong	Marine	Cohort	Wade, Prüss
Lee et al.	1997	UK	Fresh	Event	Wade
Lightfoot	1989	Canada	Fresh	Cohort	Wade, Prüss
Marino et al.	1995	Spain	Marine	Cohort	Wade
McBride et al.	1998	New Zealand	Marine	Cohort	Wade
Medema et al.	1995	The Netherlands	Fresh	Event	Wade
Medical Research Council	1995	South Africa	Marine	Cohort	Prüss
Mujeriego	1982	Spain	Marine	Cohort	Prüss
Philipp et al.	1985	UK	Marine	Event	Wade, Zmirou
Pike	1994	UK	Marine	Cohort	Wade, Prüss, Zmirou
Prieto et al.	2001	Spain	Marine	Cohort	Wade
Seyfried et al.	1985	Canada	Fresh	Cohort	Wade, Prüss, Zmirou
Stevenson	1953	USA	Fresh	Cohort	Wade, Prüss, Zmirou
UNEP / WHO	1991a	Israel	Marine	Cohort	Prüss
UNEP / WHO	1991b	Spain	Marine	Cohort	Prüss
van Asperen et al.	1998	The Netherlands	Fresh	Event	Wade, Zmirou
van Dijk	1996	UK	Marine	Cohort	Prüss
von Schirnding et al.	1992	South Africa	Marine	Cohort	Wade, Zmirou

and evaluating the potential for health effects at or below the current regulatory criteria (USEPA, 1986). The researchers concluded that (1) enterococci and to a lesser extent *E. coli* are adequate indicators (predictors) of GI illness in marine recreational waters, but fecal coliforms are not; (2) the risk of GI illness is considerably lower in studies with enterococci and *E. coli* densities below those established by EPA (1986), thus providing support for their regulatory use; (3) *E. coli* is a more reliable and consistent predictor of GI illness than enterococci or other indicators in fresh recreational waters; and (4) based

on heterogeneity analyses, studies that used a nonswimming control group and that focused on children found elevated GI illness risks.

In addition, Sinton et al. (1998) reported on differentiating the relative health risks associated with human and animal fecal material. Illness risk associated with bathing in water polluted primarily with human fecal material was reviewed based on studies from the United States (Cabelli, 1983a; Dufour, 1984), Canada (Seyfried et al., 1985a,b), Israel (Fattal et al., 1983, 1986, 1987), Egypt (Cabelli, 1983b; El-Sharkawi and Hassan, 1979), Spain (Mujeriego et al., 1982), France (Ferley et al., 1989; Foulon et al., 1983); the United Kingdom (Brown et al., 1987; Jones et al., 1989, 1991), Hong Kong (Holmes, 1989), and Australia (Corbett et al., 1993; Harrington et al., 1993). Most of these studies showed a positive correlation between GI illness and fecal indicator density; there was little equivalent evidence from waters polluted primarily with animal feces. The only study specifically designed to address swimming-associated illness in animal-impacted waters was that of Calderon et al. (1991) who found no statistically significant association between GI illness and fecal indicator bacteria densities. Based on this observation, Sinton and colleagues (1998) concluded that reliable epidemiological evidence was lacking, and that other sources of information were needed to identify and apportion human and animal fecal inputs to natural waters.

Since the publication of Sinton's review article in 1998, a number of additional epidemiological studies have been conducted, some of which further address the issue of nonwastewater effluent-contaminated recreational waters. Completed and ongoing studies are summarized in Tables III.1.2a and III.1.2b, respectively; however, a comprehensive peer-reviewed summary of that information has not, to date, been published.

The following section provides brief summaries of the epidemiological studies on non-wastewater effluent sources of contamination along with those studies on human sources that have most commonly been used as the basis for water quality criteria or standards throughout the world.

III.2 Descriptions of Key Recreational Water Epidemiological Studies

III.2.1 Selected Recreational Water Epidemiological Studies Based on Human Sources of Fecal Contamination

United States and Egypt (Cabelli, 1983a)
Cabelli (1983a) conducted epidemiological studies in the following four locations: Coney Island and Rockaway beaches in New York, New York; Alexandria beaches in Egypt; Lake Pontchartrain in New Orleans, Louisiana; and Boston Harbor in Boston, Massachusetts. The goals of the studies were to develop health effects and recreational water quality criteria for EPA. The author used similar methods for each location. The studies consisted of two interviews—one interview at the beach where individuals were recruited and a second phone interview 7 to 10 days later. Beach interviews occurred only on weekends and were limited to individuals who had swam on one or two weekend days. Mid-week swimmers were excluded from the study.

Table III.1.2a. Recently Completed Recreational Waters Epidemiological Studies—Some of Which Address Nonpoint Sources of Fecal Contamination

Reference	Location	Study Design	Contamination Source and Indicators	Health Effect(s)	Health Correlation[1]	n	Conclusions
Colford et al., 2005, 2007	United States	Cohort	Nonpoint source: dominant source avian; bacterial indicators via traditional and nontraditional methods, *Bacteroides*, viruses	14 health outcomes evaluated including GI and skin rash	Negative	8,800	Risk of illness was not correlated with levels of the traditional water quality indicators used in the study. Of particular note, the state water quality thresholds for bacterial indicators (similar to the 1986 AWQC for bacteria) were not predictive of swimming-related illnesses. Similarly, no correlation was found between increased risk of illness and increased levels of most nontraditional water quality indicators measured in the study. Diarrhea and skin rash were increased in swimmers compared with nonswimmers.
Wade et al., 2006	United States	Cohort	Wastewater effluent- impacted waters; enterococci and *Bacteroides* measured via quantitative polymerase chain reaction (qPCR)	Gastro-enteritis	Positive	5,717	Swimmers at two beaches had a higher incidence of GI illness when compared to nonswimmers. A statistically significant relationship was observed between an increased rate of GI illness and enterococci at the Lake Michigan beach, and a positive trend for enterococci at the Lake Erie beach was noted. The association between enterococci and increased risk of GI illness was significant when results for the two beaches were combined. A positive trend was observed at the Lake Erie beach for *Bacteroides*, but no trend was observed at the Lake Michigan beach.
Wiedenmann et al., 2006	Germany	Random-ized control	Wastewater-impacted waters and waters impacted by waterfowl and swimmers. *E. coli*, intestinal enterococci, *C. perfringens*, somatic coliphage, and *Pseudomonas aeruginosa*	Acute febrile infection, ear, eye, skin, urinary tract, three definitions of GI	Positive	2,196	Data were not analyzed for differences in health outcomes from exposure to animal versus human fecal contamination. Authors recommended no observed adverse effect levels (NOAELs) for water quality as follows: 100 *E. coli*, 25 intestinal enterococci, 10 somatic coliphages, or 10 *C. perfringens* per 100 mL. The authors further concluded that a NOAEL approach would be a more robust method to the complex process of setting standards.
Dwight et al., 2004	United States	Cross-sectional survey	Nonpoint source; compared urban to rural runoff	Diarrhea; vomiting, sore throat	Positive	1,873	Urban participants reported almost twice as many symptoms overall as the rural participants during the first year and slightly more during the second year of the study. In both study years, risk increased across almost every symptom category by an average of about 10% for each additional 2.5 hours of water exposure per week. The study did not measure water quality at the various sites.
Lepesteur et al., 2006	Australia	Cohort	Evaluated for fecal streptococci; source recreational users and runoff	Respiratory illness and GI illness	Positive	340	Risk of respiratory illness was highest in the 11–15 year old age group, where vigorous activity such as jumping and energetic swimming was observed. The authors suggested further exposure-related research.

[1] The health correlation represents the correlation between the health effect observed and the indicator.

Table III.1.2b. Ongoing Recreational Waters Epidemiological Studies—Some of Which Address Nonpoint Sources of Fecal Contamination

Study	Location	Study Design	Pathogen/ Pathogen Source	Health Effect(s)	Health Correlation[1]	n	Conclusions
Southern California Coastal Water Research Project (SCCWRP) Studies	United States	Cohort, prospective	30 different microbial indicators including rapid methods and new microbial indicators	20 different epidemio-logical outcomes	Not applicable (N/A)	N/A	SCCWRP is conducting a series of epidemiology studies in Southern California to examine the risk of swimming-related illness from nonpoint source polluted waters. The studies are being conducted at three different beaches. The fecal contamination at these beaches is estimated to be mostly human at one beach, primarily nonhuman at the second beach, and a mixture of human and nonhuman at the third beach. Water samples will be collected at multiple locations and times and evaluated with over 30 microbiological indicators. The indicators to be tested include traditional fecal indicator bacteria, rapid methods, new microbial indicators, microbial source tracking tools, and viruses. SCCWRP hopes that these studies will help to determine the relative risk of contracting an illness from swimming at nonpoint source beaches and whether the risk of illness under these conditions correlates with traditional indicator density and/or new indicators or methods.
Virobathe	European Union	Laboratory method evaluation	Adenovirus and norovirus	Gastro-enteritis	N/A	N/A	Virobathe is a project aimed at detection of viruses in recreational waters and is investigating molecular methods for direct viral pathogen enumeration (adenovirus and norovirus). The final report is currently being reviewed by the European Commission (EC)
Epibathe	European Union	Randomized control	Unknown	Unknown	N/A	N/A	Epibathe is a consortium of European research institutions carrying out an EC-funded project to explore the relationship between microbial indicators and health outcomes to define appropriate guideline levels. The objective is to develop the dose-response relationships between fecal indicator density (*E. coli* and enterococci) and self-reported minor disease outcomes (e.g., gastroenteritis, skin irritation, eye ailments, and ear infections/symptoms) in Mediterranean bathing waters and freshwater bathing sites in a new member state. Studies are currently being conducted in Spain and Hungary and a parallel study is under consideration in the United States (Florida). The consortium is expected to report to the EC in late 2008.

[1] The health correlation represents the correlation between the health effect observed and the indicator.

Interviews occurred on days with minimal tidal effects and during time periods of peak beach usage (from 11 am to 5 pm). For the studies based in the United States, a total of 26,676 participants were interviewed, while 23,080 participants were interviewed in Egypt. Swimmers were defined as individuals whose upper body orifices were exposed to water. Nonswimmers who attended the same beach were the control population for the study.

Water samples were collected at two to three locations along the beach, typically three to four samples were collected during the peak beach usage. Water samples were analyzed for *Salmonella*, *Enterobacter-Citrobacter*, *Klebsiella*, fecal coliforms, and staphylococci. Results were analyzed using regression analysis.

In the New York study, two beaches were sampled. The Coney Island beach was considered "barely acceptable" in terms of pollution levels, whereas the Rockaways beach was deemed "relatively unpolluted." The author found statistically significant differences in the rate of GI symptoms in swimmers compared to nonswimmers at the Coney Island, but not Rockaways beach. The author also found a higher rate of respiratory symptoms in swimmers at Rockaways beach. The symptom rate for GI illness for swimmers at Coney Island was 10 out of 1,000 swimmers compared to 2 out of 1,000 swimmers for Rockaways beach. There the author determined that measurable health effects could occur on beaches that met existing recreational guidelines and standards. When examining the relationship of the indicator densities to GI symptoms, the author found that enterococci densities provided the best correlation; *E. coli* was the second best indicator.

The author was unable to find a beach that was both heavily affected with close point-source sewage discharges and used by a large number of people. Instead, several beaches with combined sewer outfalls in Alexandria, Egypt met those requirements. The author found that swimmers in more heavily polluted beaches experienced more symptoms than swimmers at less polluted beaches. Rates of GI symptoms appeared to follow *Enterococcus* and *E. coli* densities. However, the rates of illness were not as high as expected despite higher pollution levels than the New York study.

The author then conducted a focused study on visitors to the beach that were from Cairo. In that study, the rates of illness for swimmers was higher than for nonswimmers and there was more inverse relationship at the least polluted beach for time spent swimming and rate of illness compared to the other beaches. The rate of illness for visitors from Cairo was similar to the New York study.

The Lake Pontchartrain study was located near the mouth of Bayou St. John in a brackish area with limited tidal activity. The authors found statistically different rates of illness for swimmers versus nonswimmers in symptoms of vomiting, diarrhea, stomachache, earache, and skin irritations. In general, the GI symptom rates were higher in children than adults. The authors also found that enterococci appeared to be a better indicator of illness than *E. coli*, stormwater runoff appeared to be less harmful than sewage sources, and enterococci and *E. coli* may overstate risks of illness.

The Boston Harbor study took place on two beaches in 1978 and attempted to confirm the observation that significant health effects can be observed when there are low indicator densities. Although the author did not find statistically different illness in GI symptoms between swimmers and nonswimmers, the beach with the most pollution had a greater difference in the rate of illness between swimmers and nonswimmers, even if not statistically significant.

Overall, the Cabelli (1983a) studies found that there was an increased risk in GI illness from swimming in waters in increasingly polluted seawater. Enterococci also appeared to be a better indicator for illness from swimming in seawater than *E. coli* or other fecal indicators.

United States (Dufour, 1984)
Dufour (1984) conducted a series of epidemiological studies to identify whether a model developed by EPA to predict health effects based on marine water quality could also apply to freshwater, including identifying a water quality indicator that best describes the relationship between health effects and freshwater quality and determining whether the marine water criterion was applicable to freshwater. The studies were conducted at two U.S. beaches, Lake Erie in Erie, Pennsylvania (29,976 participants) and Keystone Lake in Tulsa, Oklahoma (16,363 participants).

The Lake Erie studies were conducted in 1979, 1980, and 1982 on beaches considered to have good or excellent microbial water quality. One site was located approximately three-quarters of a mile northwest of a sewage outfall for the City of Erie. The Keystone Lake studies were conducted in the summers of 1979 and 1980, and the beaches demonstrated variable water quality for bacteria. The two beach sites at Keystone Lake were located 3 and 5 miles from an outfall for wastewater treatment facility. In 1979, the treatment plant released an average of 120,000 gallons of unchlorinated sewage per day into the lake. In 1980, this practice was discontinued, and sewage passed through a settling lagoon, an aeration basin, and was chlorinated before being released into the lake.

Water samples were tested for *E. coli* and enterococci (includes *Streptococcus faecalis* and *Streptococcus faecium*). Fecal coliforms were also monitored for two years during the Keystone Lake studies and in two years of the Lake Erie studies. Statistical analyses examined the relationship between the occurrence of GI illness in swimmers compared with a nonswimming control group. Because of the small population of nonswimmers, the nonswimming control group was pooled from an entire season of beachgoers to form a single control population. To evaluate their relationship, the incidence of illness between swimmers and nonswimmers and the indicator bacteria density at the time of swimming were used. The model controlled for age as a confounding factor. A regression analysis determined whether there was a direct relationship between swimming-associated GI illness and microbial water quality. A correlation analysis determined which water quality indicator showed the strongest relationship to a swimming-related illness.

Researchers conducted surveys at the beaches on weekends when the beaches were more crowded and monitored water quality at the time of swimming activity. There were two phases of beach interviews−the first as beachgoers were leaving the beach area and a second follow-up telephone interview 8 to 10 days after swimming. Follow-up interviews were only conducted on

beachgoers who had not gone swimming the five days prior to the initial interview. For the study, swimming was defined as having all orifices immersed in water. Interviewers asked beachgoers during the initial interview about sex, age, race and ethnicity; whether a person swam; length of time and time of day in the water; illness symptoms they may have had in the previous week; and the reason, if a nonswimmer, why they did not enter the water. Follow-up interviewers asked the beachgoers about any illness symptoms that occurred since swimming at the beach.

In the Lake Erie studies, both sites met local and state standards for water quality. In general, symptom rates for swimmers were higher than nonswimmers in all categories; however, most symptoms were not statistically significant. Statistically significant differences between swimmers and nonswimmers were found in symptoms related to enteric diseases and tended to occur at the beach with poorer water quality.

In the Keystone Lake study, symptoms for enteric diseases tended to be higher in swimmers than nonswimmers. In 1979, there was only one other group of symptoms (fever, headaches lasting greater than three hours, and backache) that showed significant differences between swimmers and nonswimmers. In 1980, statistical differences between swimmers and nonswimmers were found at one or both sampling locations with symptoms for GI, respiratory, and other illnesses.

United Kingdom (Fleisher and Kay, 2006; Fleisher et al., 1993, 1996, 1998)
Fleisher and Kay (2006) and Fleisher et al. (1993, 1996, 1998) reported the results of randomized intervention trials at separate marine bathing locations to identify the potential dose-response relationships among swimmers exposed to marine waters contaminated with domestic sewage and the potential risk of nonenteric illness (e.g., acute febrile respiratory illness, ear, eye, and skin ailments). Fleisher et al. (1996, 1998) reported the final results of the four randomized intervention trials conducted at four separate marine bathing locations and incorporated the results on two of the locations, which were presented preliminarily in Fleisher et al. (1993). Fleisher and Kay (2006) analyzed the data presented in Fleisher et al. (1993, 1996, 1998) and Kay et al. (1994) to identify and quantify any risk perception biases that may have affected the observed association between skin ailments and exposure to marine waters contaminated with domestic sewage at one of the study locations. The Fleisher et al. (1998, 1996, 1993) studies are included in the meta-analyses of Prüss (1998), Wade et al. (2003), and Zmirou et al. (2003).

The four separate trials took place at four different locations during the summers of 1989 through 1992. The study population consisted of 1,216 adults (>18 years of age) that were recruited from populations located near the study locations during the 3 weeks prior to the exposure day. No recruits participated in more than one study cohort, and study participants were blinded to the outcome illness being studied. Study recruits participated in an extensive interview and medical examination no more than two days prior to the day of exposure. Researchers gathered data on a number of potential confounding factors (e.g., age, sex, general health, illness in previous three weeks, medications taken, water contact activities, other confounding factors).

Study participants were randomized into bathers (548 participants) and nonbathers (668 participants) who were unaware of their status until they arrived at the study location beach. Bathers entered into a defined area in the water, remained in the water for at least 10 minutes,

and completely immersed their heads in the water at least 3 times. Nonbathers remained on the beach in a designated area. On the day of exposure, study participants were interviewed again about their health and dietary habits and any water contact activities experienced between the initial interview and the day of exposure. Furthermore, one week following the exposure period, study participants were medically examined and interviewed extensively again for data on their health, diet, water contact activity, and other potential risk factors. Lastly, study participants were mailed a questionnaire to fill out with any other symptoms of illness and exposure to recreational water.

