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Cholera Epidemic Associated with Consumption of Unsafe Drinking Water and Street-Vended Water—Eastern Freetown, Sierra Leone, 2012

Von D. Nguyen,* Nandini Sreenivasan, Eugene Lam, Tracy Ayers, David Kargbo, Foday Dafae, Amara Jambai, Wondimagegnehu Alemu, Abdul Kamara, M. Sirajul Islam, Steven Stroika, Cheryl Bopp, Robert Quick, Eric D. Mintz, and Joan M. Brunkard

Division of Foodborne, Waterborne, and Environmental Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia; Disease Control and Prevention, Sierra Leone Ministry of Health and Sanitation, Freetown, Sierra Leone; WHO Representative Office, Sierra Leone World Health Organization Country Office, Freetown, Sierra Leone; Environmental Microbiology Laboratory, International Centre for Diarrhoeal Disease Research, Bangladesh, Dhaka, Bangladesh

Abstract. During 2012, Sierra Leone experienced a cholera epidemic with 22,815 reported cases and 296 deaths. We conducted a matched case-control study to assess risk factors, enrolling 49 cases and 98 controls. Stool specimens were analyzed by culture, polymerase chain reaction (PCR), and pulsed-field gel electrophoresis (PFGE). Conditional logistic regression found that consuming unsafe water (matched odds ratio [mOR]: 3.4; 95% confidence interval [CI]: 1.1, 11.0), street-vended water (mOR: 9.4; 95% CI: 2.0, 43.7), and crab (mOR: 3.3; 95% CI: 1.03, 10.6) were significant risk factors for cholera infection. Of 30 stool specimens, 13 (43%) showed PCR evidence of toxigenic *Vibrio cholerae* O1. Six specimens yielded isolates of *V. cholerae* O1, El Tor; PFGE identified a pattern previously observed in seven countries. We recommended ensuring the quality of improved water sources, promoting household chlorination, and educating street vendors on water handling practices.

INTRODUCTION

Toxigenic *Vibrio cholerae* O1 causes acute, watery diarrhea, and is often fatal without appropriate treatment. During 2000–2009, sub-Saharan Africa reported over 86% of all global cholera cases and over 90% of all global cholera deaths to the World Health Organization (WHO).[1–5] Except for the explosive outbreak of cholera in Haiti that began in 2010, this trend has continued through 2012 when 27 of 48 countries that reported cholera were in sub-Saharan Africa and accounted for 117, 570 (84%) reported cases and 4,183 (91%) cholera deaths, excluding those reported by Haiti and the Dominican Republic.[6] West Africa, one of the continent's poorest regions, has historically experienced a substantial proportion of the continent's cholera burden.[6,7]

Sierra Leone, located on the West African coast, reported four cholera outbreaks from 2000 to 2010. In 2012, the country experienced its largest cholera outbreak since 1995. From January 1 to December 9, 22,815 cholera cases and 296 deaths (case fatality ratio, 1.3%) were reported to the Ministry of Health and Sanitation (MOHS) (Figure 1). Cholera was first identified during early 2012 and the outbreak appeared to be resolving by April 1 (Week 13). The number of reported weekly cases began to accelerate on July 15 (Week 28) and peaked between August 12 and 26 (Weeks 32–34). This phase of the outbreak coincided with the start of the rainy season in late June and the first confirmed case of cholera in Western Area, the seat of the capital Freetown and the most densely populated region in Sierra Leone (Figure 2). From 1990 through 2009, annual rainfall in June has been reported at 30 cm with a peak of 53 cm in August.[8]

Although cholera is not uncommon in Sierra Leone, the alarming scale and pace of the outbreak prompted the establishment of a WHO Cholera Command and Control Center (C4). As part of the international outbreak response, the Centers for Disease Control and Prevention (CDC) was invited to perform a case-control study to identify risk factors for cholera infection.

