APPENDIX U

Cryptosporidiosis-Associated Mortality Following a Massive Waterborne Outbreak in Milwaukee, Wisconsin

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ABSTRACT

[illegible] years before the [illegible] sporidiosis was listed [illegible] lying or contributing [illegible] on the death certificates [illegible] Milwaukee-vicinity [illegible] approximately 2 years after [illegible] break, this number was [illegible] whom 85% had acquired [illegible] deficiency syndrome (AIDS) [illegible] as the underlying cause of death. [illegible] the first 6 months after the outbreak, [illegible] the number of death certificates [illegible] indicating AIDS, but not cryptosporidiosis, as a cause (or [illegible] 19 (95% confidence interval = 12,26) higher than pre-outbreak trends would have predicted.

Conclusions. Waterborne outbreaks of cryptosporidium infection can result in significant mortality, particularly among immunocompromised populations. Any discussion of policies to ensure safe drinking water must consider the potential [illegible] consequences of waterborne cryptosporidiosis among immunocompromised populations. (*Am J Public Health*. 1997;87:2033–2035)

Introduction

During March and April 1993, a massive, waterborne outbreak of cryptosporidiosis occurred among residents of and visitors to Milwaukee, Wis. In Milwaukee, water obtained from Lake Michigan is chlorinated and filtered at one of two Milwaukee Water Works plants before entering the water distribution system. The source of this outbreak was Lake Michigan water contaminated with *Cryptosporidium* oocysts. This contamination was not adequately removed at one of the Milwaukee water treatment facilities, allowing *Cryptosporidium* oocysts to enter the drinking water supply. It is estimated that 403 000 residents living in a five-county area and numerous visitors to the city of Milwaukee experienced watery diarrhea during this outbreak.[1,2]

Cryptosporidiosis is characterized by watery diarrhea, often with abdominal cramping, nausea, vomiting, and fever.[2-5] In otherwise healthy persons, the infection and disease are usually self-limited; in immunocompromised hosts, however, *Cryptosporidium* infection can be unrelenting and fatal.[4,5] Understanding the potential for fatal outcomes associated with waterborne cryptosporidiosis outbreaks needs to be an important part of discussions about preventing such outbreaks. This report presents results of an analysis of death certificate data to provide an estimate of cryptosporidiosis-associated mortality during the 2 years following the massive waterborne outbreak of *Cryptosporidium* infection in Milwaukee.

Methods

Wisconsin death certificate data obtained from the Center for Health Statistics, Wisconsin Division of Health, were analyzed for April 1, 1990, through March 31, 1995. The Milwaukee waterborne cryptosporidiosis outbreak began in mid- to late March 1993.[1] For the purposes of this report, March 15, 1993, is defined as the beginning of the interval of the waterborne *Cryptosporidium* exposure that led to the Milwaukee outbreak. The overall study period encompasses approximately 2-year intervals before and after the beginning of the exposure interval. The preexposure period is defined as April 1, 1991, through March 14, 1993; the postexposure period is defined as March 15, 1993, through March 31, 1995.

The Milwaukee Water Works supplies water to 800 000 residents of the city of Milwaukee and 10 other municipalities in Milwaukee County. In addition, residents of communities within Milwaukee County and the four surrounding counties [illegible] by the Milwaukee Water Works, have frequent opportunities to consume water treated by the water works while working in, or visiting, areas supplied by it. For this reason, mortality estimates were derived for decedents whose death certificate specified residency in a five-county Milwaukee vicinity. The Milwaukee vicinity is defined as Milwaukee, Ozaukee, Racine, Washington, and Waukesha counties.

Wisconsin death certificates list the *International Classification of Diseases*, 9th revision, clinical modification (ICD-9-CM) code for the underlying cause of death and up to 20 contributing causes.[6] Cryptosporidiosis is coded with the code for coccidiosis, ICD-9-CM 007.2, which is also used for infections by the genus *Isospora*.[6] Any death that had ICD-9-CM 007.2 recorded as the underlying or a contributing cause of death on the death certificate is defined as cryptosporidiosis associated. In this study, an acquired immunodeficiency syndrome (AIDS) death is defined as any death that had AIDS (ICD-9-CM 042.0 through 044.9), but not cryptosporidiosis

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(CD-9-CM 007.2), coded as the underlying or as a contributing cause of death.

Data were analyzed with Epi Info Version 6.02 software (Centers for Disease Control and Prevention, Atlanta, Ga). Linear regression analysis was conducted and correlation coefficients, predicted values from linear regression, and confidence intervals for the predicted values were calculated with the use of Confidence Interval Analysis software.