In addition, the water quality of the bathing area was sampled in 30-minute increments during the exposure period at various designated locations (sampled every 20 m at surf, mid [1 m deep], chest depth [1.3 to 1.4 m deep], and 30 cm below the surface). These water samples were analyzed via standard methods for the following indicators: total and fecal coliforms, fecal streptococci, and *P. aeruginosa*. Total staphylococci were also counted at three of the study sites. The sampling results were assigned to each individual bather within 15 minutes of exposure and within a maximum of 10 m of the place of exposure.

Linear trend, chi-square, and multiple logistic regression analyses were used to estimate the dose-response relationship between the microbiological water quality results and occurrence of gastroenteritis, acute febrile respiratory infection, and eye and ear ailments. The statistical analyses controlled for the effects of confounding factors, which included nonexposure-related variables associated with the incidence of these outcome illnesses. The results from participants who did not take part in the follow-up interviews or who reported illnesses of interest on the actual exposure day were excluded.

Fleisher et al. (1998) indicated that bathers had statistically significant ($p<0.05$) higher rates of gastroenteritis and ear and eye ailments as compared to nonbathers. Moreover, bathers exposed to ≥60 fecal streptococci per 100 mL had a statistically significant ($p<0.001$) higher rate of acute febrile respiratory infection relative to nonbathers. In addition, the authors reported that approximately 34.4 to 65.8 percent of illnesses reported by the study cohort could be associated with bathing in marine waters contaminated with domestic sewage. Fleisher et al. (1996) also noted that fecal streptococci exposure of ≥60 per 100 mL was predictive of acute febrile respiratory infection and indicated that a fecal coliform exposure of 100/100 mL was predictive of ear ailments. No indicator was found to predict eye aliments, although the risk of eye ailments was higher for bathers versus nonbathers. There was no statistically significant dose-response relationship found for skin ailments. Fleisher and Kay (2006) noted that the observed difference in skin ailments associated with exposure between bathers and nonbathers at one of the four study locations may be attributable to "risk perception bias."

Based on the results of these studies, the authors concluded that future epidemiological studies should incorporate the same study design to help control for the large disparity observed in risk estimates reported in previously published epidemiological studies. Additionally, Fleisher et al. (1996) noted that these study results demonstrated that a single indicator would not be sufficient to establish water quality standards aimed at protecting public health. Fleisher et al. (1998) concluded that illness associated with bathing in contaminated (marine) recreational waters is a

significant threat to public health, and further studies to assess the severity of illnesses associated with bathing in recreational waters contaminated with domestic sewage should be conducted.

United Kingdom (Kay et al., 1994)
Kay et al. (1994) conducted the first randomized control study to evaluate the heath effects associated with swimming in coastal waters. This study is included in the meta-analyses of Prüss (1998), Wade et al. (2003), and Zmirou et al. (2003). The 4-year study took place at four different locations during the summers from 1989 through 1992. This study builds on the preliminary results from two of the locations that were published in Fleisher et al. (1993). Although this study also uses the same study population that was used in the Fleischer and Kay (2006) and Fleisher et al. papers (1996, 1998), it focuses specifically at the dose-response relationship between gastroenteritis and exposure to marine water of varying microbial quality.

Linear trend and multiple logistic regression analyses were used to estimate the dose-response relationship between the occurrence of gastroenteritis and microbiological water quality (exposure). The statistical analysis controlled for confounding factors, such as nonexposure-related variables associated with gastroenteritis.

The results indicated that of the indicators measured, only fecal streptococci demonstrated a statistically significant dose-response relationship with gastroenteritis. The model used for bathers exposed to more than 32 fecal streptococci/100 mL demonstrated a significant relationship with the risk of gastroenteritis, whereas the model utilized for bathers exposed to less than 32 fecal streptococci/100 mL did not show a significant relationship. The latter relationship was independent of nonwater-related predictors of gastroenteritis.

Based on their results, the authors concluded that although none of the other microbiological indicators illustrated a significant relationship between measured concentrations and occurrence of gastroenteritis, the relationship between fecal streptococci concentration measured at chest depth and gastroenteritis is robust. However, the biological basis underlying this significant relationship is not known. The authors suggested that whatever causes gastroenteritis "copartitions" in seawater with fecal streptococci. The authors' findings also suggest that coliforms seem to have little value as indicators of gastroenteritis risk from sewage pollution of coastal waters and that fecal streptococci concentrations are a better microbiological indicator.

Spain (Prieto et al., 2001)
Prieto et al. (2001) conducted a cohort study to determine whether water polluted with sewage posed a danger to a bather's health and to determine the best microbiological indicator to predict illness. The study took place during the summer of 1998 on four beaches in northern Spain and included 1,805 participants. Water samples were collected 3 days a week at 10 am in the area of the beaches that usually had the highest bather density. Water samples were tested for total coliforms, fecal coliforms, fecal streptococci, *S. aureus*, and *P. aeruginosa*.

The epidemiological study consisted of two surveys—one survey at the beach and a follow-up survey within seven days of the beach interview. The surveys focused on family groups. Once an individual was selected for an interview, all other family members were interviewed. The interviews occurred on the same days that water samples were collected and timed so that a daily

average of 15 people were interviewed. During the follow-up interview, individuals were asked about a series of symptoms and additional personal information.

For the statistical analyses, study participants were divided into swimmer and nonswimmer categories. The authors conducted a bivariate analysis to examine the relationship of bathing, water quality, and illness. A multivariate analysis was carried out to determine the dose-response relation between indicator bacteria densities and their relation to health problems.

Water quality at the four beaches exceeded the European Community Directive guide standards for total coliforms (39.8 percent of samples), fecal coliforms (57.1 percent of samples), and fecal streptococci (in 37.9 percent of the samples). In the follow-up interviews, 7.5 percent of participants reported symptoms. The rate of these symptoms was significantly greater ($p=0.011$) for "holidaymakers" (9.9 percent) and "day trippers" (8.4 percent) than for residents (5.8 percent). The study did not find a significant difference in the incidence rate of each symptom between swimmers and nonswimmers, nor did the authors find a difference in the incidence of these symptoms between swimmers who submerged their head and those who did not.

The rate of symptoms did increase with the concentration of total coliforms. Gastrointestinal symptom incidence increased with total coliforms and fecal streptococci and was significantly higher in swimmers than nonswimmers.

United States (Wade et al., 2006)

Wade et al. (2006) conducted a prospective cohort study of beachgoers at two beaches in the Great Lakes area—one beach on Lake Michigan in Indiana and the second beach on Lake Erie in Cleveland, Ohio. Both beaches were impacted by effluent from wastewater treatment plants. First, beachgoers were recruited at beaches on the weekends and holidays during the summer of 2003. Each participating beachgoer completed and returned the questionnaire prior to leaving the beach. The questionnaire asked for information on demographics, swimming and other beach activities, consumption of raw or undercooked meat or runny eggs, chronic illnesses, allergies, exposure to animals, and other related health symptoms or encounters with sick people in the previous 48 hours. Second, following a 10 to 12 day period, a telephone interview was conducted to gather information about health symptoms experienced since the beach visit, as well as on other water-related activities, contact with animals, and consumption of high-risk foods.

Water samples were also collected three times a day on each study day and analyzed for *Bacteroides* and enterococci. The water samples were analyzed via a modified version of PCR. This new method provides a faster assessment of water quality. Respiratory and GI illness and ear, eye, and skin rash symptoms were evaluated. The authors only reported the GI illness results. Swimmers at both beaches had a higher incidence of GI illness compared to nonswimmers. A statistically significant relationship was observed between increased rate of GI illness and enterococci at the Lake Michigan beach, while a positive trend for enterococci at the Lake Erie beach was noted. The association between enterococci and increased risk of GI illness was significant when results for the two beaches were combined. In terms of *Bacteroides*, a positive trend was observed at the Lake Erie beach, but no trend was observed at the Lake

Michigan beach. In addition, the association between enterococci and GI illness strengthened with increasing exposure.

Australia (Lepesteur et al., 2006)
Lepesteur et al. (2006) conducted an epidemiological study examining the relationship between incidence of disease and exposure to fecal contamination in recreational water. The study area was the Peel Harvey estuary at Mandurah Bridge beach located 70 km south of Perth in Western Australia. Water circulation is influenced by tidal currents, wind, and river flow. The water recreational area was relatively small—approximately 500 sq. m.

The surveys were conducted in the afternoons with water samples collected each day and tested for fecal streptococci. Social surveys were conducted at the beach and through follow-up interviews by telephone two weeks later. A total of 119 groups or families and 340 individuals participated in the study. The interviews included questions related to frequency of beach visits, length of stay and days of visit, age of users, and beach activities. Exposure to recreational waters consisted of any visit to the estuary over 30 minutes that involved swimming, paddling, and playing in wet sand.

During the initial interview, participants were asked to provide information on their current health status. The follow-up interview asked who swam and whether anyone became ill during the two-week period since the initial interview. Evidence of GI and respiratory illnesses was recorded. Two or more symptoms were considered an incident of illness. Confounding factors such as food and drink intake, age, sex, history of diseases, pregnancy, additional bathing, jellyfish sting, travel, and period of exposure to sun were identified during the follow-up interview. For the analysis, the authors adjusted background illness rates using age-dependent probability factors. The odd ratios with 95 percent confidence intervals were used to quantify the magnitude of effect of exposure to recreational waters for different age groups.

The water showed evidence of fecal contamination year-round with increased concentrations caused by recreational users during the bathing season. Increased fecal indicator densities were also associated with rainfall. Most survey participants conducted a number of recreational activities at any visit to the beach, with 76 percent identifying swimming as one of their activities. The average number of exposures was 17 per person per season.

Respiratory illness comprised most of the reported symptoms, with GI illness being less common. The authors observed a relationship between excess respiratory illness and exposure to fecal streptococci. The highest odds ratio was observed in the 11 to 15 year-old age range. The authors attributed this result to greater exposure to water combined with vigorous activity.

III.2.2 Selected Recreational Water Epidemiology Studies Based on Nonwastewater Effluent Sources of Fecal Contamination

United States (Calderon et al., 1991)
Calderon et al. (1991) followed the health status and swimming activities of volunteer participants. This study is included in the meta-analysis of Wade et al. (2003). The study site was a small pond (3 ac) located in a semirural and largely forested community in Connecticut. An intense sanitary survey of the surrounding watershed indicated that no human sources of fecal contamination impacted the stream that fed the pond, and there were no point sources of microbial pollution.

Water samples were analyzed for *E. coli*, enterococci, fecal coliforms, *P. aeruginosa*, and staphylococci. A total of 104 families participated in the study. Swimming was defined as a day in which an individual swam in the pond, and during swimming activity, completely submerged their head and body beneath the surface of the water. There were 1,310 exposure-days accumulated by swimmers and 8,356 person-days for nonswimmers.

Water quality data were collected on 49 days of the study. The geometric mean *E. coli* density was 51 CFU/100 mL. Rain occurred on 16 of the 49 days of the study. The geometric mean densities of *E. coli* and fecal coliforms were determined to be over two times greater on rainy days than on dry days; levels of enterococci were four times higher on rain days than dry days. The densities of staphylococci and *Pseudomonas* were about the same during rainy and dry periods.

The symptomatic GI illness rate in swimmers was 22.9 per 1,000 person-days, whereas, in nonswimmers, the rate was 2.6 per 1,000 person-days. Gastrointestinal illness was also strongly associated with swimming when adjusted for age. However, no association was found between high fecal indicator bacteria densities and GI illness in swimmers or between swimmer illness and high-volume rain days. The authors suggested that swimming-associated illness may have been due to etiological agents that were transmitted from swimmer-to-swimmer via bathing water.

The data from this study indicate that illness in swimmers was not statistically associated with densities of commonly used fecal indicator bacteria in a recreational water whose source was rainwater runoff from a forested watershed. These results led the authors to conclude that currently recommended bacterial indicators (i.e., *E. coli* and enterococci for fresh recreational waters per USEPA, 1986) are "…ineffective for predicting potential health effects associated with water contaminated by nonpoint sources of fecal pollution."

Hong Kong (Cheung et al., 1990; Holmes, 1989)
Cheung et al. (1990) and Holmes (1989) described a prospective cohort study that was conducted in two phases in the summers of 1986 and 1987. The first phase involved four popular beaches and tested the epidemiological techniques used in the second phase (main study) that incorporated results from nine beaches. The main study was undertaken to provide data for deriving health-related bathing water quality standard specific to the conditions of Hong Kong. Six of the beaches in the main study were predominately impacted by human sewage, two were

impacted by livestock wastes (mainly pig excreta), and one was impacted by a combination of sewage and livestock waste. Each phase involved two parts, epidemiological studies using beachgoer interviews and follow-up telephone interviews and water testing for microbiological quality. Beachgoers (33,083) were recruited to participate in the study during the weekends and asked for contact information and any information on potential prestudy illness or swimming activities. The following day, telephone interviews were conducted to obtain information from these recruits on water exposure and any type of food eaten while at the beach. This information allowed the researchers to categorize the participants as swimmers or nonswimmers. Additionally, 7 to 10 days after the follow-up telephone interview, the study participants were interviewed again via telephone to gather information on any poststudy, mid-week swimming activities, any illness, any perceived symptoms, and the duration of any perceived symptoms.

Water samples were collected on the weekend days from 3 sampling points (50 to 150 m apart and 1 m in depth) at each beach in the study and in locations of high bather density. The water samples were analyzed for the following microbial indicators: fecal coliforms, *E. coli, Klebsiella* spp., fecal streptococci/enterococci, staphylococci, *P. aeruginosa*, *Candida albicans*, and total fungi. The beaches were categorized as "relatively unpolluted" and "barely acceptable" for each of the microbial indicators analyzed.

Study results suggest that overall perceived symptom rates were higher for swimmers than nonswimmers, and the rates of GI illness, ear, eye, skin symptoms, respiratory illness, fever, and total illness were significantly higher for swimmers as compared to nonswimmers. Furthermore, swimming-associated symptom rates for GI, skin, and respiratory symptoms, and total illness were significantly higher at barely acceptable beaches compared to relatively unpolluted beaches, indicating that the perceived symptoms were pollution-related. The authors concluded that *E. coli* is the best indicator of health effects—mainly gastroenteritis and skin symptoms—associated with swimming at beaches in Hong Kong. Staphylococci was also correlated with ear symptoms and respiratory and total illness. A linear relationship was established between the geometric mean of *E. coli* densities and combined gastroenteritis and skin symptom rates for individual beaches. However, no significant relationship between *E. coli* and gastroenteritis or skin symptom rates, nor staphylococci and ear or sore throat symptoms, could be established.

United States (Haile et al., 1999)

Haile et al. (1999) conducted the first large epidemiological study of persons who swim in marine recreational waters contaminated by urban runoff. This study is included in the meta-analyses of Wade et al. (2003) and Zmirou et al. (2003). The exposures of interest were swimming distances from storm drains, levels of bacterial indicators (total coliforms, fecal coliforms, enterococci, and *E. coli*) and presence of human enteric viruses. The study assessed persons who immersed their heads in ocean water at three beaches in Santa Monica Bay, California.

The study included 15,492 subjects, 13,278 of whom were contacted for follow-up health interviews and 10,459 of whom were ultimately included in the analysis. Distance from the storm drain where participants swam was noted (0, 1 to 50, 51 to 100, or 400+ yds). During the follow-up telephone interview, the occurrence of the following health outcomes was noted: fever, chills, eye discharge, earache, ear discharge, skin rash, infected cuts, nausea, vomiting,

diarrhea, diarrhea with blood, stomach pain, coughing, coughing with phlegm, nasal congestion, and sore throat.

The statistical analysis addressed, (1) are there different risks of specific outcomes among subjects swimming at different distances from a storm drain? and (2) are risks of specific outcomes associated with levels of specific bacterial indicators or enteric viruses?

The results of the study indicated that the risks of several health outcomes were higher for people who swam at storm drain locations compared to those who swam various distances from a drain. Other relevant findings include the following: (1) statistically significant increases in fever, chills, ear discharge, cough and phlegm, and respiratory disease in people who swam at a storm drain than those who swam 400+ yds from a drain; (2) the increase in "highly credible gastrointestinal symptoms" (i.e., GI illness with vomiting and fever as used in USEPA, 1986) was not statistically significant for those who swam at a drain compared to those who swam 400+ yds from a drain; and (3) no dose-response relationship was associated with swimming closer to a storm drain and illness (one would expect a gradient of increasing risk of illness to swimmers the closer they are to a storm drain). Furthermore, swimmers reported adverse health outcomes more often on days when the water samples were positive for viruses, suggesting assays for viruses may be informative for predicting risk. Notably, the risk of highly credible gastrointestinal illness for those swimming at a storm drain location was estimated to be 0.018 (i.e., 18 in 1,000 bathers), which is effectively identical to the current tolerable GI illness rate of 0.019 in marine waters (USEPA, 1986). However, Haile and colleagues noted that causal effects may be higher than reported because both distance from a storm drain and detections of fecal indicator bacteria are proxies for the presence of waterborne pathogens.

Based on the results, the authors concluded that "there may be increased risk of a broad range of adverse health effects associated with swimming in ocean water subject to urban runoff….Consequently, the prospect that untreated storm drain runoff poses a health risk to swimmers is probably relevant to many beaches subject to such runoff, including areas on the East, West, and Gulf coasts of North America, as well as numerous beaches on other continents." Other researchers have concurred with the Haile study conclusions; for example, Grant et al. (2004) stated that "contamination of the surf zone by dry weather runoff apparently increases the risk that marine recreational bathers will contract diarrhea and other acute illnesses."

New Zealand (McBride et al., 1998)

McBride et al. (1998) conducted a cohort study using a modified prospective cohort ("Cabelli protocol") epidemiological study design to ascertain the association, if any, between illness rates among beachgoers and the degree of fecal contamination of marine water from either human or animal sources. This study is included in the meta-analyses of Wade et al. (2003). It was conducted at seven popular bathing beaches during the summer of 1995. Three categories of beaches were selected, two control beaches (considered minimal impact), two rural beaches (animal waste impacted), and three oxidation pond beaches (human waste-impacted). On each of the 107 interview days, groups of bathers and nonbathers were interviewed at each beach using a standard questionnaire to obtain basic field data, eligibility, demographic information, foods eaten in the past three days that could influence outcome, and contact information. Three to five days following the initial interview, study participants were contacted to distinguish beach

swimmers and paddlers from nonswimmers and for information regarding illness and onset of symptoms. A swimming-associated illness risk was calculated by comparing illness risks between people who swam or paddled in the water (1,577 total) and nonexposed individuals (2,307 total).