METHODS

We conducted a matched case-control study at two cholera treatment units (CTU) in the densely populated communities of Wellington and Kuntorloh in eastern Freetown. Freetown had an estimated population of 991,618 people, one-sixth of the country's inhabitants, residing in a 357 km^2 metropolitan area[9]; these two adjacent urban slums experienced rapid growth during and after the Sierra Leone civil conflict. Water pipes of various sizes were visible above ground with frequent leaks and illegal connections. Average per capita income for citizens of Sierra Leone was US $580 in 2012.[8]

Cases were defined as the first person ≥ 5 years of age with acute watery diarrhea and severe dehydration in a household, who was hospitalized for at least 1 night and treated with intravenous fluids between September 10 and 21, 2012. Consecutive patients admitted to the Wellington and Kuntorloh cholera treatment units were evaluated for recruitment into the study. Cases were excluded if they did not meet the case definition or did not consent to be interviewed. Within 2 days of case enrollment, interviewers proceeded to the case's home to perform household observations. Controls were matched to cases on geography and age group. Control households were randomly chosen after spinning a bottle in front of the case's home. One enumerator proceeded to the second nearest home or compound as directed by the bottle, and another enumerator visited the second home in the opposite direction. If no suitable control existed in the chosen household, the enumerator proceeded to the next nearest home in the same direction and continued in this manner until an appropriate control was found. Households were eligible for control recruitment if

* Address correspondence to Von D. Nguyen, Division of Foodborne, Waterborne, and Environmental Diseases, Centers for Disease Control and Prevention, 1600 Clifton Rd. NE, MS - A38, Atlanta, GA 30333. E-mail: dly1@cdc.gov

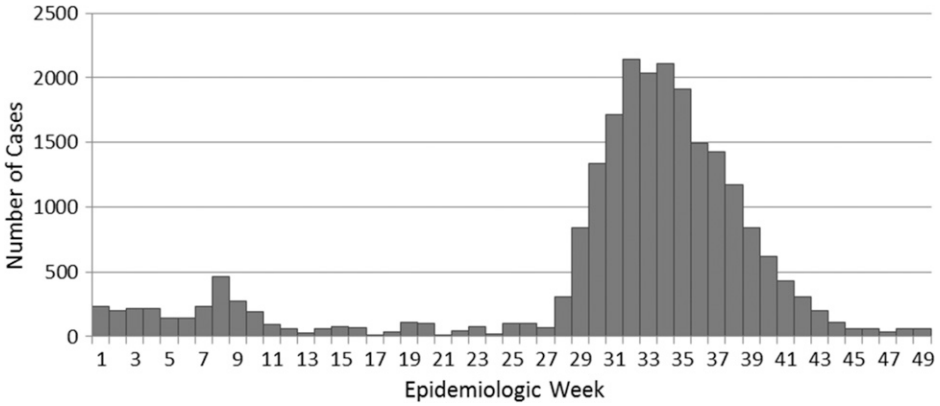

FIGURE 1. Epidemic curve for *Vibrio cholerae* outbreak, Sierra Leone, January 1–December 9, 2012 (N = 22,815).

there was no history of diarrheal illness since June 23, 2012, the date of the first confirmed cholera case in Freetown. In eligible households, controls were enrolled if they belonged to the same age group (5–15, 16–30, 31–45, and over 45 years of age) as the case, lived at the residence for the 5 days before symptom onset in the matched case-patient, and consented to participate in the study.

A standardized questionnaire was developed from the results of hypothesis-generating interviews conducted with hospitalized cholera patients, and was used to interview all case-patients and controls. Enumerators were trained before launching the study to ensure consistent administration of the questionnaire. As part of the emergency public health response to this outbreak, this study did not require human