Results

From April 1, 1991, through March 31, 1995, 58 cryptosporidiosis-associated deaths occurred among residents of the Milwaukee vicinity; 4 occurred during the preexposure period, and 54 occurred during the postexposure period (Figure 1). During the same period, 10 cryptosporidiosis-associated deaths occurred among Wisconsin residents living outside the Milwaukee vicinity; 4 occurred during the preexposure period, and 6 occurred during the postexposure period (Figure 1).

Among Milwaukee-vicinity postoutbreak cryptosporidiosis-associated deaths, cryptosporidiosis (ICD-9-CM 007.2) was recorded as the underlying cause of death for 78%; for the remainder, cryptosporidiosis was recorded as a contributing cause (Table 1). AIDS was the underlying cause of death for 85% of postoutbreak cryptosporidiosis-associated deaths among residents of the Milwaukee vicinity. The demographic characteristics of the postoutbreak cryptosporidiosis-associated deaths among residents of the Milwaukee vicinity (Table 2) are consistent with those of persons with AIDS in this area.

During the 3 years prior to the outbreak (April 1990 through March 1993), there was a linear increase in the number of AIDS deaths among residents of the Milwaukee vicinity (r^2 = .88) (Figure 2). If we extrapolate this trend through the postoutbreak period, the number of AIDS deaths predicted during each 6-month interval would be 59 (95% confidence interval [CI] = 52, 66) during April through September 1993; 63 (95% CI = 54, 72) during October 1993 through March 1994; 66 (95% CI = 56, 77) during April through September 1994; and 70 (95% CI = 58, 82) during October 1994 through March 1995.

Among residents of the Milwaukee vicinity, 78 AIDS deaths were identified during the first 6-month postoutbreak interval (April through September 1993)—19 (95% CI = 12, 26) more than predicted from the preoutbreak trend (Figure 2). During the next two 6-month intervals (April through September 1993 and October 1993 through March 1994), the number of AIDS deaths identified (48 and 46, respectively) was significantly lower than predicted. During the last 6-month interval analyzed (October 1994 through March 1995), the 64 AIDS deaths identified were not significantly different from what was predicted by the preoutbreak trend.

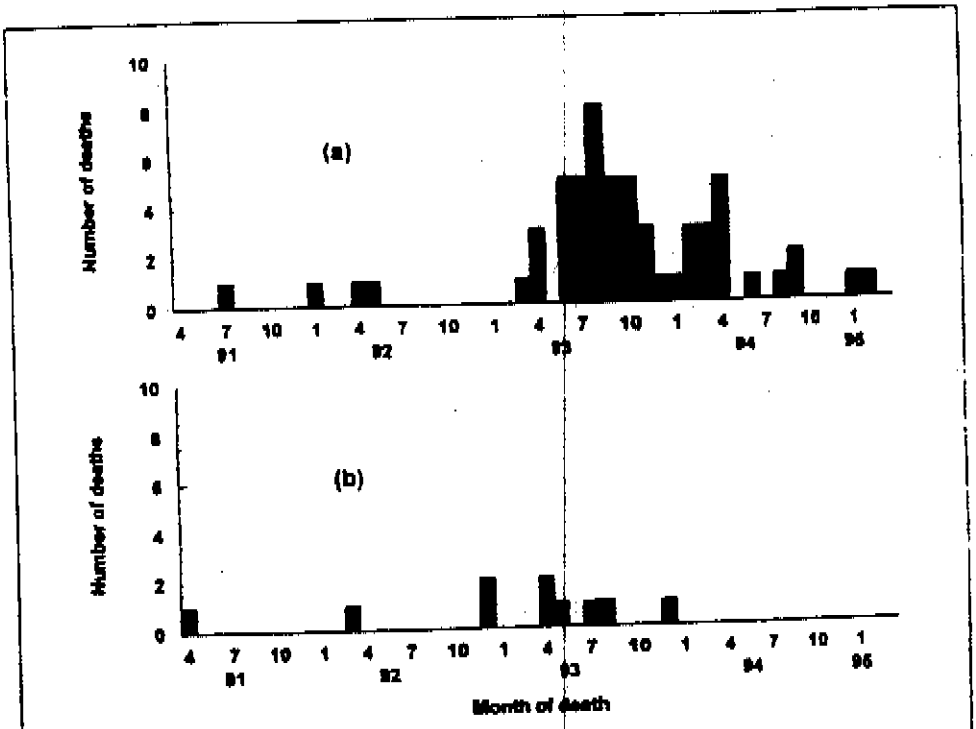

FIGURE 1—Cryptosporidiosis-associated deaths among Wisconsin residents of (a) the 5-county Milwaukee vicinity (n = 58) and (b) the 67 counties outside the Milwaukee vicinity (n = 10), by month of death: April 1, 1991, through March 31, 1995.