Additionally, water quality samples were taken on the same day as the initial interview and analyzed for fecal coliforms, *E. coli*, and enterococci. Samples were taken approximately 10 cm below the water surface, twice per day, from the adult chest and knee depth, and generally from three locations spaced evenly along the beach. Water samples were collected on days when significant bathing occurred, regardless of weather.

Log-linear modeling of the results demonstrated a statistically significant association between illness and enterococci concentration, with the most significant associations observed among paddlers and long-duration swimmers (i.e., swimming more than 30 minutes). No significant relationships were found between the other fecal indicators and health risks. Furthermore, no significant differences in illness risks were found between the human- and animal-impacted beaches, although illness risks at both types of beaches were significantly higher than the control beaches.

The researchers concluded that the overall study results were affected by unexpectedly low levels of indicator organisms at the impacted beaches. In addition to requiring a greater range of fecal contamination at study beaches, the researchers concluded that any future studies designed to strengthen the current study conclusions would require larger (sufficient) numbers of bathers at the study locations.

Germany (Wiedenmann et al., 2006)
Wiedenmann et al. (2006) performed randomized controlled studies at five public freshwater bathing sites (four lakes and one river site) in Germany that had complied with current European standards for at least the three previous bathing seasons. These studies were conducted to obtain a greater scientific basis for the definition of recreational water quality standards. Sources of fecal contamination potentially impacting the waterbodies included treated and untreated municipal sewage, agricultural runoff, and contamination from water fowl. Data were not analyzed for differences in health outcomes from exposure to animal versus human fecal contamination.

A total of 2,196 participants were recruited from the local population. After a pilot study at one of the five locations that included only adults, the study was expanded to include children aged 4 years and up for the other four locations. Approximately two to three days before exposure, participants were interviewed and administered a brief medical examination. Volunteers deemed unfit for participation were excluded from the study.

On the day of exposure, study participants were randomized into equal groups of bathers and nonbathers. After arriving at their designated locations, study participants were interviewed a second time for information on symptoms occurring after the first interview and diet from the preceding two to three days. Bathers were exposed for 10 minutes in designated areas and had to

immerse their head at least three times. Following their exit from the water, bathers weres asked to report whether they had accidentally swallowed water.

During the exposure period, water samples for microbiological analysis were collected at 20-minute intervals and analyzed for *E. coli*, enterococci, *C. perfringens*, somatic coliphages, aeromonads, and pyocyanine-positive *P. aeruginosa*. Furthermore, one week following the exposure period, study participants were medically examined and interviewed again. Three weeks later, study participants were mailed a questionnaire to complete.

Data were analyzed for the following disease outcomes: acute febrile respiratory infection, common cold, ear and eye ailments, skin infections or symptoms, urinary tract infections, and three definitions of gastroenteritis that were based on the responses obtained from the questionnaires. Dose-response effects with no-observed-adverse-effect-levels (NOAELs) were determined for the three different definitions of gastroenteritis and four fecal indicator organisms, *E. coli*, enterococci, *C. perfringens*, and somatic coliphages. Relative risks for bathing in waters with levels above NOAELs compared with nonbathing ranged from 1.8 (95 percent CI, 1.2 to 2.6) to 4.6 (95 percent CI, 2.1 to10.1), depending on the definition of gastroenteritis that was used. Additionally, swallowing water as compared to not swallowing water resulted in significantly higher attributable risks above NOAELs than below NOAELs. Moreover, swallowing water below NOAELs did not result in any significant effect compared with nonbathing, while swallowing water above NOAELs always produced a significant effect. Based on the results of this study, the authors concluded that the NOAELs for water quality should be as follows: 100 *E. coli*, 25 enterococci, 10 somatic coliphages, or 10 *C. perfringens* per 100 mL. The authors further concluded that a NOAEL approach would be a more robust method to the complex process of setting standards.

United States (Colford et al,. 2005, 2007)
Colford et al. (2005) conducted an epidemiological study in Mission Bay, California, where historically, nearly 20 percent of the routine bacterial samples failed state water quality standards, but the dominant fecal source appears to be nonhuman. Microbial source tracking in Mission Bay has indicated that human fecal sources are minor contributors to overall microbial loading. Moreover, Mission Bay has an unusually long hydraulic residence time compared to other coastal systems, which likely affects the age and viability of waterborne fecal material.

The study focused on whether (1) water contact increased the risk of illness during the two weeks following exposure to water, (2) there were associations between illness and measured levels of traditional indicators of water quality among those individuals with water contact, and (3) there were associations between illness and measured levels of nontraditional (alternative) indicators of water quality among those individuals with water contact.

Nearly 8,800 participants were recruited for this cohort study from the 6 most popular swimming beaches in Mission Bay on weekends and holidays during the summer of 2003. Each participant provided their current state of health and degree of water exposure during their day at the beach. On the same day, water quality was monitored for traditional fecal indicator bacteria (enterococci, fecal coliforms, total coliforms). Participants provided their current state of health and degree of water exposure during their day at the beach. On the same day, water quality was

monitored for traditional fecal indicator bacteria (enterococci, fecal coliforms, and total coliforms). A subset of samples was also measured for nontraditional or alternative indicators, such as *Bacteroides* and viruses (somatic and male-specific phages, adenoviruses, and noroviruses). This subset was also tested with new methods for measuring traditional indicators, such as chromogenic substrate or quantitative PCR.

Ten to fourteen days later, the participants were contacted by phone and interviewed about symptoms of illness that occurred since their visit to the beach, including GI illnesses (diarrhea, nausea, stomach pain, cramps, vomiting, and highly credible GI illness [using two definitions]); respiratory illnesses (cough, cough with phlegm, nasal congestion or runny nose, sore throat, and significant respiratory illness); dermatologic outcomes (skin rash and infected cuts or scrapes); and nonspecific symptoms (fever, chills, eye irritation, earache, ear discharge, and eye irritation or redness). Multivariate analyses assessed relationships between health outcomes and degree of water contact or levels of water quality indicators. These analyses were adjusted for confounding covariates such as age, gender, and ethnicity.

Of the measured health outcomes, only skin rash and diarrhea consistently were significantly elevated in swimmers compared to nonswimmers. For diarrhea, this risk was strongest among children 5 to 12 years old. However, illness risk was not correlated with levels of the traditional water quality indicators used in the study. Of particular note, the state water quality thresholds for bacterial indicators (similar to the 1986 AWQC for bacteria) were not predictive of swimming-related illnesses. Similarly, no correlation was found between increased risk of illness and increased levels of most nontraditional water quality indicators measured in the study. Although a significant association was observed between the levels of male-specific coliphage and highly credible GI illness, nausea, cough, and fever, these associations were based on far fewer participants. Thus, the researchers urged caution in extrapolating these results.

The researchers concluded that the results of the study suggest the need for further evaluation of traditional indicators in circumstances where nonpoint sources of microbial pollution are the dominant fecal contributors to recreational waters.

United States (Dwight et al., 2004)

Dwight et al. (2004) compared rates of reported health symptoms among surfers in urban North Orange County, California and rural Santa Cruz County, California during the winters of 1998 and 1999 to determine whether symptoms were associated with exposure to urban runoff. Two cross-sectional surveys of 1,873 surfers were conducted. Surfers were selected as the study population because of their regular exposure to coastal waters. Data were gathered on reported health symptoms (diarrhea, vomiting, and sore throat) experienced during the previous three months.

Logistic regression was used to estimate adjusted odds ratios comparing symptom reporting rates between the two counties, stratified by year. The model included county, water exposure, gender, age, occupation, educational level, annual income, political outlook, and level of concern about water quality.

The results indicate that the urban participants (North Orange County) reported almost twice as many symptoms overall compared to the rural participants (Santa Cruz County) during the first year and slightly more during the second year. In both study years, risk increased across almost every symptom category by an average of about 10 percent for each additional 2.5 hours of water exposure per week. The study did not measure water quality at the various sites.

Based on the results, the authors concluded that discharging untreated urban runoff onto public beaches can pose health risks. Furthemore, large-scale epidemiological studies are needed to further characterize the health risks of people exposed to urban runoff in coastal waters.

United States (SCCWRP 2007-ongoing)
The SCCWRP is conducting a series of epidemiological studies to examine the risk of swimming-related illness from waters polluted by nonpoint sources of pollution. The studies are being conducted at three diverse beaches with contamination ranging from mostly human (untreated or poorly treated), to primarily nonhuman (likely avian), or a mixture of human and nonhuman fecal sources. Water samples at the various beaches will be collected at multiple locations and times and tested for over 30 different microbiological indicators. These indicators are grouped into five categories and include the following: (1) traditional fecal indicator bacteria; (2) rapid methods; (3) new, alternative microbial indicators; (4) microbial source tracking tools; and (5) viruses.

The studies are based on a prospective cohort design. Swimmers will be interviewed at the beach to determine their eligibility to participate in the study and contacted 10 to 14 days later for a follow-up interview. During the second interview, study participants will be asked to describe illness symptoms that have occurred since the initial interview. The SCCWRP hopes that these studies will help evaluate the relative risk of contracting an illness from swimming at beaches polluted with nonpoint sources. Furthermore, the researchers hope to determine whether the risk of illness under these conditions correlates with traditional indicator densities and/or new indicators or methods.

IV. OUTBREAK REPORTS FOR RECREATIONAL AND DRINKING WATERS

IV.1 Waterborne Disease Surveillance and Outbreak Reporting in the United States

In the Unites States, formal surveillance data on the occurrence and causes of waterborne disease outbreaks are collected through collaboration between EPA, the Council of State and Territorial Epidemiologists, and CDC. The goals of the surveillance program include characterizing the epidemiology of outbreaks, identifying the agents causing outbreaks as well as trends and risk factors, identifying deficiencies in providing safe drinking water, encouraging health officials to investigate outbreaks, and fostering government and international agency collaboration on waterborne disease prevention (Liang et al., 2006). State, territorial, and local public health departments are responsible for investigating and reporting outbreaks to CDC. Because this reporting is voluntary and originates with the investigating agency, it is called "passive" surveillance.

The number of outbreaks reported is a significant underestimate of the actual number of outbreaks that occur; the actual number reported varies depending on the issues mentioned above, and the extent of underestimation is unknown overall. However, CDC is collaborating with EPA to prepare an estimate of how many waterborne illnesses occur in the United States (Craun and Calderon, 2006; Messner et al., 2006). The adequacy of this approach depends on good surveillance data and reasonable estimates for under-reporting cases.

CDC defines an outbreak as at least two people who are linked by location of exposure to water, time, and illness (Liang et al., 2006). Evidence must implicate water as the probable source of the outbreak, which is often difficult. CDC uses a classification system to rank the strength of the data available based on an outbreak investigation. The classification system is two-pronged, based on epidemiological and water quality data, and ranges from Class I, which indicates adequate epidemiological and water quality data, to Class IV, which indicates that epidemiologic data was available, although limited, but that the water quality data was either absent or inadequate. CDC weights epidemiologic data more heavily and will include an outbreak that does not include any water quality information, provided the epidemiological evidence is sufficient (Liang et al., 2006).

Despite the problems noted above, surveillance studies provide the best information available on waterborne disease outbreaks, and such data are critical to adequately characterizing microbial hazards (Embrey, 2002).

IV.2 Summary of CDC Surveillance Reports on Drinking Water

CDC systematically tracks the following water-related parameters in their reports: type of water system (e.g., community, individual, noncommunity, and bottled); water source (e.g., well, spring, and pond); and the water treatment deficiency (e.g., source water contamination, treatment deficiency, and distribution deficiency). The classification for deficiencies is further broken down by point of contamination and whether or not the water was treated or meant to be

ingested. Although the report narrative occasionally mentions evidence of a sewage leak or other pathogen origin, based on standard reporting parameters, it is impossible to determine systematically whether the source of contamination was of human or animal origin.

Craun et al. (2006) summarized CDC statistics on waterborne disease since the first year that data were collected in 1920 to 2002, including outbreaks caused by chemical and microbial contamination of drinking and recreational water. During that time, over 880,000 illnesses have been reported, including over 1,000 deaths—mainly due to typhoid fever before 1940. In terms of microbial etiologies, *Giardia* and *Cryptosporidium* have been the most frequently identified from 1990 to 2002, which the authors attribute to their high infectivity and imperviousness to many conventional water treatment practices. Addtionally, reports have identified a wider variety of pathogens in recent years, including newly emerging pathogens, such as *Cyclospora*. Although laboratory testing has improved dramatically, many etiologies of outbreaks of waterborne disease are still undetermined.

The deficiencies linked with waterborne disease outlets have also changed. Water treatment deficiencies have become a much less important cause (probably due to strengthened regulations and better water treatment practices), while distribution system problems have increased in importance. Further, the proportion of outbreaks associated with surface water sources of drinking water has decreased, while the proportion of outbreaks associated with groundwater has stayed constant. Because the predominant pathogens and causes identified in outbreaks have changed over the years, it is likely that new regulations or better enforcement of water treatment regulations, as well as advances in both treatment and laboratory detection methodologies, will affect future surveillance reports.

Starting in its 2002 report, CDC began disaggregating details of individual outbreaks and including data on water quality parameters when available. Table IV.2.1 below summarizes CDC data on individual drinking water-related disease outbreaks from the last three reports covering outbreaks from 1999 to 2004 (Blackburn et al., 2004; Lee et al., 2002; Liang et al., 2006). Outbreaks that may have been related to animal contamination are shaded gray.

IV.3 Summary of Selected Drinking Water Outbreaks Reported in the United States and Internationally

To augment the results reported by CDC, a comprehensive literature search using the U.S. National Library of Medicine's PubMed system was conducted (see Appendix A for further information). Search terms included "drinking AND water AND outbreak," which resulted in 495 hits and "waterborne AND outbreak," which produced 448 hits. The results from these searches were combined and the result was included in our review if the abstract showed evidence that the etiologic agent was detected in the source water (Table IV.3.1). Although this is a higher bar than what CDC uses to evaluate outbreak evidence, the large quantity of international articles with questionable outbreak investigation methods required a more stringent standard for inclusion in the review.

The summary of reports from outside the United States and U.S. reports older than 1999 that may provide useful information, but need to be interpreted within their context. For example,

countries in Northern Europe (Finland, Norway, and Sweden) often have municipal water supply systems that use unchlorinated groundwater in areas of sparse population (Hanninen et al., 2003), which makes the water supply susceptible under certain common conditions—every outbreak but one reported in Finland between 1998 and 1999 occurred in undisinfected groundwater (Miettinen et al., 2001). Also, several giardiasis outbreaks were associated with beavers, but none has been reported in the past 15 years. In the 1980s, researchers considered beavers to be a primary source of zoonotic *Giardia* in the environment; however, recent research suggests that the importance of animals as a risk factor for human infections is unclear (Hunter and Thompson, 2005; Lane and Lloyd, 2002). The outbreaks that are possibly related to animal sources are shaded in Table IV.3.1.

Table IV.2.1. Select Waterborne Disease Outbreaks Associated with Drinking Water in the United States Reported by the CDC (1999 to 2004)*

Reference	Location	Etiologic Agent	Number of Cases	Water Source	Water Quality/Environmental Information	Pathogen Source
Liang et al., 2006	Montana	*S. typhimurium*	70	Well	Coliforms detected; disinfection malfunction; cross-connection	Possibly poultry
Liang et al., 2006	Washington	*Campylobacter* spp. (spp.)	110	Well	Coliforms detected; untreated source; cross-connection	Unknown
Liang et al., 2006	Ohio	*C. jejuni, Shigella* spp.	57	Pond	Coliforms and *E. coli* detected; etiologic agents not detected; untreated source	Unknown
Liang et al., 2006	Wisconsin	*C. jejuni*	20	Well	Coliforms and *E. coli* detected; untreated source	Unknown
Liang et al., 2006	Illinois	Unidentified	180	Well	*E. coli* detected; chlorinated source	Unknown
Liang et al., 2006	Pennsylvania	Norovirus	70	Pond	Coliforms detected; cross-connection with untreated, unpotable water	Probably snow melt and/or septic system
Liang et al., 2006	New York	*C. jejuni, Entamoeba* spp., *Giardia* spp.	27	Well	Broken sewer line swamped the well head	Sewage
Liang et al., 2006	Ohio	*C. jejuni*, *C. lari*, *Cryptospordium* spp., and *Helicobacter canadensis*	82	Well	*Cryptosporidium* spp. detected; cross-connection with untreated, unpotable water	Unknown
Liang et al., 2006	Pennsylvania	Unidentified	174	Well	Coliforms detected; sewage pipe break noted; chlorination unit broken	Sewage
Blackburn et al., 2004	Arizona	*Naegleria fowleri* (meningoencephalitis)	2 (2 deaths)	Well	Coliforms and *N. fowleri* detected; untreated source	Unknown
Blackburn et al., 2004	Indiana	*Cryptosporidium* spp.	10	Well	Filtration system bypassed intentionally; well located in a high-density septic tank area	Possibly sewage
Blackburn et al., 2004	New York	*Giardia intestinalis*	6	Well/spring	Power outage resulted in negative pressure in the distribution system	Possibly sewage
Blackburn et al., 2004	Alaska	*C. jejuni*, *Yersinia enterocolitica*	12	Well	Coliforms detected; undisinfected source; documented contamination with surface water	Surface water