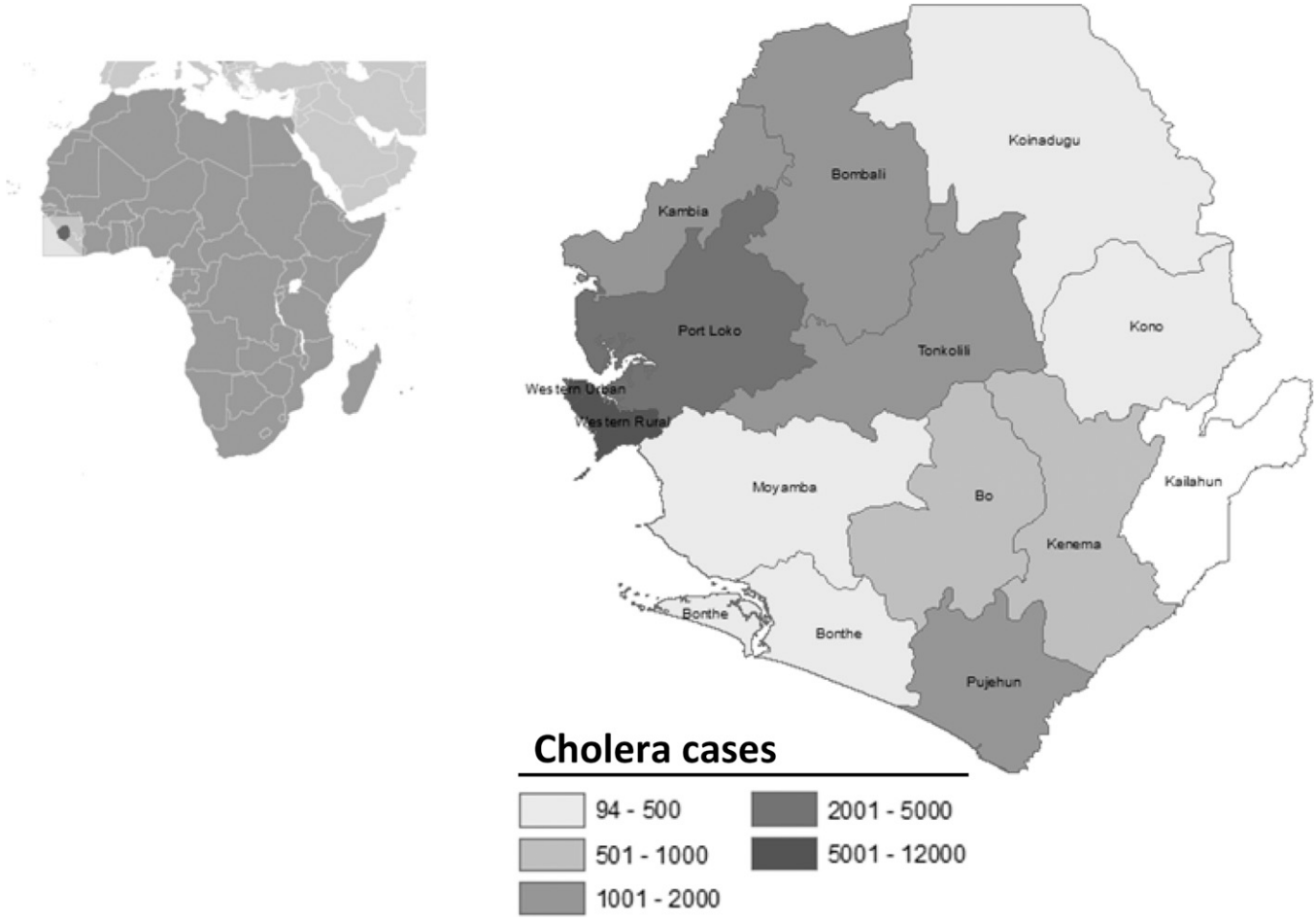

FIGURE 2. Cholera case distribution by district as of October 5, 2012. Source: World Health Organization.

subjects review. Verbal informed consent was obtained from all cases and controls. Case-patients were asked about exposures in the 5 days before illness onset, and controls were asked about exposures in the same 5-day period as the matched case-patient. Subjects were asked about water exposures in an open-ended format to identify all drinking water sources during the exposure period. Drinking water sources were categorized as improved or unimproved using definitions from the WHO/United Nations Children's Fund (UNICEF) Joint Monitoring Program (JMP) for Water Supply and Sanitation.[10]

Subjects were initially asked if they drank unsafe water within the exposure period. During the questionnaire piloting phase, however, the team learned that participants did not consistently understand the meaning of unsafe water. Radio-based social messaging campaigns in response to the outbreak informed the study population that water needed to be treated with chlorine to make it safe to drink. In this context, enumerators suggested that untreated water provided the best translation for unsafe water, and the questionnaire was altered to ask if participants drank unsafe or untreated water in the 5 days before illness onset. The questionnaire also assessed food exposures and participation in activities that had previously been identified as cholera risk factors, such as preparing a body for burial,[11] and attending funerals[11,12] or social events.[13]

Demographic data were collected from all survey participants. Cholera prevention knowledge was assessed among controls; knowledge of prevention measures and drinking water chlorination were not assessed at case households because case-patients received cholera education during hospitalization and chlorine tablets upon discharge. These activities would introduce bias into a comparison between cases and controls. Household observations were conducted, and stored household drinking water was tested for detectable free chlorine residual using the *N*,*N* diethyl-*p*-phenylenediamine (DPD) method (Hach Co., Loveland, CO). The DPD method was also used to test a convenience sample of public water sources, including three public taps and one spring, for detectable free chlorine residual.

