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Estimating the primary etiologic agents in recreational freshwaters impacted by human sources of faecal contamination[☆]

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ABSTRACT

Epidemiology studies of recreational waters have demonstrated that swimmers exposed to faecally-contaminated recreational waters are at risk of excess gastrointestinal illness. Epidemiology studies provide valuable information on the nature and extent of health effects, the magnitude of risks, and how these risks are modified or associated with levels of faecal contamination and other measures of pollution. However, such studies have not provided information about the specific microbial agents that are responsible for the observed illnesses in swimmers. The objective of this work was to understand more fully the reported epidemiologic results from studies conducted on the Great Lakes in the US during 2003 and 2004 by identifying pathogens that could have caused the observed illnesses in those studies. We used a Quantitative Microbial Risk Assessment (QMRA) approach to estimate the likelihood of pathogen-induced adverse health effects. The reference pathogens used for this analysis were Norovirus, rotavirus, adenovirus, *Cryptosporidium* spp., *Giardia lamblia*, *Campylobacter jejuni*, *Salmonella enterica*, and *Escherichia coli* O157:H7. Two QMRA-based approaches were used to estimate the pathogen combinations that would be consistent with observed illness rates: in the first, swimming-associated gastrointestinal (GI) illnesses were assumed to occur in the same proportion as known illnesses in the US due to all non-foodborne sources, and in the second, pathogens were assumed to occur in the recreational waters in the same proportion as they occur in disinfected secondary effluent. The results indicate that human enteric viruses and in particular, Norovirus could have caused the vast majority of the observed swimming-associated GI illnesses during the 2003/2004 water epidemiology studies. Evaluation of the time-to-onset of illness strongly supports the principal finding and sensitivity analyses support the overall trends of the analyses even given their substantial uncertainties.

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1. Introduction

Epidemiology studies on recreational waters have demonstrated that swimmers exposed to faecally-contaminated recreational waters are at risk of excess gastrointestinal (GI) illness (Prüss, 1998; Wade et al., 2003; Zmirou et al., 2003). Moreover, studies conducted by the US EPA and others have further demonstrated exposure–response relationships between faecal indicator organisms such as enterococci and *E. coli* and swimming-associated gastrointestinal illness (Cabelli et al., 1982; Dufour, 1984; Fleisher et al., 1996; Haile et al., 1999; Kay et al., 1994; Wade et al., 2006), and comprehensive literature reviews have concluded general support for these associations in over 20 epidemiology studies conducted around the world (Prüss, 1998; Wade et al., 2003; Zmirou et al., 2003).

However, little is known about the specific microbial agents that are responsible for the observed illnesses in swimmers. Nearly 30 years ago, Cabelli (1983) stated: “An intensive program should be initiated towards establishing the etiology of the gastroenteritis observed in these studies and developing methods for quantifying the agent(s) in environmental waters.” While several studies have attempted to collect biologic specimens (blood or stool) as part of epidemiologic research at beach sites, to date these efforts have been largely unsuccessful in identifying the agents responsible for the observed increase in GI symptoms among swimmers (Jones et al., 1991). Information on the etiology of illnesses observed during epidemiology studies is important for several purposes including understanding and managing risks (Ashbolt et al., 2010), developing appropriately protective public health regulations, and potentially extending the observed health relationships to other waters whose characteristics and/or sources are not necessarily covered by the epidemiologic studies (Schoen and Ashbolt, 2010; Soller et al., 2010).

We used a Quantitative Microbial Risk Assessment (QMRA) paradigm to understand more fully the reported epidemiologic results from the US EPA National Epidemiological and Environmental Assessment of Recreational (NEEAR) water studies conducted on the Great Lakes in the US during 2003 and 2004 (Wade et al., 2006, 2008) and suggest which pathogens represent the observed illnesses in those studies. US EPA specifically selected the Great Lakes sites because they were thought to be impacted by sewage treatment plant effluent. Beaches were located in proximity to point-source tributaries that received treated wastewater from communities with populations of at least 38,000 and with flow rates of over 10 million gallons per day. These sewage treatment plants provided secondary treatment as well as disinfection with chlorine or ultraviolet radiation during the summer (Wade et al., 2008). QMRA is a process that estimates the likelihood of adverse human health effects that can occur following exposure to pathogens (ILSI, 1996, 2000). This work is one component of a more comprehensive US EPA effort to conduct QMRA on waters impacted by a variety of faecal contamination sources to help facilitate Ambient Water Quality Criteria development and/or implementation for recreational waters (U.S. EPA, 1986, 2007).

Quantitative methods to characterize human health risks associated with exposure to pathogens were first published in the 1970s (Dudley et al., 1976; Fuhs, 1975) and have

proliferated in the literature since that time addressing exposures in diverse media including water (Crabtree et al., 1997; Gerba et al., 1996; Haas, 1983; Mena et al., 2003; Regli et al., 1991; Rose et al., 1991; Teunis et al., 1997), food (Buchanan et al., 1998, 2000; Farber et al., 1996), and other media (Brooks et al., 2005; Eisenberg et al., 2004, 2008; Riley et al., 2003). Surprisingly, few QMRA studies have been published that specifically address risks in recreational waters (Ashbolt et al., 1997; Rose et al., 1987; Roser et al., 2007; Schoen and Ashbolt, 2010; Soller et al., 2003, 2006, 2010).

Here, we employ QMRA to estimate feasible concentrations of the pathogens of public health concern in a human-impacted recreational water that would be consistent with the observed NEEAR GI illnesses reported (Wade et al., 2006, 2008). Specifically, using the observed rates of swimming-associated GI illness and presumed faecal contamination source to the waterbodies in the NEEAR studies, we identify the likely etiologic agents causing GI illness. This work is intended to enhance interpretation of results of the epidemiology study and possible extension of the findings to sites for which epidemiologic studies are not planned.

2. Methods

The analysis entailed matching illness rates observed during the course of epidemiologic studies with predicted illness rates computed using estimated pathogen densities consistent with municipal wastewater/human faecal pollution sources. The analyses began with the faecal indicator densities (*Enterococcus* qPCR calibrated cell equivalents, CCE) observed during the epidemiologic investigations on the Great Lakes (Wade et al., 2006, 2008). Using the published relationship between qPCR signal and GI illness (Wade et al., 2008), the faecal indicator levels were used to estimate the expected value for the rate of swimming-associated illness for each study day.

Additional data were then used to estimate reference pathogen densities in the recreational water consistent with the observed rate of swimming-associated illness on each of the study days. Two complementary approaches were employed to derive those concentrations: 1) a health-based approach in which illnesses are assumed to occur in swimmers in the same proportion as reported illnesses occur in the US due to all non-foodborne sources and, 2) a publically-owned treatment works [POTW] effluent-based approach in which reference pathogens were assumed to occur in the recreational waters in the same proportion as they are reported to occur in disinfected secondary effluent.

2.1. Analysis of faecal indicator data from the NEEAR studies

These data comprised 1600 observations for 78 individual beach exposure days. These data were reduced as reported in the literature (Wade et al., 2008) resulting in a total of 78 daily average mean *Enterococcus* qPCR values. The swimming-associated GI illness rate among all swimmers as a function of daily average \log_{10} *Enterococcus* qPCR CCE was based on a linear probability regression model that adjusted for

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