Reference	Location	Etiologic Agent	Number of Cases	Water Source	Water Quality/Environmental Information	Pathogen Source
Blackburn et al., 2004	Wisconsin	*C. jejuni*	13	Well	*C. jejuni* detected; untreated source; documented contamination with surface water; well located near a chicken coop	Possibly chicken feces
Blackburn et al., 2004	Arizona	Norovirus	71 (1 death)	Well	Unsanitary water dispensing and ice making documented	Unknown
Blackburn et al., 2004	Connecticut	Norovirus	142	Well	Coliforms and *E. coli* detected; heavy rains probably caused surface water infiltration; untreated source	Unknown
Blackburn et al., 2004	Kansas	Norovirus	86	Well	Coliforms detected; untreated source	Unknown
Blackburn et al., 2004; Anderson et al., 2003	Wyoming	Norovirus	230 (1 hospitalization)	Well	Coliforms and norovirus detected; wells located near septic tanks or outhouses; overloaded septic system documented	Sewage
Blackburn et al., 2004	Pennsylvania	Unknown	19	Well	No chlorine residual detected	Unknown
Lee et al., 2002	Florida	*G. intestinalis*	2	Well	Inadequate chlorine documented; pigs maintained near well	Possibly pig feces
Lee et al., 2002	Minnesota	*G. intestinalis*	12	Well	Coliforms detected; cross-connection with sewage pipe documented	Sewage
Lee et al., 2002	New Mexico	*G. intestinalis*	4	River	Inadequate or nonexistent filtration	Unknown
Lee et al., 2002	Colorado	*G. intestinalis*	27	River	*Giardia* detected; treatment malfunction documented	Unknown
Lee et al., 2002	Florida	*G. intestinalis*	2	Well	Coliforms detected; cross-connection with animal troughs; inadequate chlorine	Possibly animal feces
Lee et al., 2002	New Hampshire	*G. intestinalis*	5	Well	Filtration system malfunction; nonexistent disinfection	Unknown
Lee et al., 2002	Florida	*C. parvum*	5	Well	System break documented; low levels of chlorine residual	Unknown
Lee et al., 2002	Missouri	*S. typhimurium*	124 (17 hospitalizations)	Well	Inadequate chlorination	Unknown
Lee et al., 2002	New York	*C. jejuni*, *E. coli* O157:H7	781 (71 hospitalizations; 2 deaths)	Well	*E. coli* O157:H7 detected; nonexistent disinfection; heavy rains; cross-connection with septic system; possible cross-connection with manure storage	Probably sewage; possibly animal feces
Lee et al., 2002	Texas	*E. coli* O157:H7	22	Well	Inadequate chlorination	Unknown

Reference	Location	Etiologic Agent	Number of Cases	Water Source	Water Quality/Environmental Information	Pathogen Source
Lee et al., 2002	Idaho	*E. coli* O157:H7	4	Canal	Coliforms detected (100/100mL); untreated source	Possibly agricultural run-off
Lee et al., 2002	Idaho	*C. jejuni*	15	Well	Coliforms detected; potential surface water and agricultural runoff documented	Possibly agricultural runoff
Lee et al., 2002	Utah	*C. jejuni, E. coli* O157:H7, *E. coli* O111	102	Irrigation water	*C. jejuni* detected; nonpotable source	Unknown
Lee et al., 2002	California	*E. coli* O157:H7	5	Creek	Filtered source	Possibly human feces; possibly deer feces
Lee et al., 2002	Ohio	*E. coli* O157:H7	29 (9 hospitalizations)	Surface	Coliforms detected; possible cross-connection with animal-contaminated source documented	Possibly animal feces
Lee et al., 2002	Multistate	*Salmonella Bareilly*	84	Spring/well	Coliforms and *E. coli* detected; adequate treatment documented	Unknown
Lee et al., 2002	New Mexico	Small round-structured virus	70	Spring	Coliforms detected; proximity to latrine and septic system documented	Possibly sewage
Lee et al., 2002	West Virginia	Norwalk-like virus	123	Well	Coliforms detected; sewage contamination documented	Sewage
Lee et al., 2002	Kansas	Norwalk-like virus	86	Well	Coliforms detected; improper well construction documented	Unknown
Lee et al., 2002	California	Norwalk-like virus	147	Well	Coliforms detected; untreated source	Unknown
Lee et al., 2002	Florida	Unknown	4	Well	Coliforms detected; improper well construction documented; untreated source	Unknown
Lee et al., 2002	Florida	Unknown	3	Well	Coliforms detected; cross-connection with irrigation well documented	Unknown
Lee et al., 2002	Florida	Unknown	6	Surface	Cross-connection with irrigation well documented; irrigation well proximate to a commercial septic system and garbage container	Unknown
Lee et al., 2002	Florida	Unknown	3	Well	No chlorine residual detected; well proximate to a chicken coop	Possibly chicken feces
Lee et al., 2002	California	Unknown	31	Well	Coliforms detected; untreated source	Unknown
Lee et al., 2002	Washington	Unknown	46	Creek	Untreated source	Unknown
Lee et al., 2002	Washington	Unknown	68	Well	Coliforms detected; horse manure detected at the site	Unknown

Reference	Location	Etiologic Agent	Number of Cases	Water Source	Water Quality/Environmental Information	Pathogen Source
Lee et al., 2002	Florida	Unknown	71	Well	Coliforms detected; *Cryptosporidium* oocysts detected; well contamination with lake water documented	Unknown
Lee et al., 2002	Florida	Unknown	2	Well	Coliforms detected; heavy rainfall and unsanitary well conditions documented; untreated source	Unknown
Lee et al., 2002	California	Unknown	63	Irrigation	Coliforms detected; nonpotable source	Unknown

* Outbreaks that may have been related to animal contamination are shaded gray.

Table IV.3.1. Select Waterborne Disease Outbreaks Associated with Drinking Water*

Reference	Location	Etiologic Agent	Number of Cases	Water Source	Water Quality/Environmental Information	Pathogen Source
Alamanos et al., 2000	Greece	*Shigella sonnei*	288 (91 hospitalizations)	Well	*S. sonnei* detected with same resistance profile as clinical isolates; coliforms and fecal streptococci detected; source not regularly disinfected; no structural or functional well damaged noted; well situated near a milk factory	Unknown
Amvrosieva et al., 2001	Belarus	Echovirus 30 (aseptic meningitis; gastroenteritis)	460	River and well	Echovirus 30 and other enteroviruses detected in drinking water and source water;	Unknown
Amvrosieva et al., 2006	Belarus	Echovirus 30, echovirus 6, coxsackievirus B5 (meningitis; encephalitis; herpangina; myocarditis)	1,351	Surface reservoir	Etiologic agents detected in surface and finished water	Unknown
Atherton et al., 1995	England	*Cryptosporidium*	125	Reservoir	Oocysts detected; heavy rain; filter at treatment works had received maintenance	Unknown
Bell et al., 1995; Bowie et al., 1997; Isaac-Renton et al., 1998	Canada	*Toxoplasma gondii* (retinochoroiditis; toxoplasmosis; lymphadenopathy)	100 (plus 12 congenital cases)	Well/reservoir	*T. gondii* oocysts undetected; however, source water is unfiltered and chloraminated, which would not remove oocysts. Multiple epidemiological measures indicated the municipal water supply as the outbreak source; cats trapped near reservoir were seropositive; heavy rain	Probably feline feces (domestic/feral/wild)
Brown et al., 2001	Bermuda	Norwalk-like virus	448	Rainwater catchment	Coliforms, Norwalk-like virus, and *E. coli* detected; water supply deficiencies documented	Probably sewage
CCDR, 2000; Bruce-Grey-Owen Sound Health Unit, 2000	Canada	*E. coli* O157:H7, *Campylobacter* spp.	1,346 (65 hospitalizations, 6 deaths)	Well	Coliforms and *E. coli* O157:H7 detected; *Campylobacter* spp. and *E. coli* O157:H7 detected in animal manure of adjacent farms (cattle isolates identical to human isolates); heavy rain and flooding; chlorinated source	Probably cattle feces
CDC, 1996	Idaho, U.S.	*S. sonnei*	82	Well	Coliforms detected, but *S. sonnei* tests negative; heavy rain; poorly draining sewage documented	Possibly sewage
CDC, 1998a	Tajikistan	*S. typhii*	8,901 (95 deaths)	River/ground mix	4–400+ CFU/100 mL fecal coliforms detected at tap; major treatment failures; low pressure resulted in cross-connection with wastewater; inadequate chlorination; multiple distribution system failures	Unknown
Dworkin et al, 1996	Washington, U.S.	*C. parvum*	86	Wells	Coliforms and *C. parvum* presumptive oocysts detected; untreated source; treated wastewater from an irrigation system leaking into well documented; cattle grazing near well documented;	Treated wastewater
Glaberman et al., 2002	Northern Ireland	*C. parvum* (bovine genotype)	129	Municipal supply (otherwise unknown)	Unknown	Unknown

Reference	Location	Etiologic Agent	Number of Cases	Water Source	Water Quality/Environmental Information	Pathogen Source
Glaberman et al., 2002	Northern Ireland	*C. parvum* (human genotype)	117	Municipal supply (otherwise unknown)	Unknown	Sewage
Glaberman et al., 2002	Northern Ireland	*C. parvum* (human genotype)	230	Municipal supply (otherwise unknown)	*C. parvum* detected	Wastewater
Goldstein et al., 1996	Nevada, U.S.	*C. parvum*	78 (20 deaths in immuno-compromised patients with cryptosporidiosis)	River/lake	No coliforms or oocysts detected in source or finished water during study; presumptive oocysts detected intermittently after study; municipal supply receives filtration and disinfection; no malfunctions detected	Unknown
Hafliger et al., 2000	Switzerland	Norwalk-like virus	1,750+	Well	Coliforms, enteroviruses, and Norwalk-like viruses detected; identical genotypes of clinical and water samples documented; wastewater system defect documented	Sewage
Hanninen et al., 2003	Finland	*C. jejuni*	400	Well	Coliforms (1 CFU); *C. jejuni;* and *E. coli* (1 CFU) detected in well source and tap water; heavy precipitation	Surface water
Hanninen et al., 2003	Finland	*C. jejuni*	1,000	Well	Coliforms (1–66 CFU); *E. coli* (1–630 CFU); enterococci (3–1,080 CFU) and *C. jejuni* detected in well source and infiltrating surface water; *C. coli* found in porcine fecal samples; the variety of strains of *Campylobacter* spp. found and proximity to a farm indicated animal fecal contamination; heavy precipitation; undisinfected source	Possibly animal feces
Hayes et al., 1989	Georgia, U.S.	*Cryptospridium*	13,000	Surface	*Cryptosporidium* oocysts detected; oocysts detected in cattle feces; filtered and chlorinated source; suboptimal flocculation and filtration noted; sewage overflow documented; heavy precipitation	Possibly cattle feces or sewage
Howe et al., 2002	England	*C. parvum*	58	Spring/reservoir	*C. parvum* detected (0.1–0.9 oocysts/L); genotyping of oocysts suggested animal source; disinfected but unfiltered source; spring collection chambers in poor repair; cattle feces on top of spring covers documented; manure spread on field within 5 m of one well head; heavy rain	Probably cattle feces
Isaac-Renton et al., 1994	Canada	*G. duodenalis*	124	Creek	*G. duodenalis* cysts detected in water and beavers; beaver activity noted near water intake; beaver, water, and case cysts of the same karotype; unfiltered source	Beaver feces
Istre et al., 1984	Colorado, U.S.	*G. lamblia (intestinalis)*	20	Creek	*G. lamblia* cysts detected; treatment malfunction resulted in inadequate chlorination; other treatment deficiencies documented; two beaver dams proximate to water intake; heavy snowfall	Possibly beaver feces
Kent et al., 1988	Massachusetts, U.S.	*G. lamblia (intestinalis)*	703	Surface reservoir	Coliforms detected (1–41 CFU/100 mL); *G. lamblia* cysts detected (7–80/100 gal); disinfected but unfiltered source; malfunction in chlorinator documented; beaver activity documented; *G. lamblia* cysts detected in beaver feces	Possibly beaver feces
Kukkula et al., 1999	Finland	Norwalk-like virus	1,700–3 ,000 (estimated)	Lake	Coliforms (38–48 CFU/100mL) in tap water detected; Norwalk-like virus detected; filtered and disinfected source; inadequate chlorination documented	Unknown

Reference	Location	Etiologic Agent	Number of Cases	Water Source	Water Quality/Environmental Information	Pathogen Source
Kuusi et al, 2004	Finland	*C. jejuni*	463 (9 of 113 cases questioned were hospitalized)	Wells/reservoirs	Coliforms and *C. jejuni* detected; untreated source; heavy precipitation and infiltration of surface water into wells noted; wells and reservoirs accessible to people and animals	Unknown
Licence et al., 2001	Scotland	*E. coli* O157:H7	6	Spring	Coliforms, *E. coli* and *E. coli* O157:H7 detected in water and sheep feces; untreated source; sheep and deer grazing near source; human, water, and sheep samples indistinguishable	Sheep feces
MacKenzie et al., 1995	Wisconsin, U.S.	*C. parvum*	403,000 (estimated)	Lake	*C. parvum* oocysts detected (0.7–13.2 oocysts/100 L); filtered and disinfected source; increased turbidity; heavy precipitation/runoff	Possibly cattle feces; possibly sewage
Maurer and Sturcheler, 2000	Switzerland	*C. jejuni, S sonnei*, enteropathogenic *E. coli*, small round structured viruses	1,607–2,213 (estimated)	Well	Enteroviruses, small round structured virus detected; sewage pump failure documented	Sewage
Melby et al., 1991	Norway	*C. jejuni*	680	Lake/river	Coliforms and *C. jejuni* detected (different serotype from human isolates); untreated source; sheep grazing nearby noted; sheep infected with *campylobacter* noted	Possibly sheep feces
Navin et al., 1985	Nevada, U.S.	*G. lamblia (intestinalis)*	324	Wells/river/reservoir	*Giardia* cysts detected in source and treated water (10 cysts/3,800L) supply and a beaver in the reservoir water distribution site; source water disinfected but not filtered	Possibly beaver feces
Nygard et al., 2003	Sweden	Norovirus	200	Well	Norovirus detected; untreated source; sewage overflow documented	Probably sewage
O'Reilly et al, 2007; Fong et al., 2007	Ohio, USA	*C. jejuni*, norovirus, *G intestinalis*, *S. typhimurium*	1,450 (21 hospitalizations)	Well/lake	Coliforms (0.1–90 CRI/100 mL); *E. coli* (0.1–4.0 CFU/100 mL); enterococci (0.1–6.6 CFU/100 mL); *Arcobacter* spp.; coliphages; enteric viruses; *C. jejuni; Salmonella* spp., adenovirus, *Cryptosporidium* spp., and *Giardia* spp. detected (all well water samples); heavy rain; irregular sewage disposal noted; geology noted to be prone to contaminate aquifer; possible cross-connection with Lake Erie	Probably sewage
Olsen et al., 2002	Wyoming, USA	*E. coli* O157:H7	157 (4 hemolytic uremic syndrome cases)	Spring	Coliforms detected; untreated source; surface water contamination documented; risk of animal contamination noted; heavy rain and runoff; deer and elk feces near spring documented (but negative for *E. coli* O157:H7)	Possibly deer and elk feces
Parshionikar et al., 2003; Gelting et al., 2005; Anderson et al., 2003	Wyoming, USA	Norovirus	84	Well	Coliforms and norovirus detected; overloaded wastewater disposal system noted; heavy precipitation; chlorinator malfunction	Sewage
Ramakrishna et al., 1996	India	*V. cholerae* O139	475	Wells/surface reservoirs	Coliforms (10–1,800 MPN/L) and *V. cholera*e O139 detected; chlorination had been discontinued	Unknown

Reference	Location	Etiologic Agent	Number of Cases	Water Source	Water Quality/Environmental Information	Pathogen Source
Stirling et al., 2001	Canada	*C. parvum*	1,907 (50 hospitalizations)	River	*C. parvum* oocysts detected (no coliforms detected); disinfected and filtered source water; treatment deficiency documented which resulted in increased turbidity; chlorine residual levels normal	Unknown
Swerdlow et al., 1992	Peru	*V. cholerae* O1	16,400 (6.673 hospitalizations; 71 deaths)	Wells	Coliforms (1–1,800/100 mL), *V. cholorae* O1 detected; untreated source; low water pressure and poor water line integrity noted	Unknown

* Outbreaks that may have been related to animal contamination are shaded gray.

IV.4 Descriptions of Drinking Water Outbreaks with Animal Related-Pathogen Sources

Information in the two outbreak summary tables (Tables IV.2.1 and IV.3.1) indicates that the pathogen source in the majority of drinking water-related outbreaks remains unknown. The majority of outbreaks in both tables occurred in groundwater sources, and often, surface water contamination of the spring or well is the extent of the implication. Although the source of pathogens in surface water could be humans or animals, surface water that is subject to agricultural runoff would have a closer link to animal pathogens. However, most reports provide little detail regarding pathogen source, leaving a critical information gap for the purposes of this paper.

In this review of drinking water outbreaks, several studies have linked pathogens isolated from patients with water samples, animals, or both (e.g., CCDR 2000; Howe et al., 2002; Licence et al., 2001). The suspected animal sources include beavers, cats/cougars, deer, elk, pigs, cattle, and chickens/poultry. The animal-related pathogens in these outbreaks were *Giardia intestinalis*, *Cryptosporidium* spp., *E. coli* O157:H7, *Campylobacter* spp., *Toxoplasma gondii*, and *S. typhimurium.* In addition, Krewski and colleagues (2002) describe waterborne disease outbreaks in British Columbia from 1980 to 2000. Of the 24 outbreaks in the records, the suspected source of the pathogen (mostly *Giardia* and *Campylobacter*) was an animal in 21 outbreaks; however, the largest of those outbreaks was caused by *Cryptosporidium* of human origin. A summary of 288 Canadian drinking water outbreaks reported between 1974 and 2001 linked 44 (or 15 percent) with animal sources (Schuster et al., 2005); about half of the 25 outbreaks linked to private water supplies in England and Wales listed animals as possible contributing factors (Said et al., 2003).

Furthermore, in some studies, investigators hypothesize about the contamination based on circumstantial evidence. For example, Lee et al. (2002) described a *Giardia* outbreak where pigs were housed near a well while Olsen et al. (2002) noted deer and elk feces near a spring that was the source of water causing an *E. coli* O157:H7 outbreak in Wyoming. As laboratory methods have become increasingly sophisticated, more investigators are using molecular techniques to track pathogen sources. Most investigating agencies do not have the resources to perform such in-depth testing. As these techniques become more common and affordable, however, investigators will likely use them more often to shed light on outbreak causes. Three studies are summarized below to illustrate how advanced laboratory methods can link outbreaks with animal sources.