All case-patients were asked to provide a stool sample during hospitalization. Stool samples were tested for *V. cholerae* using Crystal *Vc* rapid diagnostic tests (Crystal VC, Code No. 25995A, Span Diagnostics Ltd., Surat, India), and cultured using standard methods at the Sierra Leone Central Public Health Reference Laboratory and the CDC Enteric Disease Laboratory Branch (EDLB).[14] The EDLB also performed polymerase chain reaction (PCR) to confirm the identification, the biotype, and the presence of cholera toxin genes, and antimicrobial susceptibility testing using disk diffusion and pulsed-field gel electrophoresis.[15,16]

Data were entered into an Epi Info 7 (CDC, Atlanta, GA) database and cleaned and analyzed using SAS version 9.3 (SAS Institute, Cary, NC). Exposures were analyzed using conditional logistic regression Firth estimation. Firth estimated matched odds ratios (mOR) and penalized likelihood 95% confidence intervals (CIs) are reported. In simple logistic regression models, exposures with Wald χ^2 P values < 0.10 were considered for inclusion in the multivariable logistic regression model. All eligible exposures were included in the model and eight variables were excluded until all remaining variables were significant at $P < 0.05$. Interactions and confounding were assessed. Additional Firth penalized likelihood estimations were performed on the subgroup of 13 PCR-positive cases.

RESULTS

We enrolled 49 cases and 98 age- and neighborhood-matched controls. The median age was 23 years (5–50) for cases and 25 years (6–77) for controls. Cases and controls were similar in gender and household size. A higher percentage of controls than cases had completed some secondary school but the difference was not statistically significant. Over 95% of participants in both groups used at least one improved water source; however, 20% of cases and 11% of controls also used unimproved drinking water sources. Almost all participants stored their drinking water at home and more than 60% of both groups shared their latrine with other households (Table 1). All case-patients experienced acute watery diarrhea and dehydration requiring intravenous fluids. A majority of case-patients reported nausea and vomiting (89%); other symptoms include fever (47%), loss of consciousness (47%), and leg cramps (33%).

In simple logistic regression models using Firth's penalized likelihood estimations, exposures significantly associated with cholera-like illness included unsafe water, street-vended food and drink, street-vended water, a fermented sugar drink (omolay), palm wine (poyo), and crab. Consuming milk, okra, potato leaf, hot rice, and reheated rice were found to be protective against cholera. No particular street-vended food items were implicated as risk factors for cholera. A number of other food and beverage exposures implicated in previous studies were not significantly associated with cholera infection[17] (Table 2).

In the multivariable logistic regression model, unsafe water, street-vended water, and crab remained significantly associated with cholera infection (Table 3). Street-vended food and drink were no longer significantly associated with cholera in multivariable analysis. Hot rice appeared to be protective but the association did not reach statistical significance. Statistical testing showed no effect modification between variables. Confounding with education was not evident.

To better understand water quality, stored drinking water was tested in 76 control households; of these, 30 samples (40%) showed the presence of free chlorine residual. Testing of four public water sources in the study area showed no evidence of chlorination.

TABLE 1

Demographic and household characteristics of cholera case-control study participants, Sierra Leone 2012

Characteristic	Case* ($N = 49$)	Control* ($N = 98$)	P value†
Female	24 (49%)	56 (57%)	0.35
Some secondary school	21 (47%)	53 (59%)	0.18
Used improved water source	47 (96%)	94 (96%)	1.0
Used improved and unimproved water source	10 (20%)	11 (11%)	0.13
Stored drinking water	42 (89%)	93 (95%)	0.22
Shared latrine with other households	29 (62%)	62 (65%)	0.74
Median household size (range)	7 (2–27)	7 (1–24)	

*For each variable, participants for whom no information was available were excluded.
†χ^2 test for association.