Discussion

This analysis indicates that among residents of the Milwaukee vicinity, the number of cryptosporidiosis-associated deaths increased markedly following the waterborne outbreak. Fifty-four cryptosporidiosis-associated deaths occurred during the 2-year postoutbreak period compared with 4 in the 2 years before the outbreak. This represents more than a 13-fold increase in cryptosporidiosis-associated mortality. If, in this population, 4 cryptosporidiosis-associated deaths in 2 years are expected under typical circumstances, then during the 2 years following the outbreak, an additional 50 cryptosporidiosis-associated deaths occurred.

This estimate should be interpreted with caution for several reasons. Death cer-

TABLE 1—Underlying Cause of Death for Cryptosporidiosis-Associated Deaths (n = 54) among Residents of the Milwaukee Vicinity, March 16, 1993, through March 31, 1995

Underlying Cause of Death	ICD-9-CM Code*	No. of Deaths	Percentage of Total
AIDS	042.0–044.9	46	85
Coccidiosis	007.2	4	7
Unspecified viral hepatitis	70.9	1	2
Neoplasm of the brain	239.6	1	2
Heart failure, unspecified	428.9	1	2
Alcoholic cirrhosis of the liver	571.2	1	2

TABLE 2—Demographic Characteristics of Cryptosporidiosis-Associated Deaths (n = 54) among Residents of the Milwaukee Vicinity, March 16, 1993, through March 31, 1995

	No. of Deaths	Percentage of Total
Sex		
Male	49	91
Female	5	9
Race/ethnicity		
White, non-Hispanic	41	76
Black, non-Hispanic	7	13
Hispanic	6	11
County of residence		
Milwaukee	47	87
Racine	2	4
Waukesha	5	9

Note Median age at death was 35 years (range, 1–69 years).

tificate data do not include the date of onset, place of infection, or other information that can definitively link any individual death to exposure to contaminated drinking water from the Milwaukee municipal water supply. Some of the decedents may have been infected with *Cryptosporidium* elsewhere, at a different time, or from a different source.

Because of the large amount of publicity associated with the Milwaukee cryptosporidiosis outbreak, awareness of cryptosporidiosis was higher during the postexposure period than during the preexposure period. This increased awareness resulted in increased testing for *Cryptosporidium* by health care providers and a subsequent increased likelihood that cryptosporidiosis would be listed as a cause of death. If this happened, some of the apparent increase in postexposure mortality could have resulted from increased awareness and thus not represent a true increase in occurrence. While this possibility should be considered, the effect, if any, that this factor had on the mortality estimates cannot be determined from these data.

Another consideration is whether cryptosporidiosis-associated mortality was underreported on death certificates. We analyzed AIDS mortality trends among residents of the Milwaukee vicinity who did not have cryptosporidiosis recorded on their death certificates, and we noted a significant increase in AIDS mortality during the first 6 months after the outbreak, followed by two 6-month intervals with lower-than-expected AIDS mortality, and then a return to expected levels. This pattern is consistent with premature AIDS mortality among persons who would have otherwise died later. These observations suggest that premature mortality was associated with the outbreak and that, at least among persons with AIDS, cryptosporidiosis as a cause of death was underreported on death certificates during the postexposure period.

Estimates of cryptosporidiosis-associated mortality based on death certificate reporting alone should, therefore, be regarded as minimum estimates. It is very likely that additional cryptosporidiosis-related deaths occurred after this outbreak, however, a more precise estimation of the number of additional deaths would require additional studies.

The Milwaukee cryptosporidiosis outbreak was the largest outbreak of waterborne disease ever reported in the United States.[1] Our analysis indicates that this outbreak was associated with a substantial number of deaths, particularly in immunocompromised populations. The Milwaukee population is not unique in its susceptibility to the severe consequences of a waterborne cryptosporidiosis outbreak. In many other metropolitan areas in the United States, the immunocompromised population is considerably larger than in Milwaukee. Indeed, in 1992, just prior to the outbreak, the annual reported AIDS case rate in the Milwaukee metropolitan area ranked 78th among the case rates of 98 metropolitan areas in the United States with populations of 500 000 or more.[8]

Cryptosporidium contamination of surface water is quite common. Studies indicate that *Cryptosporidium* oocysts are present in 67% to 97% of surface waters tested throughout the United States.[9-11] Furthermore, the number of ingested *Cryptosporidium* oocysts required to cause illness is quite low. A recent report noted that the median human infective dose could be as low as 132 oocysts.[12] The ubiquitous nature of this protozoan in surface water, its high infectivity, and the large numbers of individuals at risk of severe disease underscore the potential for fatal outcomes associated with waterborne *Cryptosporidium* outbreaks such as occurred in Milwaukee. To prevent future loss of life from waterborne *Cryptosporidium* outbreaks, it is essential to ensure that all persons have access to safe drinking water.