- An outbreak of *E. coli* O157 resulted from drinking from an untreated, private supply sourced from a spring located in an area where deer and sheep grazed (Licence et al., 2001). Water samples taken one week before the first illness had elevated coliforms (11 CFU/100 mL) and *E. coli* (15 CFU/100 mL) showing fecal contamination. After the outbreak, *E. coli* O157 isolates collected from patient stool, water, and sheep feces were all identical based on pulsed field gel electrophoresis analysis. Interestingly, the six cases were tourists to the area, while the permanent residents who were also exposed to the contaminated water did not experience any GI symptoms. The authors concluded that low levels of contamination may have provided some level of immunity.

- Researchers also used pulsed field gel electrophoresis to link *Giardia* isolates from water, beavers, and stool samples from cases from an outbreak in British Columbia (Isaac-Renton et al., 1994). A creek used as the drinking water source was chlorinated, but not filtered. The source water was found to be heavily contaminated with *Giardia* cysts, and an infected beaver was living near the water intake. When the animal was removed, water samples continually tested negative for *Giardia* cysts.
- A well documented and highly publicized outbreak that was linked to an animal source occurred in Walkerton, Ontario from May to June, 2000. In that municipal water system outbreak, *E. coli* O157:H7 and *Campylobacter* spp. were implicated in 1,346 reported cases of gastroenteritis, including 65 hospitalizations, 27 cases of hemolytic uremic syndrome, and six deaths (CCDR, 2000). One particular well was implicated as the source of contamination based on microbiological testing and a history of susceptibility to surface water influence. In addition, hydrological modeling indicated that the well could have been contaminated by an adjacent farm—especially under the record-breaking rain conditions present before the outbreak. Subsequent microbiological testing supported this model. The *Investigative Report of the Walkerton Outbreak of Waterborne Gastroenteritis* (Bruce-Grey-Owen Sound Health Unit, 2000) reported that "The molecular subtyping and phage-typing of the *E. coli* O157:H7 and the *Campylobacter* spp. isolates from this farm were identical to those found in the majority of the human cases. While investigators could not prove the pathogens were present prior to the outbreak, the evidence suggested the pathogens likely originated from cattle manure on this farm." Other testing of deer feces was negative for *E. coli*.

Finally, in another outbreak in British Columbia, *T. gondii* was implicated (Bowie et al., 1997). The cysts were never detected in the water source; however, the authors concluded that the strength of the epidemiological and environmental data was indisputable. Although rarely implicated as a waterborne pathogen, *T. gondii* can have devastating sequelae in fetuses whose mothers are infected—especially in the first part of pregnancy. Toxoplasmosis can also be fatal in immunocompromised people. Felines are the definitive hosts in the *Toxoplasma* life cycle, and the cysts they excrete are extremely hardy and long-lived in the environment. In this outbreak, cats and cougars were implicated as the source of the pathogen.

IV.5 Summary of Selected Recreational Water Outbreaks

The CDC tracks waterborne disease and outbreaks from drinking water and recreational water, with the latter including recreational water outbreaks since 1978. CDC's analysis of recreational water includes swimming pools, wading pools, spas, waterslides, interactive fountains, wet decks, and fresh and marine bodies of water; it does not include recreational waters associated with cruise ships.

A summary by Craun et al. (2005) that analyzed the recreational water outbreaks in the United States between 1970 and 2000 illustrates the difficulties associated with addressing all the data gaps in an outbreak investigation. During that period, CDC received reports of 259 outbreaks associated with recreational water. Over two-thirds were associated with either bacteria or protozoa; over 15 percent had unidentified etiologies and only 7 percent were viral. However, CDC estimates that approximately 80 percent of the annual illnesses overall in the United States

due to known pathogens are of viral origin (Mead et al., 1999). The trend in the number of outbreaks that are reported annually has increased dramatically since 1990, probably due to a combination of better reporting practices and the occurrence of more outbreaks.

Of the 259 recreational water outbreaks reported, only half included any information about possible sources of the contamination or contributing factors. In untreated recreational water outbreaks, feces or ill bathers in the water, bather overcrowding, and the presence of children in diapers accounted for 90 percent of the assumed sources; however, Craun et al. (2005) did not indicate that these attributions were anything more than assumptions and were not reinforced by laboratory analysis, other than measures of microbial indicators. Further, most of the related assumptions were based on eyewitness reports such as adults seen rinsing diapers in the water and the measure of bather density was subjective. Craun et al. estimated that 18 percent of the outbreaks were associated with animals and 21 percent were associated with sewage contamination—although the proportion actually confirmed by laboratory analysis is unknown. Some animal-associated outbreaks may have been classified based on the etiological agent itself. For example, because the source of *Leptospira* is animal urine and leptospirosis is not spread from person-to-person, any waterborne outbreaks of leptospirosis can be assumed to have an animal source (CDC, 2005). Although most cases of leptospirosis occur in Hawaii, a large outbreak in 1998 of triathletes in Illinois and Wisconsin caused 375 illnesses and 28 hospitalizations (CDC, 1998b).

A summary of the CDC's surveillance reports since 2000 is provided below:

- During 2001 to 2002 (Yoder et al., 2004), the largest number of recreational water outbreaks occurred since reporting began in 1978. In total, 65 outbreaks were reported, with 30 involving gastroenteritis. Fifty percent of the outbreaks involving gastroenteritis were associated with *Cryptosporidium* in treated drinking water, followed by *E. coli* (25 percent) and then norovirus (25 percent) in freshwater.
- Results from 2003 to 2004 indicate that 26 states and Guam reported a total 62 outbreaks associated with recreational water (excluding *Vibrio* cases) (Dziuban et al., 2006). Of the 62 cases, 20 were confirmed as bacterial, 15 as parasitic, and 6 as viral. For the remaining cases, the etiologic agents were chemical- or toxin-mediated or were not identified. Primary illnesses resulting from these outbreaks included gastroenteritis, dermatitis, and acute respiratory illness. Other illnesses included amebic meningoencephalitis, meningitis, leptospirosis, otitis externa, and mixed illnesses. In addition, 16 states reported a total of 142 *Vibrio* cases associated with recreational water. Data for the *Vibrio* cases were analyzed separately to avoid substantially altering total waterborne disease outbreak numbers when compared with previous reports. Of the *Vibrio* cases, 70 resulted in hospitalization and 9 patients died. The most frequently isolated *Vibrio* species was *V. vulnificus*; others included *V. parahaemolyticus*, *V. cholerae*, *V. damsela*, *V. fluvialis*, nonspeciated *Vibrio*, and mixed *Vibrio*. Nearly all *Vibrio* cases were exposed to recreational water in a coastal state.

CDC data on recreational water-related disease outbreaks from the last three reports covering 1999 to 2004 (Dziuban et al., 2006; Lee et al., 2002; Yoder et al., 2004) are summarized in Table IV.5.1. Individual studies identified in the scientific literature also are included in the

Table IV.5.1. Select Waterborne Disease Outbreaks Associated with Recreational Water in the United States Reported by the CDC (1999 to 2004)*

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Wheeler et al., 2007[2]	California	2004	*Cryptosporidium*	Acute gastrointestinal illness (AGI)	315	Water park	*Cryptosporidium* oocysts detected in backwash and on sand filters; *Cryptosporidium* or *Giardia* not detected from the county park lake and three wells; fecal coliform not detected in well-water	Unknown
Dziuban et al., 2006[3]	California	2004	*Cryptosporidium*	AGI	336	Pool	*Cryptosporidium* oocysts detected in backwash; *Cryptosporidium* or *Giardia* not detected from the county park lake and three wells; fecal coliform not detected in well-water	Unknown
Dziuban et al., 2006	Colorado	2004	*Cryptosporidium*	AGI	6	Pool	Unknown	Unknown
Dziuban et al., 2006	Florida	2004	Norovirus	AGI	42	Waterslide	Unknown	Child with diarrhea
Dziuban et al., 2006	Georgia	2004	*Cryptosporidium*	AGI	14	Pool	Unknown	Unknown
Dziuban et al., 2006	Guam	2004	*Leptospira* spp.	Leptospirosis	3	River	Unknown	Unknown
Dziuban et al., 2006	Idaho	2004	Norovirus	AGI	140	Pool	Unknown	Unknown
Dziuban et al., 2006	Illinois	2004	*P. aeruginosa*	Skin, acute respiratory infection (ARI)	16	Pool, spa	Unknown	Unknown
Dziuban et al., 2006	Illinois	2004	*P. aeruginosa*	Skin	5	Spa	Unknown	Unknown

[2] Also referenced in Dziuban et al. (2006)
[3] See Wheeler et al. (2007) for additional information

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Dziuban et al., 2006	Illinois	2004	*Cryptosporidium*	AGI	37	Pool, wading pool	Unknown	Unknown
Dziuban et al., 2006	Illinois	2004	*Cryptosporidium*	AGI	8	Pool	Unknown	Unknown
Dziuban et al., 2006	Minnesota	2004	Norovirus	AGI	9	Lake	Unknown	Unknown
Dziuban et al., 2006	Missouri	2004	*Giardia intestinalis*	AGI	9	Lake	Unknown	Unknown
Dziuban et al., 2006	North Carolina	2004	*P. aeruginosa*	Skin	41	Spa	Unknown	Unknown
Dziuban et al., 2006	Ohio	2004	*C. hominis*	AGI	160	Pool, wading pool, interactive fountain	Although both *Cryptosporidium* oocysts and *Giardia* cysts were identified in the pool water, only *Cryptosporidium* oocysts were isolated from clinical specimens.	Unknown
Dziuban et al., 2006	Ohio	2004	*P. aeruginosa*	Ear, skin	119	Pool, spa	*Pseudomonas* detected	High bather load
Dziuban et al., 2006	Oregon	2004	*P. aeruginosa*	Skin	2	Spa	Unknown	Unknown
Dziuban et al., 2006[4]	Oregon	2004	Norovirus	AGI	39	Lake	Unknown	Unknown
Dziuban et al., 2006	Vermont	2004	Norovirus	AGI	70	Pool	Unknown	Unknown
Dziuban et al., 2006	Wisconsin	2004	*P. aeruginosa*	Skin, AGI	22	Pool, spa	Unknown	Unknown
Dziuban et al., 2006	Wisconsin	2004	*Cryptosporidium*	AGI	6	Pool	Unknown	Unknown
Dziuban et al., 2006	Arkansas	2003	*Cryptosporidium*	AGI	4	Pool	Unknown	Unknown
Dziuban et al., 2006	Connecticut	2003	Echovirus 9	Neurological	36	Pool	Unknown	High bather load

[4] See Keen et al. (1994) for additional information

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Dziuban et al., 2006	Connecticut	2003	Microbe-resistant *Staphylococcus* infection	Skin	10	Spa	Unknown	Unknown
Dziuban et al., 2006	Georgia	2003	*S. sonnei*	AGI	13	Lake	Fecal coliform detected	Bathers
Dziuban et al., 2006	Idaho	2003	*Cryptosporidium*	AGI	4	Lake	Unknown	Unknown
Dziuban et al., 2006	Illinois	2003	*P. aeruginosa*	Skin	52	Spa	*Pseudomonas* detected	Potted plant
Dziuban et al., 2006	Iowa	2003	*Cryptosporidium; G. intestinalis*	AGI	63	Wading pool	Unknown	Unknown
Dziuban et al., 2006	Kansas	2003	*C. hominis*	AGI	617	Pools, wading pools	Unknown	Likely children who continued to swim while ill with diarrhea
Dziuban et al., 2006	Maryland	2003	*S. sonnei*; *P. shigelloides*	AGI	65	Lake	Fecal coliform and *E. coli* detected	Diapers, dumping waste
Dziuban et al., 2006[5]	Mass.	2003	*G. intestinalis*	AGI	149	Pool	Not conducted	Unknown
Dziuban et al., 2006	North Carolina	2003	*N. fowleri*	Neuro	1	Lake	Unknown	Unknown
Dziuban et al., 2006	Ohio	2003	*P. aeruginosa*	Skin	17	Pool, spa	Unknown	Unknown
Dziuban et al., 2006	Ohio	2003	*P. shigelloides*	AGI	3	Lake	Unknown	Unknown
Dziuban et al., 2006	Oregon	2003	*S. sonnei*	AGI	56	Interactive fountain	Fecal coliform and *E. coli* detected	Unknown
Dziuban et al., 2006	Wyoming	2003	*P. shigelloides*	AGI	2	Reservoir	Unknown	Unknown
Katz et al., 2006[6]	Mass.	2003	*G. intestinalis*	AGI	149	Pool	Not conducted	Childrens' pool

[5] See Katz et al. (2006) for additional information
[6] Also referenced by Dziuban et al. (2006)

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Iwamoto et al., 2005[7]	Georgia,	2003	*S. sonnei*	Gastroenteritis	17	Lake	Fecal coliform detected (160 fecal coliform bacteria per 100 mL); *Shigella* not detected	Fecal contamination of lake water by an infected swimmer
Yoder et al., 2004	Alaska	2002	*P. aeruginosa*	Skin	110	Pool/spa	*P. aeruginosa* detected	Unknown
Yoder et al., 2004	Alaska	2002	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	3	Pool/spa	Unknown	Unknown
Yoder et al., 2004	Colorado	2002	*P. aeruginosa*	Skin	12	Pool/spa	*P. aeruginosa* detected	Unknown
Yoder et al., 2004	Florida	2002	*N. fowleri*	Meningo-encephalitis	1	Lake	Unknown	Unknown
Yoder et al., 2004	Florida	2002	*N. fowleri*	Meningo-encephalitis	1	Lake	Unknown	Unknown
Yoder et al., 2004	Georgia	2002	*Cryptosporidium* spp.	Gastroenteritis	3	Wading pool	Unknown	Fecal accident
Yoder et al., 2004	Georgia	2002	*N. fowleri*	Meningo-encephalitis	1	River	Unknown	Unknown
Yoder et al., 2004	Iowa	2002	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	24	Pool/spa	Unknown	Unknown
Yoder et al., 2004	Maine	2002	*E. coli* O157:H7	Gastroenteritis	9	Wading pool	Unknown	Unknown
Yoder et al., 2004	Maryland	2002	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	3	Spa	Unknown	Unknown
Yoder et al., 2004	Mass.	2002	*Cryptosporidium* spp.	Gastroenteritis	767	Pool	Unknown	Unknown

[7] Also referenced in Dziuban et al. (2006)

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Yoder et al., 2004	Minnesota	2002	Norovirus	Gastroenteritis	11	Lake	Unknown	Unknown
Yoder et al., 2004	Minnesota	2002	Norovirus	Gastroenteritis	36	Pool	Unknown	Unknown
Yoder et al., 2004	Minnesota	2002	*Cryptosporidium* spp.	Gastroenteritis	52	Indoor pool	Unknown	Unknown
Yoder et al., 2004	Minnesota	2002	*Cryptosporidium* spp.	Gastroenteritis	41	Pool	Unknown	Unknown
Yoder et al., 2004	Minnesota	2002	*Cryptosporidium* spp.	Gastroenteritis	16	Pool	Unknown	Unknown
Yoder et al., 2004	Ohio	2002	*P. aeruginosa*	Skin	18	Spa	*P. aeruginosa* detected	Unknown
Yoder et al., 2004	Ohio	2002	*P. aeruginosa*	Skin	31	Pool/spa	*P. aeruginosa* detected	Unknown
Yoder et al., 2004	Oregon	2002	*Avian schistosomes* (suspected etiology based on clinical syndrome and setting)	Skin	19	Lake	Unknown	Unknown
Yoder et al., 2004	Texas	2002	*Cryptosporidium hominis*	Gastroenteritis	54	Wading pool	The species of *Cryptosporidium* that infects humans and monkeys	Diaper-aged children
Yoder et al., 2004	Wisconsin	2002	Norovirus	Gastroenteritis	15	Pool	Unknown	Unknown
Yoder et al., 2004	Wisconsin	2002	Norovirus	Gastroenteritis	44	Lake	*E. coli* detected	Bathers or dumping sewage from boats
Yoder et al., 2004	Wyoming	2002	*Cryptosporidium* spp.	Gastroenteritis	3	Lake	Unknown	Unknown
Yoder et al., 2004	Wyoming	2002	*G. intestinalis*	Gastroenteritis	2	River	Unknown	Human or animal contamination
Causer et al., 2005[8]	Illinois	2001	*Cryptosporidium*	Gastroenteritis	358	Pool	*Cryptosporidium* oocysts detected	Fecal accident by park visitor

[8] Also referenced in Yoder et al. (2004)

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Yoder et al., 2004	Colorado	2001	*S. sonnei*	Gastroenteritis	33	Interactive fountain	Unknown	Unknown
Yoder et al., 2004	Florida	2001	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	34	Pool	Unknown	Unknown
Yoder et al., 2004	Florida	2001	*P. aeruginosa*	Skin	53	Spa	*P. aeruginosa* detected	Unknown
Yoder et al., 2004	Florida	2001	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	7	Spa	Unknown	Unknown
Yoder et al., 2004[9]	Illinois	2001	*Cryptosporidium hominis*	Gastroenteritis	358	Pool	*Cryptosporidium* oocysts detected; the species of *Cryptosporidium* that infects humans and monkeys	Fecal accident by park visitor
Yoder et al., 2004	Iowa	2001	*S. sonnei*	Gastroenteritis	45	Wading pool	Unknown	Unknown
Yoder et al., 2004	Maine	2001	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	21	Spa	Unknown	Unknown
Yoder et al., 2004	Maryland	2001	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	8	Spa	Unknown	Unknown
Yoder et al., 2004	Minnesota	2001	*E. coli* O157:H7	Gastroenteritis	20	Lake	Fecal coliform detected	Geese
Yoder et al., 2004	Minnesota	2001	Norovirus	Gastroenteritis	40	Lake	Unknown	Unknown