TABLE 2

Firth's penalized likelihood estimations of exposures evaluated during cholera outbreak, Sierra Leone 2012

Characteristic	Case* (N = 49)	Control* (N = 98)	mOR	95% CI
Crab	21 (43%)	25 (26%)	2.14†	(1.05–4.6)
Alcoholic beverage	7 (14%)	7 (7%)	2.00	(0.68–6.0)
Lime	24 (50%)	38 (39%)	1.71	(0.84–3.6)
Milk	27 (55%)	70 (71%)	0.48†	(0.23–0.98)
Okra	20 (42%)	58 (59%)	0.47†	(0.21–0.99)
Omolay (fermented drink)	6 (13%)	1 (1%)	8.67†	(1.8–83)
Potato leaf	41 (85%)	94 (96%)	0.22†	(0.06–0.74)
Poyo (palm wine)	6 (13%)	2 (2%)	5.20†	(1.3–28)
Cold rice	20 (41%)	31 (32%)	1.46	(0.72–2.9)
Hot rice	45 (92%)	96 (99%)	0.17†	(0.02–0.90)
Reheated rice	23 (47%)	71 (73%)	0.35†	(0.17–0.71)
Sugar cane	4 (8%)	9 (9%)	0.93	(0.26–2.9)
Vended food/drink	42 (86%)	50 (51%)	5.11†	(2.3–13)
Vended water	38 (78%)	36 (37%)	5.99†	(2.7–16)
Unsafe water	30 (71%)	41 (43%)	4.55†	(2.0–12)
Contact with cholera patient	8 (17%)	8 (9%)	2.22	(0.78–6.6)
Attended funeral	7 (15%)	13 (13%)	1.12	(0.40–2.9)
Attended wedding	8 (17%)	18 (19%)	0.86	(0.34–2.0)

*For each variable, participants for whom no information was available were excluded.
†Statistically significant at $P < 0.05$.
mOR = matched odds ratio; CI = confidence interval.

The investigation team asked a number of questions among control households to assess knowledge of cholera in the community. When asked "How can you prevent cholera?": 79 (81%) controls responded with "wash your hands," 66 (67%) stated "drink treated water," 38 (38%) responded "eat properly heated food," and only 1 (1%) believed that "cholera cannot be prevented." When asked "How do you get cholera?": 73 (74%) responded "by eating contaminated food" and 69 (70%) stated "by drinking contaminated water." Among 47 control participants who were "making their drinking water safe," 41 (87%) reported that they chlorinated or boiled their water. Effective water filters were not readily available in the study population.

Stool samples were collected from 30 cases; 15 were positive for *V. cholerae* serogroup O1 by a commercial rapid diagnostic test and 6 of these yielded isolates of *V. cholerae* O1, serotype Ogawa, biotype El Tor. The PCR showed that 13 of the 15 RDT positive samples possessed molecular evidence of toxigenic *V. cholerae* O1 biotype El Tor. Antimicrobial susceptibility testing of the six isolates showed resistance to trimethoprim-sulfamethoxazole and susceptibility to nalidixic acid, tetracycline, ciprofloxacin, and doxycycline inferred from tetracycline susceptibility. Pulsed-field gel electrophoresis (PFGE) analysis exhibited a pattern that had previously been seen in seven countries: Angola, Guinea-Bissau, Togo, Kenya, Tanzania, Bangladesh, and India.

TABLE 3

Conditional multivariable logistic regression analysis of risk factors associated with cholera infection, Sierra Leone 2012

Characteristic	mOR	95% CI
Crab	3.29*	(1.03–10.56)
Okra	0.49	(0.13–1.81)
Hot rice	0.04	(0.002–1.24)
Vended water	9.7*	(2.01–43.72)
Unsafe water	3.43*	(1.07–11.04)
Secondary education	0.47	(0.113–1.91)

*Statistically significant at $P < 0.05$.
mOR = matched odds ratio; CI = confidence interval.