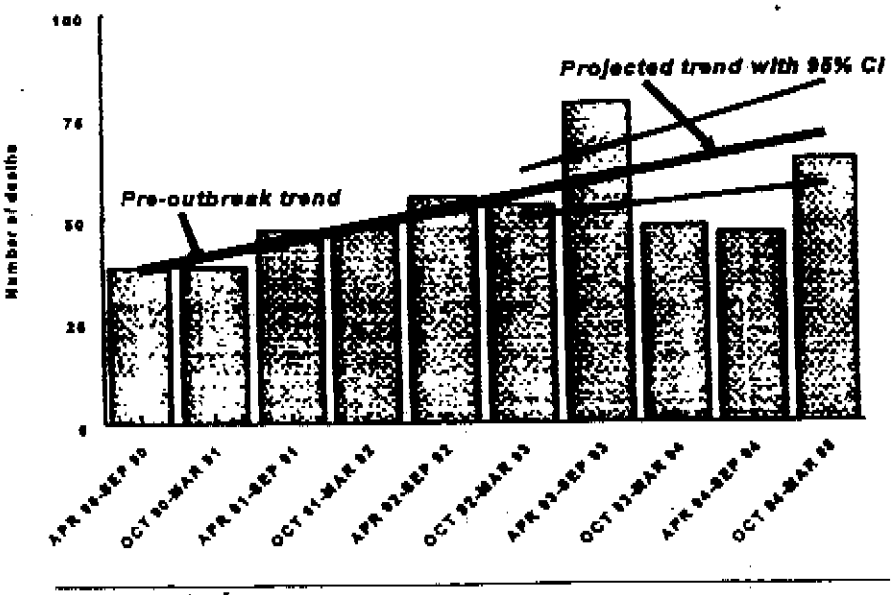

FIGURE 2—AIDS deaths, excluding cryptosporidiosis-associated deaths, among residents of the Milwaukee vicinity, by 6-month interval, and projected linear trend based on deaths from April 1990 through March 1993.

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Predisposing Factors for Individuals' Lyme Disease Prevention Practices: Connecticut, Maine, and Montana

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ABSTRACT

Objectives. This study examined factors that predispose individuals to protect against Lyme disease.

Methods. Knowledge, attitude, and practice questions concerning Lyme disease prevention were included in the Behavioral Risk Factor Surveillance surveys in Connecticut, Maine, and Montana. A total of 4246 persons were interviewed.

Results. Perceived risk of acquiring Lyme disease, knowing anyone with Lyme disease, knowledge about Lyme disease, and believing Lyme disease to be a common problem were significantly associated with prevention practices.

Conclusions. Predisposing factors differ substantially between states and appear related to disease incidence. Personal risk, knowing someone with Lyme disease, and cognizance about Lyme disease and acting on this information are consistent with social learning theories. (*Am J Public Health*. 1997;87:2035–2038)

Introduction

Lyme disease is caused by infection with the spirochete *Borrelia burgdorferi*, acquired from the bite of an infective *Ixodes scapularis* tick in the northeastern and upper midwestern United States or *Ixodes pacificus* in the West.[1] First described in 1977 as a chronic arthritis among children living in Connecticut,[2] Lyme disease has become an important emerging infectious disease over the past decade, accounting for more than 90% of all reported cases of vector-borne illness in the United States.[3] In 1996, 16 461 cases of Lyme disease were reported to the Centers for Disease Control and Prevention (CDC) by 45 state health departments.[4] The overall trend has been an average 15% annual increase in reported cases since 1991, when all 50 states adopted the national Lyme disease case surveillance definition. Although considerable knowledge of the biology and ecology of Lyme disease has been accumulated,[5–11] the prevalence of behavioral risk factors for Lyme disease has not been well defined. No studies have systematically investigated the factors that motivate individuals to take health-directed personal protective measures against Lyme disease. Recommended personal protective measures against tick bites include wearing light-colored clothing, long-sleeve shirts, and long pants; tucking pant legs into socks; using a tick repellent on clothing and exposed skin; or practicing a combination of these.[12–14]

The purpose of this study was to characterize Lyme disease–related knowledge, attitudes, and behavioral risk factors of per-

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