[9] See Causer et al. (2005) for additional information

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Yoder et al., 2004	Minnesota	2001	*E. coli* O26:NM	Gastroenteritis	4	Lake	Unknown	Unknown
Yoder et al., 2004	Minnesota	2001	*P. aeruginosa*	Skin	6	Spa	*P. aeruginosa* detected	Unknown
Yoder et al., 2004	Nebraska	2001	*Cryptosporidium* spp.	Gastroenteritis	157	Pools	Unknown	Unknown
Yoder et al., 2004	Nebraska	2001	*Cryptosporidium* spp.	Gastroenteritis	21	Pool	Unknown	Unknown
Yoder et al., 2004	Nebraska	2001	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	9	Pool/spa	Unknown	Unknown
Yoder et al., 2004, 2004	Oklahoma	2001	*N. fowleri*	Meningo-encephalitis	1	Lake	Unknown	Unknown
Yoder et al., 2004	Penn.	2001	*P. aeruginosa*	Skin	2	Spa	*P. aeruginosa* detected	Unknown
Yoder et al., 2004	Penn.	2001	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	42	Spa	Unknown	Unknown
Yoder et al., 2004	Penn.	2001	*Bacillus* spp.	Skin	20	Spa	*Bacillus* detected	Unknown
Yoder et al., 2004	Penn.	2001	*Staphylococcus* spp. (suspected etiology based on clinical syndrome and setting)	Skin	3	Spa	Unknown	Unknown
Yoder et al., 2004	South Carolina	2001	*E. coli* O157:H7	Gastroenteritis	45	Lake	Fecal coliforms detected	Fecal contamination
Yoder et al., 2004, 2004	Texas	2001	*N. fowleri*	Meningo-encephalitis	1	Lake	Unknown	Unknown
Yoder et al., 2004, 2004	Texas	2001	*N. fowleri*	Meningo-encephalitis	1	Lake	Unknown	Unknown

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Yoder et al., 2004, 2004	Texas	2001	*N. fowleri*	Meningo-encephalitis	1	Lake	Unknown	Unknown
Yoder et al., 2004, 2004	Texas	2001	*N. fowleri*	Meningo-encephalitis	1	Lake	Unknown	Unknown
Yoder et al., 2004	Washington	2001	*P. aeruginosa*	Skin	3	Spa	*P. aeruginosa* detected	Unknown
Yoder et al., 2004	Wisconsin	2001	*P. aeruginosa* (suspected etiology based on clinical syndrome and setting)	Skin	7	Spa	Unknown	Unknown
Yoder et al., 2004	Wyoming	2001	*Cryptosporidium* spp.	Gastroenteritis	2	Flow-through pool/hot spring	Unknown	Unknown
Mathieu et al., 2004[10]	Ohio	2000	*C. parvum*	Gastroenteritis	749 suspected cases; 144 laboratory confirmed	Pool	*C. parvum* (both the human and bovine genotypes) detected in water and sand filter samples	Fecal accidents
Lee et al., 2002	Alaska	2000	*P. aeruginosa*	Skin	29	Pool/hot tub	*P. aeruginosa* detected	High bather load
Lee et al., 2002	Arkansas	2000	*P. aeruginosa*	Skin	26	Pool/hot tub	Unknown	Unknown
Lee et al., 2002	California	2000	*N. fowleri*	Meningo-encephalitis	1	Mudhole	Unknown	Unknown
Lee et al., 2002	California	2000	*Schistosomes* (suspected etiology based on clinical syndrome and setting)	Skin	6	Pond	Unknown	Unknown

[10] Also referenced in Lee et al. (2002)

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Lee et al., 2002	California	2000	*Schistosomes* (suspected etiology based on clinical syndrome and setting)	Skin	4	Pond	Unknown	Unknown
Lee et al., 2002	Colorado	2000	*C. parvum*	Gastroenteritis	112	Pool	Unknown	Unknown
Lee et al., 2002	Florida	2000	*C. parvum*	Gastroenteritis	3	Pool	Unknown	Fecal material
Lee et al., 2002	Florida	2000	*C. parvum*	Gastroenteritis	5	Pool	Unknown	Ill child
Lee et al., 2002	Florida	2000	*C. parvum*	Gastroenteritis	19	Pool	Unknown	Unknown
Lee et al., 2002	Florida	2000	*C. parvum*	Gastroenteritis	5	Pool	Unknown	Fecal accidents by ill child
Lee et al., 2002	Florida	2000	*N. fowleri*	Meningo-encephalitis	1	Unknown	Unknown	Unknown
Lee et al., 2002	Florida	2000	*P. aeruginosa* (suspected etiology based on clinical syndrome)	Skin	6	Hot tub	Unknown	Unknown
Lee et al., 2002	Georgia	2000	*C. parvum*	Gastroenteritis	36	Pool	Unknown	Unknown
Lee et al., 2002	Guam	2000	*Leptospira interrogans*	Leptospirosis	21	Lake	Unknown	Unknown
Lee et al., 2002	Maine	2000	*P. aeruginosa*	Skin	9	Hot tub/pool	Unknown	Unknown
Lee et al., 2002	Maine	2000	*P. aeruginosa*	Skin	11	Hot tub	*P. aeruginosa* detected	Unknown
Lee et al., 2002	Minnesota	2000	*C. parvum*	Gastroenteritis	220	Lake	Unknown	Washing babies while changing diapers
Lee et al., 2002	Minnesota	2000	*S. sonnei*	Gastroenteritis	15	Lake/pond	Unknown	Unknown
Lee et al., 2002	Minnesota	2000	*C. parvum*	Gastroenteritis	7	Pool	Unknown	Unknown

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Lee et al., 2002	Minnesota	2000	*C. parvum*	Gastroenteritis	6	Pool	*C. parvum* not detected	Unknown
Lee et al., 2002	Minnesota	2000	*S. sonnei*	Gastroenteritis	25	Lake	*Shigella* not detected	Fecal accidents
Lee et al., 2002	Minnesota	2000	*C. parvum*	Gastroenteritis	4	Pool	Unknown	Unknown
Lee et al., 2002	Minnesota	2000	*P. aeruginosa*	Skin	16	Hot tub	Unknown	Unknown
Lee et al., 2002	Missouri	2000	*Shigella flexneri*	Gastroenteritis	6	Wading pool	Unknown	Unknown
Lee et al., 2002	Nebraska	2000	*C. parvum*	Gastroenteritis	225	Pools	Unknown	Fecal accidents
Lee et al., 2002[11]	Ohio	2000	*C. parvum*	Gastroenteritis	700	Pool	Unknown	High bather load; multiple fecal accidents
Lee et al., 2002	South Carolina	2000	*C. parvum*	Gastroenteritis	26	Pool	Coliforms not detected	Unknown
Lee et al., 2002	Texas	2000	*N. fowleri*	Meningo-encephalitis	1	Lake	Unknown	Unknown
Lee et al., 2002	Washington	2000	*P. aeruginosa* (suspected etiology based on clinical syndrome)	Skin	10	Pool/hot tub	Unknown	Unknown
Lee et al., 2002	Wisconsin	2000	Norwalk-like virus	Gastroenteritis	9	Pool	Unknown	Unknown
Bruce et al., 2003[12]	Washington	1999	*E. coli* O157:H7	Gastroenteritis, 3 cases of hemolytic uremic syndrome	37	Lake	*E. coli* O157:H7 detected	Possible fecal accident.; no agricultural sources identified
Lee et al., 2002	Arkansas	1999	*P. aeruginosa*	Skin	10	Pool	*P. aeruginosa* detected	Unknown
Lee et al., 2002	Colorado	1999	*P. aeruginosa*	Skin	19	Hot tub	*P. aeruginosa* detected	Unknown

[11] See Mathieu et al. (2004) for additional information
[12] Also referenced in Lee et al. (2002)

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Lee et al., 2002	Colorado	1999	*P. aeruginosa* (suspected etiology based on clinical syndrome)	Skin	5	Hot tub	Unknown	Unknown
Lee et al., 2002[13]	Connecticut	1999	*E. coli* O121:H19	Gastroenteritis	11	Lake	*E. coli* and shiga toxins not detected in lake; total and fecal coliforms detected (results differ slightly from that stated in McCarthy paper)	Toddler with severe diarrhea
Lee et al., 2002	Florida	1999	*Campylobacter jejuni*	Gastroenteritis	6	Pool	Unknown	Unknown
Lee et al., 2002	Florida	1999	*S. sonnei*, *C. parvum*	Gastroenteritis	38	Interactive fountain	Coliforms detected; fecal coliforms not detected	Unknown
Lee et al., 2002	Florida	1999	*C. parvum*	Gastroenteritis	6	Pool	Unknown	Unknown
Lee et al., 2002	Florida	1999	*E. coli* O157:H7	Gastroenteritis	2	Ditch water	Unknown	Unknown
Lee et al., 2002	Florida	1999	*N. fowleri*	Meningo-encephalitis	1	Pond	Unknown	Unknown
Lee et al., 2002	Idaho	1999	Norwalk-like virus	Gastroenteritis	25	Hot springs	Unknown	Unknown
Lee et al., 2002	Mass.	1999	*G. intestinalis*	Gastroenteritis	18	Pond	Total coliforms detected	Unknown
Lee et al., 2002	Minnesota	1999	*C. parvum*	Gastroenteritis	10	Pool	Unknown	Unknown
Lee et al., 2002	Nebraska	1999	*E. coli* O157:H7	Gastroenteritis	7	Wading pool	Unknown	Unknown
Lee et al., 2002	New York	1999	Norwalk-like virus	Gastroenteritis	168	Lake	Unknown	Feces

[13] See McCarthy et al. (2001) for additional information

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness/Symptoms	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Lee et al., 2002	Oregon	1999	*Schistosomes* (suspected etiology based on clinical syndrome and setting)	Skin	2	Lake	Unknown	Unknown
Lee et al., 2002	Vermont	1999	*P. aeruginosa* (suspected etiology based on clinical syndrome)	Skin	9	Hot tub	Unknown	Unknown
Lee et al., 2002[14]	Washington	1999	*E. coli* O157:H7	Gastroenteritis	36	Lake	*E. coli* O157:H7 detected	Unknown
Lee et al., 2002	Wisconsin	1999	*C. parvum*	Gastroenteritis	10	Pool	Unknown	Unknown
Lee et al., 2002	Wisconsin	1999	*E. coli* O157:H7	Gastroenteritis	5	Lake/pond	Total and fecal coliform detected by did not exceed regulatory levels; one sample tested for *E. coli* O157:H7 was negative	Unknown
Samadpour et al., 2002[15]	Washington	1999	*E. coli* O157:H7	Gastroenteritis	36	Lake	*E. coli* O157:H7 detected in human and duck feces	Possible human feces, but duck feces could have helped to sustain contamination for a longer period of time
McCarthy et al., 2001[16]	Connecticut	1999	*E. coli* O121	Gastroenteritis; hemolytic uremic syndrome	11	Lake	*E. coli* not detected in lake water; *E. coli* detected in water from storm drain; Shiga toxin–producing strain not detected (results differ slightly from those reported in Lee et al., 2002)	Toddler with severe diarrhea

* Outbreaks that may have been related to animal contamination are shaded gray.

[14] See Bruce et al. (2003) and Samadpour et al. (2002) for additional information
[15] Also referenced in Lee et al. (2002)
[16] Also referenced in Lee et al. (2002)

table (see Appendix A). Outbreaks caused by *Legionella* spp. were not included in the table because they are not relevant to the analysis. Table IV.5.1 is sorted by outbreak year (descending order) followed by author and year, and then location (alphabetically). Outbreaks that may have been related to animal contamination are shaded gray.

IV.6 Summary of Selected Recreational Water Outbreaks Reported in the United States and Internationally

To augment the results reported by the CDC, a comprehensive literature search was conducted (see Appendix A for further detail). Studies for which the Abstracts from the literature identified were reviewed, and studies were included if the abstract showed evidence that the etiologic agent was detected in the source water were included in Table IV.6.1. As mentioned previously, this is a higher bar than what the CDC uses to evaluate outbreak evidence, but the large number of international articles with questionable outbreak investigation methods required a more stringent standard for inclusion. The articles are cross-referenced to the CDC table accordingly. The table is sorted by outbreak year (descending order) followed by study author (alphabetically) and location (alphabetically). Outbreaks that may have been related to animal contamination are shaded gray.

IV.7 Descriptions of Recreational Water Outbreaks with Animal Related-Pathogen Sources

Information from the two outbreak summary tables (Tables IV.5.1 and IV.6.1) indicates that the pathogen source in the majority of recreational water-related outbreaks remains unknown. All of the outbreak studies that cited an animal source were based in natural water sources that include lakes, streams, a swimming area with water fed by a brook, and a canal. The etiologic agents were *E. coli* spp. (n=5), *Schistosomes* spp. (n=2), and *Leptospira* spp. (n=1). *E. coli* was associated with cattle, deer, or duck feces; *Schistosomes* spp. was associated with snails; and *Leptospira* spp. was associated with rat urine. Five studies identified animal sources as part of their environmental investigation. Three additional studies cited animal-related pathogens as a potential source; however, environmental sampling was not conducted to support this claim. Summaries of these studies are provided below.

IV.7.1 Environmental Sampling Including Animal Sources

Cornwell, Southwest England (cattle feces)
In 2004, seven children were infected with *E. coli* O157:H7 after playing in a stream located in Cornwell, southwest England (Ihekweazu et al., 2006). Environmental samples from the stream and cattle grazing on the surrounding fields above the stream were analyzed; both the water and cattle feces were found to be positive for *E. coli* O157:H7. Stool samples from all seven cases were confirmed *E. coli* phage type 21/28, though none of the environmental isolates were phage type 21/28, including the cattle feces. Regardless, cattle feces, according to the study authors, remains the most likes source of stream contamination. Heavy rainfall occurring two days prior to the outbreak increased the likelihood that cattle feces were the potential source of contamination since the fecal matter could have been washed into the stream. Another potential source of contamination was sewage from overflow drains located around the stream.

Table IV.6.1. Select Waterborne Disease Outbreaks Associated with Recreational Water

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness	Number of Cases	Water Source	Water Quality Information	Pathogen Source
CCDR, 2005	British Columbia, Canada	2004	*E. coli* O157:H7	Gastroenteritis	10 (8 (confirmed; 2 probable)	Pool	Elevated fecal indicators detected (sample 1: 2,200 fecal coliforms/100 mL, 1500 *E. coli*/100 mL; sample 2: 16,000 fecal coliforms/100 mL, 14,400 *E. coli /100 mL); E. coli* O157:H7 not detected	Overloaded and blocked storm sewer
Ihekweazu et al., 2006	Cornwall, U.K.	2004	*E. coli* O157	Confirmed *E. coli* O157 phage type 21/28	7	Stream	*E. coli O157* detected in stream and in cattle feces in the catchment area of the stream; none of the environmental isolates were phage type 21/28	Cattle feces and/or sewage overflow
Verma et al., 2007	Trafford, U.K.	2004	*E. coli* O157	Gastroenteritis and hemolytic uremic syndrome (HUS)	8 GI and 2 HUS	Pool	*E. coli* O157 not detected; blockage to chlorine system noted	Unknown
Jones et al., 2006	Southwest England, U.K.	2003	*Cryptosporidium*	Gastroenteritis	63	Pool	Coliform detected (2,100 coliforms, 40 *E. coli* per cu mm)	Unknown
Louie et al., 2004	Surrey, Canada	2003	*C. parvum*	Crypto-sporidiosis	33	Pool	*C. parvum* detected	Fecal accidents
Enk et al., 2003	Minas Gerais, Brazil	2002	*Schistosoma mansoni*	Acute schistosomiasis	17	Pool (water provided by nearby brook)	*Schistosoma mansoni* detected in nearby snails	Snails
Hoebe et al., 2004	The Netherlands	2002	Norovirus	Gastroenteritis	90	Pool	Coliform bacteria detected (>1,000 organisms/mL); enterococci detected (3,500 organisms/100 mL); *E. coli* detected (7,700 organisms/100 mL); norovirus detected	Likely children in water fountain
Tate et al., 2003	U.K.	2002	*P. aeruginosa*	Folliculitis	35	Pool	*P. aeruginosa* not detected in water, but detected on inflatable toy	Infected bather
Bruneau et al., 2004	Montreal-Centre, Canada	2001	*E. coli* O157:H7	Gastroenteritis	4	Beach	6-162 fecal coliforms/100mL detected. Three strains of *E. coli* O157:H7 found	Fecal accidents (by children or adults)

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Hauri et al., 2005	Kassel, Germany	2001	Echovirus 30	Aseptic meningitis	215	Pond	An echovirus 30 sequence obtained from one pond water sample showed a 99% nucleotide and 100% amino-acid homology with patient isolates. Weekly testing for total coliforms, fecal coliforms, enterococci, *Staphylococcus aureus* never exceed European Union bathing guidelines	Likely human feces
Maunula et al., 2004	Helsinki, Finland	2001	Norovirus; astrovirus	Gastroenteritis	242	Pool	Fecal contamination not detected 2 weeks prior to outbreak; *E. coli* detected in outlet well 2 weeks after outbreak; norovirus and astrovirus detected. Norovirus detected in outlet well as much as 8 months after incident	Unknown
Perra et al., 2002	Rochefort, France	2001	*Leptospira* spp.	Leptospirosis	5 (3 confirmed and 2 possible)	Canal	*E. coli* detected (between 100 and 2,000/100 mL); 130 rats trapped; prevalence of seropositive rodents was 30.8%	Rodent urine
Feldman et al., 2002	California, U.S.	1999	*E. coli* O157:NM	Gastroenteritis	7 (3 confirmed and 4 possible)	Lake	Total and fecal coliform counts spiked 2 weeks prior to outbreak	Possibly from feces from humans, cattle, or deer
Lévesque et al., 2002	Quebec City, Canada	1999	*Schistosomes*	Cercarial dermatitis	53	Lake	Fecal coliforms and fecal streptococci detected but at very low levels; *P. aeruginosa* not detected; *Staphylococcus aureus* detected by at levels are found in good quality surface water in Canada Ocellate cercaria and non-ocellate isolated detected in snails	Snails
Fields et al., 2001	Wisconsin, U.S.	1998	*L. micdadei*	Pontiac Fever	45	Spa/pool	Heterotrophic bacteria detected; *L. micdadei* detected	Unknown
Fiorillo et al., 2001	Alberta, Canada	1998	*P. aeruginosa*	Pseudomonas hot-foot syndrome	40	Pool	*P. aeruginosa* detected in inlets, the floor, and a drain to the pool	Unknown
Lim et al., 2004	Minnesota, U.S.	1998	*Cryptosporidium*	Gastroenteritis	26	Pool	*Cryptosporidium* not detected	Unknown

Reference	Location	Year of Outbreak	Etiologic Agent	Predominant Illness	Number of Cases	Water Source	Water Quality Information	Pathogen Source
Morgan et al. 2002	Illinois, U.S.	1998	*Leptospira* spp.	Leptospirosis	98	Lake	One of the 27 lake water samples detected a pathogenic *Leptospira* spp.; however, no organism was isolated. Two samples were culture positive; however, both isolated organisms were saprophytic *Leptospira* spp.	Likely runoff due to unusually rainy season, however animal reservoir with epidemic strain was not found
Cransberg et al., 1996	The Netherlands	1993	*E. coli* O157:H7	Hemolytic uremic syndrome	4	Lake	O157:H7-DNA not detected, but sampling occurred ~2 weeks after date of exposure	Possibly human or cattle feces
Joce et al., 1991	Doncaster, U.K.	1988	*Cryptosporidium*	Gastroenteritis	79	Pool	*Cryptosporidium* oocysts detected in learner pool; *Cryptosporidium* oocysts not detected in main pool; rotavirus detected in learner pool	Sewage from main sewer
Porter et al., 1988	New Jersey, U.S.	1985	*Giardia*	Gastroenteritis	9	Pool	Coliform not detected; standard bacterial plate counts of 1-2 per mL detected	Fecal accident
Sorvillo et al., 1988	California, U.S.	1985	*Shigella sonnei*	Gastroenteritis	68	Human-made lake	High fecal coliform detected	Direct bather contamination
Khabbaz et al., 1983	Georgia, U.S.	1981	*P. aeruginosa*	Skin	75	Pool(s)	*P. aeruginosa* serotype 0:9 detected	Unknown
Reid and Porter, 1981	U.K.	1980	*P. aeruginosa*	Otitis externa	18	Pool	*P. aeruginosa* detected in swabs from various places around pool and vacuum bag used to clean pool	Unknown
Baron et al., 1982	Michigan, U.S.	1979	Norwalk agent	AGI	121	Lake	Fecal coliform detected but within acceptable range (<200 colonies per 100 mL of water)	Unknown
Kappus et al., 1982	Ohio, U.S.	1977	Norwalk agent	AGI	103	Pool	Moderate numbers of fecal coliform detected; total coliform (1/100 mL), fecal coliform (1/100 mL), and fecal streptococci (4/100 mL to 9/100 mL) detected in samples sent to EPA; pool chlorinator was not functioning at time of outbreak	Unknown
Rosenberg et al., 1976	Iowa, U.S.	1974	*S. sonnei*	Gastroenteritis	31	River	Fecal coliform detected (mean=17,500 organisms per 100 mL); *Shigella sonnei* detected	Likely sewage treatment plant

Minas Gerais, Brazil (snails)
Seventeen cases of acute schistosomiasis occurred in Minas Gerais, Brazil in 2002 (Enk et al., 2003). Affected individuals bathed in a swimming pool at a holiday resort. Water in the pool was provided from a nearby stream. Environmental sampling from the stream included a survey of site snails (mollusks), which tested positive for *Schistosoma mansoni*.

Quebec City, Canada (snails)
In August 1999, 53 cases of cercarial dermatitis were associated with a recreational tourist lake in Quebec City, Quebec Canada (Lévesque et al., 2002). Water samples showed low concentrations of fecal pollution. *Pseudomonas aeruginosa* was not detected, though *Staphylococcus aureus* was detected at levels corresponding to good quality surface water in Canada. Snails were analyzed for schistosomes, and two forms of furcocercaria type cercariae were identified. The snails were located in only in one location, which is where 42 of the 53 cases occurred.

Rochefort, France (rodent urine)
Five cases of leptospirosis were diagnosed in Rochefort, France in 2001 among teenagers who had swum in the Genouillé canal (Perra et al., 2002). Water samples revealed moderate quality according to France's swimming water criteria (*E. coli* between 100 to 2,000/100 mL). An environmental survey also included trapping rodents and conducting a serological status analysis. Results of this analysis showed that the prevalence of seropositive rodents was 30.8 percent, 23.1 percent, 4.6 percent, and 3.8 percent for total antigens, *L. icterohaemorrhagiae*, *L. saxkoebing*, and *L. australis*, respectively. Consequently, rodent urine was believed to be the primary source of contamination.

Washington, U.S. (duck feces)
In 1999, an outbreak of *E. coli* O157:H7 occurred at a state lake in Vancouver, Washington (Samadpour et al., 2002). The number of cases was not reported; however, the CDC summary of recreational water outbreaks in 1999 and 2000 reported an *E. coli* O157:H7 outbreak that also occurred at a state lake in Vancouver and had 396 cases. Environmental sampling occurred a month after the outbreak and consisted of 108 samples, including water, soil, sand, sediment, and animal fecal matter (cow, coyote, deer, duck, and rabbit). *E. coli* O157:H7 was recovered from both the water and the duck fecal samples. Water samples tested positive for *Stx*, *eaeA*, and *hly* genes by a PCR technique; duck fecal samples tested positive for *Stx* and *eaeA* genes. These virulence factors (*Stx*, *eaeA*, and *hly* genes) were also found in patient stool samples. Additionally, the study used a pulsed-field gel electrophoresis to compare duck feces and water; all isolates resulted in the same restriction fragment patterns. According to the authors, regardless of the evidence, duck feces could still not be confirmed as the primary fecal contamination source. Ducks and the lake could have been transiently infected by the contaminated water, so the initial source of contamination could be human feces or anther animal feces. Also, the delay between the outbreak and the sampling may support the notion that duck feces were not the original source.

IV.7.2 Environmental Sampling not Including Animal Sources

California, U.S. (cattle or deer feces)
An outbreak of seven cases of *E. coli* O157:nonmotile (NM) occurred at a lake in California during August 1999 (Feldman et al., 2002). Potential sources of contamination were fecal matter from cattle or deer based on the presence of a herd of cattle on the opposite bank of the river and the observation by a park manager of numerous deer in the area. Fecal matter from these animals, however, was not analyzed for *E. coli* O157:NM.

Minnesota, U.S. (geese feces)
According to the CDC summary of recreational water outbreaks in 2002 and 2001, there was an outbreak of *E. coli* O157:H7 at a lake beach in which 20 cases were identified (Yoder et al., 2004). A brief case description indicated that an environmental investigation revealed a high level of fecal coliforms. According to local officials, the large number of geese that occupied the beach during the summer may have contributed to the elevated overall fecal coliform (and thus potentially *E. coli* O157:H7) levels.

The Netherlands (cattle feces)
In 1993, four cases of hemolytic uremic syndrome in children were reported in the Netherlands (Cransberg et al., 1996). All four affected individuals bathed in a shallow recreational lake within a 5-day period. *E. coli* O157:H7 was isolated in the stool of two of the patients. Water samples were taken 16 days after the latest date of patient contamination; however, no O157:H7 DNA was detected in filter-concentrated lake water after using PCR enhancement. According to the study authors, the contamination source could have been human- or cattle-based. Cattle feces were identified as a plausible source because the water in the lake is derived from ditches, which drain their water from meadows with cattle. Cattle feces, however, were not analyzed for O157:H7.

V. COMPILATION OF DATA AND SUMMARY

Numerous epidemiological investigations have been conducted since the 1950s to evaluate the association between the density of suitable fecal indicators and the risk of illness to recreational water users. Reviews of these studies are provided in Prüss (1998); Sinton et al. (1998); Wade et al. (2003); and Zmirou et al. (2003). Taken as a whole, the weight of evidence from these studies indicates that fecal indicator bacteria (fecal *streptococcus*/*Enterococcus*, in particular) are able predict GI and respiratory illnesses from exposure to recreational waters (Prüss, 1998; Wade et al., 2003; Zmirou et al., 2003). This broad base of information stems from studies conducted in the following countries (NZME, 2003):

- Australia (Corbett et al., 1993; Harrington et al. 1993)
- Canada (EHD, 1980; Lightfoot, 1989; Seyfried et al., 1985a,b)
- Egypt (Cabelli, 1983b; El-Sharkawi and Hassan, 1979)
- France (Ferley et al., 1989; Foulon et al., 1983)
- Hong Kong (Cheung et al., 1990; Kueh et al., 1995)
- Israel (Fattal et al., 1986, 1987, 1991)
- Netherlands (Medema et al., 1995, 1997)
- New Zealand (McBride et al., 1998)
- South Africa (von Schirnding et al., 1992, 1993)
- Spain (Mariño et al., 1995; Mujeriego et al., 1982)
- United Kingdom (Alexander et al., 1992; Balarajan et al,. 1991; Fewtrell et al., 1992, 1994; Fleisher et al. 1996; Kay et al., 1994)
- United States (Cabelli et al., 1983a; Dufour, 1984; Stevenson, 1953)

However, as indicated above, most of these studies investigated waters that were impacted or influenced by wastewater effluent, and close inspection reveals that few studies addressed sources of contamination other than wastewater effluent in the investigated waters.In fact, prior to 1999, the only peer-reviewed publications that substantially addressed this topic were Cheung et al. (1990), Calderon et al. (1991), and McBride et al. (1998). In recent years, researchers have conducted several additional epidemiological studies focusing on waters not predominately impacted by wastewater effluent, including Haile et al. (1999); Dwight et al. (2004); Wiedenmann et al. (2006); and Colford et al. (2007). Additionally, the SCCWRP is also conducting a series of epidemiological studies that investigate recreational water with various contamination sources other than wastewater effluent. Section III.2 provided a brief overview of each of these studies.

Review of the epidemiological studies that address recreational water predominantly impacted by fecal contamination sources other than wastewater effluent indicates that the results are equivocal. On one hand, Colford et al. (2007) found that the incidence of swimmer illness was not associated with any of the traditional fecal indicators at a marine beach with primarily avian contamination. Similarly, Calderon et al. (1991) found no statistically significant association between swimmers' illness risk and animal fecal contamination in a freshwater pond. On the other hand, 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. Furthermore, the marine bathing study in New Zealand (McBride et al. 1998) indicated that illness risks posed by animal versus human fecal material were not substantially different; however, the study's limited range of beach contamination precluded the development of a detailed statistical model of health risks versus indicator density.

In the first study to be conducted in waters directly impacted by urban runoff, Haile et al. (1999) reported rates of illnesses in Southern California similar to those conducted in waters contaminated with domestic sewage. However, this nonpoint runoff source was known to have human sources of fecal contamination (Colford et al., 2007). Similarly, Dwight and colleagues (2004) found that surfers exposed to Southern California urban runoff had higher illness rates than surfers exposed to Northern California rural runoff. The results from the Hong Kong marine water study (Cheung et al., 1990) and the German freshwater study (Wiedenmann et al., 2006) are more difficult to interpret regarding risks from human versus nonhuman sources because in both studies, the analyses combined the results from sites with different predominant contamination sources.

In reviewing outbreak information for drinking water and recreational waters, several overarching points emerge. One is that the pathogen source in the majority of drinking water-related outbreaks remains unknown. The source of pathogens in drinking water outbreaks in many cases could have been humans or animals; however, most reports offered little detail, leaving a critical information gap for the purposes of this review. Keeping in mind the previously noted limitations of outbreak investigations, it is noteworthy that several outbreak investigation studies were able to link pathogens isolated from patients with water samples, animals, or both using laboratory analysis (e.g., CCDR 2000; Howe et al., 2002; Licence et al., 2001). Other reports used circumstantial evidence to link animal waste to outbreaks, but although compelling, laboratory results were not available to confirm the contamination source. The animal sources linked to outbreaks included beavers, cats/cougars, deer, elk, pigs, cattle, and chickens/poultry, and the corresponding animal-related pathogens in these outbreaks were *Giardia intestinalis*, *Cryptosporidium* spp., *E. coli* O157:H7, *Campylobacter* spp., *Toxoplasma gondii*, and *S. typhimurium*. In general, 14 percent of the drinking water outbreaks reported by the CDC from 1999 to 2004 were possibly animal-related (Blackburn et al., 2004; Lee et al., 2002; Liang et al., 2006). In addition, about 15 percent of Canadian outbreaks between 1974 and 2001 were linked to animal sources (Schuster et al., 2005) and about half of the outbreaks linked to private water supplies in England and Wales listed animals as possible factors (Said et al., 2003).

Given that outbreaks are known to be a notoriously poor measure of the actual number of infections and illnesses caused by waterborne pathogens (Craun, 2004), those investigations that link pathogens isolated from patients, water samples, or both with animals provide unequivocal evidence that human illnesses can and do occur from animal-based contamination. Unfortunately, the drinking water outbreak literature does not substantially enhance the current ability to differentiate risks from animal- versus human-related pathogen sources for recreational water exposures in a quantitative manner.

The recreational water outbreak literature (Craun et al., 2005) indicates that of the 259 recreational water outbreaks that occurred in the United States between 1970 and 2000, only approximately half included any information about possible sources of the contamination or contributing factors to it. Over two-thirds of the outbreaks that did list possible sources were associated with either bacteria or protozoa; over 15 percent had unidentified etiologies; and 7 percent were viral. In untreated recreational water outbreaks (streams, lakes, etc.), feces or ill bathers in the water, bather overcrowding, and the presence of children in diapers accounted for the vast majority of the assumed sources; however, the report by Craun et al. (2005) did not indicate that laboratory analyses, other than microbial indicator measures, supported these attributions. Their analysis estimated that 18 percent of these outbreaks were associated with animals and that the likely etiologic agents included *E. coli* spp., *Schistosomes* spp., and *Leptospira* spp. *E. coli* was associated with cattle, deer, or duck feces; *Schistosomes* spp. were associated with snails; and *Leptospira* spp. were associated with rat urine. Similar to the drinking water outbreak compilation, the recreational water outbreak literature does not appear to enhance substantially the current state of knowledge on quantitatively characterizing risks from animal-related pathogen sources compared with human sources for recreational water exposures.

Given that relatively few investigations worldwide have evaluated the risk to human health from recreational exposure to waters primarily impacted by sources of contamination other than wastewater effluent, and that the potential range of those sources is broad, the findings from this literature review are not surprising. In fact, the results of this literature review seem consistent with WHO's (2004) report on zoonoses, which indicated the following:

- Inadequate information exists on differentiating human versus animal strains of human pathogens, both in the field (e.g., pathogen typing and microbial source tracking) and analytically (e.g., relative infectivity and pathogenicity). Both of these areas are priorities for targeted research.
- Currently available surveillance data on both sporadic (endemic) and outbreak diseases are of limited use in understanding the importance of zoonotic waterborne infection. Surveillance for waterborne disease in general and waterborne zoonoses in particular has failed to provide a meaningful indication of the associated burden of disease, even in countries with established surveillance systems.
- The risk of exposure to animal-contaminated water is unknown. Studies to define the risk associated with swimming in animal-contaminated water have not clearly indicated that this type of exposure results in an excess illness rate. These results of these studies do not support the premise that all fecally contaminated waters should be treated the same. New research to define the risk of illness posed by animal fecal wastes is needed (Till et al., 2004).

Furthermore, because pathogens can evolve rapidly depending on their previous host environments due to changing host factors and other mechanisms of phenotypic change, it is reasonable to suspect that zoonotic pathogens propagated in animals would be different from pathogens from human sources. However, data remain incomplete on how zoonotic pathogens are attenuated or increase infectivity or virulence when passaged through animal hosts.

Although conclusive information on differentiating human versus animal sources of pathogens is lacking, several research organizations and countries have suggested novel approaches for addressing risks from nonhuman sources (e.g., NZME, 2003; WSSA, 2003). For example, New Zealand, where about 80 percent of total notified illnesses are zoonotic and potentially waterborne, has recently updated its recreational water quality criteria to address the issue of animal-source waterborne contamination by basing its freshwater guidelines principally on the risks associated with campylobacteriosis using *E. coli* concentrations as an indicator (Till and McBride, 2004).

In 1992, the New Zealand Department of Health issued provisional microbiological water quality guidelines for recreational waters, which advised that exposure to animal fecal microorganisms was much less of a risk than exposure to pathogens of human origin. In 1998, Sinton et al. concluded that there was no reliable epidemiological information on the relative risks to humans associated with human and animal fecal pollution in water, but that the high ratio of grazing animals to humans in the country made animal fecal sources a greater health risk to humans than previously assumed. Because *Campylobacter*-related illness is of great public health concern in New Zealand, research has been especially focused on the dairy industry and its impact on microbial water quality (Till and McBride, 2004). Thus, although New Zealand's current marine water guidelines (NZME, 2003) derive directly from WHO's 2003 recommendations, the country bases its freshwater guidelines principally on the risks associated with campylobacteriosis (using *E. coli* concentrations as an indicator). Notably, Till and McBride (2004) stated that current investigations in New Zealand have focused on determining the source of human *Campylobacter* infections in part because that information "…may help to shed light on the relative health risk of animal versus human wastes."

Based on the results of this detailed literature review, it appears that: (1) insufficient information is available to support a robust and quantitative characterization of the relative risks of human illness from the range of various sources of fecal contamination in recreational waters, and (2) epidemiological studies are more likely to provide salient insights than outbreak investigations. Continued epidemiological work in this arena is consistent with ongoing international efforts to understand and manage human health risks associated with exposure to waterborne zoonoses (Till and McBride, 2004).

Tools that complement epidemiology may help address the characterization of the relative risks of human illness from various sources of fecal contamination in recreational waters; for example, quantitative microbial risk assessment (QMRA) is one alternative that could supplement traditional epidemiological investigations to identify potential excess human health risks for defined pathways of particular pathogens (Carr and Bartram, 2004). Because QMRA has the potential to provide much greater sensitivity in quantifying risk than epidemiological studies (Carr and Bartram, 2004), the potential synergy from using both methods could be helpful to manage risks of water-related infectious diseases (see Bartram et al., 2001). To date, data gaps have limited the degree to which QMRA has been used to inform public policy decisions and manage recreational waters. The greatest data deficiencies appear to be a lack of dose-response and environmental occurrence data for many waterborne zoonotic pathogens (Carr and Bartram, 2004; Till and McBride, 2004).

In addition, more robust environmental testing in recreational and drinking water outbreak investigations using rapidly evolving molecular methods is another complementary tool that could supplement traditional epidemiological investigations in characterizing the relative risks of human illness from the range of various sources of fecal contamination in recreational waters. A notable drawback to this approach is that state and local health departments, who are responsible for investigating outbreaks, are unlikely to have the technical or financial resources to pursue this sort of testing, so extramural funding would likely be needed.

Finally, even if or when sufficient information becomes available to support robust and quantitative characterization of the relative risks of human illness from the range of fecal contamination sources in recreational waters, a number of issues will likely remain regarding the use of indicator organisms to predict human illnesses from exposure to these waters. The essential intent of fecal indicators is that they represent the overall "pathogenicity" of the water (NZME, 2003). Within this context, it is not surprising that indicator organisms correlate well to some pathogens in some waters but correlate poorly in other cases (e.g., Ashbolt et al., 1993; Elliott and Colwell, 1985; Ferguson et al., 1996; Grabow et al., 1989; Jiang et al., 2001). If new or revised water quality criteria are to address quantitatively the various sources of fecal contamination of potential interest in recreational waters covering diverse geographic regions, the approach will likely require a set or toolbox of indicators (as measured by specific methods). Recent research documenting the extreme variability in densities of traditional indicators over very short time periods (Boehm, 2007) underscores the need for a variety of indicator/method combinations that address a range of contamination sources and geographic areas.

In summary, both human and animal feces in recreational waters continue to pose threats to human health. Although the public health importance of waterborne zoonotic pathogens is being increasingly recognized, it is still not well characterized. Policy makers and researchers have often assumed that the human health risk from pathogens associated with domestic and agricultural animal and wildlife feces is less than the risk from human feces, in large part because viruses are predominately host-specific. This literature review illustrates a lack of detailed and unequivocal information concerning the relative risks of human illness from various sources of fecal contamination in recreational waters. In addition, the ability to measure how the infectivity and virulence of known waterborne zoonotic pathogens are affected when passaged through animal hosts remains in its infancy. Thus, the findings of this literature review support the perspectives set forth by WHO (2004) and indicate that addressing fecal contamination sources in recreational water quality criteria will require additional research to better define the risk posed by animal fecal matter.

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APPENDIX A

LITERATURE SEARCH STRATEGY AND RESULTS

The literature search strategy consisted of a number of combined approaches. Search terms and a synopsis of information needed were given to a professional librarian to search the online DIALOG databases. To supplement the DIALOG searches, individual authors used free search engines on the internet to find articles pertaining to specific information needed. Experts that participated in EPA's Experts Scientific Workshop on Critical Research Needs for the Development of New or Revised Recreational Water Quality Criteria[1] were contacted by email and requested to contribute literature they felt was important. The titles of literature cited in specific reports, books, review articles, and conference proceedings were evaluated for relevance.

A.1 Initial Literature Search Strategy Conducted by Professional Librarian

Selection of DIALOG data base files used for this search:

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  File 155:MEDLINE(R) 1950-2007/Nov 12
         (c) format only 2007 Dialog
  File 266:FEDRIP 2007/Sep
         Comp & dist by NTIS, Intl Copyright All Rights Res
  File 245:WATERNET(TM) 1971-2007Jul
         (c) 2007 American Water Works Association
  File 144:Pascal 1973-2007/Oct W4
         (c) 2007 INIST/CNRS
  File 117:Water Resources Abstracts 1966-2007/Aug
         (c) 2007 CSA.
  File  40:Enviroline(R) 1975-2007/Sep
         (c) 2007 Congressional Information Service
  File 110:WasteInfo 1974-2002/Jul
         (c)  2002 AEA Techn Env.
  File 143:Biol. & Agric. Index 1983-2007/Oct
         (c) 2007 The HW Wilson Co
  File   6:NTIS 1964-2007/Nov W3
         (c) 2007 NTIS, Intl Cpyrght All Rights Res
  File   5:Biosis Previews(R) 1926-2007/Nov W2
         (c) 2007 The Thomson Corporation
  File  73:EMBASE 1974-2007/Nov 12
         (c) 2007 Elsevier B.V.
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Search strategies used for this search:

#1: Waterborne terms NEAR fecal terms

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S1       3210   WATERBORNE()(DISEASE? ? OR OUTBREAK? ? OR ZOONOT? OR ZOONOS?
                OR EPIDEMIC? ? OR EPIDEMIOLOG?)
S2         43   WATERBORNE()(HEPATITIS OR ROTAVIRUS? OR NOROVIRUS?)
S3     232098   FECAL OR FECES FAECAL OR (LIVESTOCK OR BOVINE OR CATTLE)(S)
```

[1] Report from this workshop: http://www.epa.gov/waterscience/criteria/recreation/.

```
                CONTAMINATION
S4        1637  PATHOGEN()FECAL()SHEDDING OR PATHOGENS (S) SEWAGE OR RAW()
                SEWAGE()CONTAMINATION
S8         207  S1 (S) S3
S9         250  S2 OR S8
S10        140  RD S9  (unique items--deduped; no year/language limits)
```

#2: Pathogen terms NEAR Sewage terms AND (Waterborne or Recreational Water Terms)

```
S4        1637  PATHOGEN()(FECAL OR FAECAL)()SHEDDING OR PATHOGENS (S) SEWAGE
                OR RAW()SEWAGE()CONTAMINATION
S29       1282  S4 NOT (S9 OR S13 OR S16 OR S21)
S30        717  RD S29  (unique items)
S31        110  S30 AND (S11 OR S12 OR S1) (WATERBORNE/RECREATIONAL WATER
                SETS)
```

#3: (Remaining Pathogen terms OR Indicator Terms) AND Selected Authors

```
S34        592  S30 NOT (S31 OR S33) [REMAINING PATHOGEN RESULTS]
S35       2164  S7 NOT (S9 OR S13 OR S16 OR S33 OR S21 OR S31) [INDICATORS]
S36       1078  RD S35  (unique items)
S37       2011  AU=(ASHBOLT ? OR PAYMENT ?  OR PRUSS ?)
S38      33410  S37 OR AU=(BYAPPANAHALLI? OR COLFORD ? OR DUFOUR ?  OR
                MCBRIDE ?  OR GANNON ? OR CICMANEC? OR COTRUVO?)
S39       38    (S34 OR S36) AND (S37 OR S38) [AUTHORS ON THOSE 2 TOPICS]
```

#4: (Remaining Pathogen terms OR Indicator Terms) AND Waterborne or Recreational Water Terms)

```
S5      462847  CLOSTRIDIUM OR BACTEROIDES OR TOTAL()(FECAL OR FAECAL)()
                COLIFORM OR E()COLI OR ECOLI OR ENTEROCOCC? OR FECAL()
                STREPTOCOCC? OR COLIPHAGE
S6      540854  INDICATOR? ?
S7        2551  S5 (5N) S6
S42       1611  (S34 OR S36) NOT S39
S43        243  S42 AND (S1 OR S11 OR S12) [combined w/waterborne/
                recreational terms]
```

Dates: 1985-present [2002-present is more crucial]
Language: no restrictions

Descriptions of these files are available at http://library.dialog.com/bluesheets/.

Retrieve: titles and year
Format: MS Word
Additional information: Interested in international and domestic journals and government reports.

Search terms:
Fecal waterborne disease* (i.e., * allows for multiple endings)
Waterborne zoono* (for zoonotic, zoonosis, zoonoses)
Waterborne outbreak*
Waterborne disease epidemiology
Livestock contamination of water
Fecal load
Pathogen fecal shedding
Pathogens AND sewage
Microbial quality of effluent
Waterborne Hepatitis
Waterborne rotavirus*
Waterborne Norovirus*
Raw sewage contamination
Clostridium AND indicator*
Bacteroides AND indicator*
total fecal coliform, AND indicator*
E. coli and Indicator*
Enterococcus AND indicator*
fecal *streptococcus* AND indicator*
coliphage AND indicator*

Key authors:
Ashbolt N
Payment P
Prüss A
Byappanahalli M
Colford J
Dufour A
McBride GB
Gannon VPJ
Cicmanec JL
Cotruvo JA

Specific literature:
WHO. (2004) Waterborne Zoonoses. http://www.who.int/water_sanitation_health/diseases/zoonoses.pdf
All MMWR supplements on waterborne illness (1983 to present)
Airlie report (USEPA, 2007) references

Objectives of this literature search:

- All epidemiological studies of waterborne illness due to ambient recreational exposures (any location)
- *Morbidity and Mortality Weekly Report* surveillance summaries and papers that evaluate trends based on CDC data for waterborne illnesses due to recreational exposures (ambient and pools)
- Papers on outbreaks due to ambient recreational exposures (not pools)
- References to illness resulting from waterborne exposure to fecal materials from a variety of sources (emphasis on animal-derived waste, including point and nonpoint sources)
- Prevalence of fecal indicators in humans and animals (livestock and wild animals): (total and fecal coliform, *E. coli*, *Enterococcus*, fecal *streptococcus*, *Bacteroides*, coliphage (somatic and F+), and *Clostridium)* variation of quantities in fecal material (temporal and within a population)
    - References for a Fecal Shedding Table (average for a species): columns – indicator and pathogen shedding quantification, ratio of indicator to pathogens (if known or can be calculated without being misleading), primary literature citations; rows – species
        - Variation and uncertainty in shedding data (how much and how frequently do infected animals shed pathogens in feces)
        - magnitude and duration of fecal excretion of specific pathogens during illness
- Pathogen prevalence in herds or wild animals
    - number of infected per herd
    - geographic distribution of herds
    - seasonal, temporal, event driven (calving) variation
- Pathogen prevalence in raw and treated sewage (mostly human source) – likely variation
- human point and nonpoint source contaminant levels quantified in receiving waters

A.2 Literature Search Strategy and Results Specific to Epidemiological Studies Conducted in Recreational Waters

The following terms were searched on the Web of Science:

- Recreational water + epidemiology + fecal matter
- Recreational water + epidemiology + nonpoint source
- Recreational water + epidemiology + point source
- Recreational water + epidemiology + animal fecal matter
- Recreational water + epidemiology + animal waste
- Recreational water + exposure to fecal matter
- Recreational water + epidemiological studies + microbiological indicators of fecal matter
- Recreational water + epidemiology + fecal matter + European Union
- Recreational water + epidemiology + fecal matter + United Kingdom

A summary of the results of this literature search is provided in Table A.2.1

Table A.2.1. Summary of Epidemiological Studies in Recreational Waters

Search Topic	# Citations	Citations Reviewed (Web information only)	# Citations of Interest
Recreational water + epidemiology + fecal matter	50	Yes	1
Recreational water + epidemiology + nonpoint source	30	Yes	3
Recreational water + epidemiology + point source	30	Yes	3
Recreational water + epidemiology + animal fecal matter	20	Yes	1
Recreational water + epidemiology + animal waste	30	Yes	4
Recreational water + exposure to fecal matter	30	Yes	1
Recreational water + epidemiological studies + microbiological indicators of fecal matter	40	Yes	5
Recreational water + epidemiology + fecal matter + European Union	40	Yes	2
Recreational water + epidemiology + fecal matter + United Kingdom	40	Yes	4
New + recreational waters + epidemiological studies + EU	60	Yes	10

The results of this literature review were then combined into a library with the relevant citations from a reverse literature citation search on several of the key epidemiological studies (e.g., the Wade et al., 2003; Zimrou et al., 2003; Prüss 1998; and Sinton et al., 1998 review articles), and duplicates were discarded. Additionally, publications describing studies conducted in Europe were obtained for free from Dr. David Kay, one of the key investigators of epidemiological studies in Europe (http://www.aber.ac.uk/iges/staff/kaydavid.shtml). Furthermore, unpublished information on current and ongoing activities in the European Union was obtained through personal communications with Dr. Kay and a review of the relevant activity's website.

A.3 Literature Search Strategy and Results Specific to Outbreak Data

Drinking Water

The purpose of the literature search was to capture published articles on waterborne disease outbreaks that identified an etiological pathogen and linked it to a drinking water source. Due to the broad nature of the topic, many more papers have been published in the peer-reviewed literature related to drinking water outbreaks, and this literature search by no means represents all of them. The searches were performed on December 5 to 6, 2007.

A Google Scholar search of "waterborne AND outbreak" resulted in 9,940 hits, and a search of "drinking AND water AND outbreak" resulted in 42,900 hits, which were not reviewed. A search of Scirus scientific database of "waterborne AND outbreak" resulted in 13,694 hits which were not reviewed.

The following terms were searched on the National Library of Medicine's PubMed database:

- water + outbreak

- waterborne + outbreak
- drinking + water + outbreak
- zoono* + water + outbreak (title only)

A summary of the results of this literature search is provided in Table A.3.1.

Table A.3.1. Summary of Outbreak Investigations in Drinking Water

Search Topic	# Citations	Citations Reviewed	# Citations of Interest
Water AND outbreak	3,344	No	
Waterborne AND outbreak	448	Yes	118
Drinking AND water AND outbreak	497	Yes	146
Zoono* AND water AND outbreak	81	Yes	0

The results of the two searches that produced citations of interest were combined, and duplicates and articles in languages other than English were discarded. The resulting list was narrowed to 55 articles, which were chosen based on an indication in the abstract that the etiological pathogen in the outbreak was actually detected in the drinking water source.

Recreational Water

For the literature search regarding recreational water outbreaks, the following terms were searched on the U.S. National Library of Medicine's PubMed system:

- (beach OR lake OR stream OR pond OR swim OR recreation) AND water AND (outbreak OR epidemic)

A summary of the results of this literature search is provided in Table A.3.2.

Table A.3.2. Summary of Outbreak Investigations in Recreational Water

Search Topic	# Citations	Citations Reviewed	# Citations of Interest
(Beach OR lake OR stream OR pond OR swim OR recreation) AND water AND (outbreak OR epidemic)	52	Yes	40

Abstracts from the literature identified were reviewed, and studies were included if the abstract showed evidence that the etiologic agent was detected in the source water.

A.4 Summary of Literature Search Results

This process resulted in a total order of 365 citations of which a total of 273 (75 percent) were received during the expedited writing process, not all of which could be reviewed. There are

many more papers in the peer-reviewed literature and this by no means represents all of them. Of the articles reviewed, 182 citations were included in the white paper.

A.5. Supplemental Literature Search Strategy

In addition to the literature search conducted by the professional librarian several other resources were consulted.

The flowing experts in the field were contacted directly by email and asked to suggest references:

Nicholas Ashbolt, USEPA
Thomas Atherholt, New Jersey Department of Environmental Protection
Michael Beach, Centers for Disease Control and Prevention
Bart Bibler, Florida Department of Health
Alexandria Boehm, Stanford University, California
Rebecca Calderon, USEPA
Jennifer Clancy, Clancy Environmental Consultants
Jack Colford, University of California, Berkeley
Elizabeth Doyle, USEPA
Alfred Dufour, USEPA
Lee Dunbar, Connecticut Department of Environmental Protection
Lora Fleming, University of Miami School of Medicine and Rosenstiel School of Marine and Atmospheric Sciences, Florida
Charles Hagedorn, Virginia Tech
Joel Hansel, USEPA
Lawrence Honeybourne, Orange County Health Care Agency, Santa Ana, California
Donna Francy, U.S. Geological Survey
Roger Fujioka, University of Hawaii, Manoa
Toni Glymph, Wisconsin Department of Natural Resources
Mark Gold, Heal the Bay, California
Paul Hunter, University of East Anglia, U.K.
Dennis Juranek, Centers for Disease Control and Prevention (retired)
David Kay, University of Wales, U.K.
Sharon Kluender, Wisconsin State Laboratory of Hygiene
Erin Lipp, University of Georgia
Graham McBride, National Institute of Water and Atmospheric Research, New Zealand
Charles McGee, Orange County Sanitation District, California
Samuel Myoda, Delaware Department of Natural Resources
Charles Noss, USEPA
Robin Oshiro, USEPA
James Pendergast, USEPA
Mark Pfister, Lake County Health Department, Illinois
John Ravenscroft, USEPA
Stephen Schaub, USEPA
Mark Sobsey, University of North Carolina, Chapel Hill

Jeffrey Soller, Soller Environmental, California
Michael Tate, Kansas Department of Health and Environment
Peter Teunis, RIVM (National Institute of Public Health and the Environment), Netherlands
Gary Toranzos, University of Puerto Rico, Rio Piedras
Timothy Wade, USEPA
John Wathen, USEPA
Stephen Weisberg, Southern California Coastal Water Research Project
David Whiting, Florida Department of Environmental Protection
Richard Zepp, USEPA

In addition to contacting experts in the field, specific reports were obtained and the titles of the references cited in the reports were reviewed for relevance.

- NRC. 2004. Indicators for waterborne pathogens. National Academies Press. Washington, DC. 315 pp.
- USEPA. 2007. Report of the experts scientific workshop on critical research needs for the development of new or revised recreational water quality criteria. EPA 823-R-07-006. Available online at http://www.epa.gov/waterscience/criteria/recreation
- References cited by the Natural Resources Defense Council reviewers of the EPA Critical Path Science Plan
- Prüss (1998) Review of Epidemiological Studies on Health Effects from Exposure to Recreational Water. *International Journal of Epidemiology* 27:1-9.
- Wade, T.J., Pai, N., Eisenberg, J.N., and Colford, Jr., J.M. 2003. Do U.S. Environmental Protection Agency water quality guidelines for recreational waters prevent gastrointestinal illness? A systematic review and meta-analysis. Environmental Health Perspectives 111(8):1102-1109.
- Zmirou, D., Pena, L., Ledrans, M., and Letertre, A. 2003. Risks associated with the microbiological quality of bodies of fresh and marine water used for recreational purposes: Summary estimates based on published epidemiological studies. Archives of Environmental Health 58(11): 703-711.
- Sinton, L.W., Finlay, R.K., Hannah, D.J. 1998. Distinguishing human from animal fecal contamination in water: A review. New Zealand Journal of Marine and Freshwater Research 32: 323-348.
- Boehm et al. 2008. A sea change ahead for recreational water quality criteria (peer review in progress)

In addition, Clancy Environmental Consultants, ICF International, Soller Environmental, WaltJay Consulting, and EPA's Health and Ecological Criteria Division all maintain extensive literature databases and reference lists from previously completed projects. All of those in house resources were also sources of literature.