TABLE 4

Firth's penalized likelihood estimations of PCR positive subgroup, Sierra Leone 2012

Characteristic	Case* (N = 13)	Control* (N = 26)	mOR	95% CI
Crab	8 (62%)	10 (38%)	2.2	(0.64–9.6)
Milk	7 (54%)	20 (77%)	0.31	(0.05–1.4)
Okra	6 (46%)	15 (58%)	0.66	(0.17–2.4)
Omolay (fermented drink)	4 (33%)	0 (0%)	18†	(1.9–2,385)
Potato leaf	13 (100%)	25 (96%)	1.6	(0.08–219)
Poyo (palm wine)	4 (33%)	0 (0%)	18†	(1.9–2,385)
Reheated rice	8 (62%)	21 (81%)	0.46	(0.12–1.7)
Vended food/drink	13 (100%)	15 (58%)	18.4†	(2.1–2,420)
Vended water	7 (64%)	9 (36%)	33.8†	(4.3–4,359)
Unsafe water	13 (100%)	8 (31%)	4.7	(0.90–47)

*For each variable, participants for whom no information was available were excluded.
†Statistically significant at $P < 0.05$.
mOR = matched odds ratio; CI = confidence interval.

Analysis among the 13 PCR-positive cases and their matched controls, showed that exposure to omolay, poyo, street-vended food and drink, and vended water were significantly associated with cholera (Table 4). The small sample size, however, did not allow for credible multivariable logistic regression modeling.

DISCUSSION

Despite near universal access to improved water sources among cases and controls, use of these sources was not protective or sufficient to prevent epidemic cholera transmission. The WHO/UNICEF JMP confirmed the near universal availability of improved water sources with its 2010 water supply and sanitation estimates showing 83% coverage for urban areas in Sierra Leone.[18] Our analysis identified drinking unsafe or untreated water as a significant risk factor for cholera. The JMP recognized that water sources classified as improved may not consistently provide water that is safe to drink and free of fecal material.[19] A recent report addressed the discrepancy between improved and microbiologically safe water sources, suggesting that although the JMP estimated only 780 million people still used unimproved water sources, the true number of people who face a "significant sanitary risk" may be as high as 1.8 billion.[20]

Our investigation suggested three ways in which improved water supplies in Freetown could have been rendered unsafe. First, disruption of the municipal water system with frequently observed leaking pipes, widespread illegal connections for household use, and frequent power outages may have resulted in reduced pressure and back siphonage, creating opportunities for the introduction of fecal contamination into the distribution system before water reached downstream taps. Second, water from improved sources may not have been adequately chlorinated to sustain effective chlorine residual levels throughout the distribution system. Third, drinking water could have been contaminated during transport to homes or during household storage. Investigations of cholera outbreaks in other settings showed that pathogenic microbes entered piped water networks through cross-contamination with sewer pipes, and clandestine connections allowed entry of surface contaminants.[13,21] In urban areas such as Freetown, categorizing public taps as

an improved water source could create a false sense of security if water infrastructure is poorly maintained. A classification system such as the rapid assessment of drinking-water quality (RADWQ), which measures water safety using microbiological and chemical standards, may provide a more meaningful assessment of drinking water safety.[19] In circumstances where microbiological assessments cannot be performed, protection against waterborne pathogens such as *V. cholerae* could be improved by assuring adequate chlorination of piped water and promoting treatment of water stored in the home.

In response to the outbreak, water, sanitation, and hygiene (WASH) strategies by the government and non-governmental organizations included educating the public about household chlorination and distributing chlorine tablets throughout Freetown. A majority of the study population understood that cholera was transmitted by contaminated water and could be prevented by drinking water treated with chlorine. Chlorination of stored drinking water in 40% of control households after a month of intensive public messaging indicated that part of the population responded to water treatment recommendations. Programs to distribute water treatment products to households and efforts to chlorinate water collected from public sources have been shown to be effective in epidemic settings.[22,23]

Availability of street-vended foods was ubiquitous in Freetown, the largest city in Sierra Leone, where a majority of both cases (86%) and controls (51%) visited street vendors during the exposure period. Although the univariable analysis found "street-vended food and drink" to be a risk factor for cholera, the multivariable analysis showed that this finding was driven by the consumption of street-vended water. Street-vended food and drink have previously been shown to harbor a wide variety of enteric pathogens including Shiga toxin-producing *Escherichia coli*, *Listeria*, *Salmonella*, and *V. cholerae*.[24–27] Prevention of food- and waterborne disease from street vended food and beverages requires that vendors have access to safe water, hygiene, and sanitation facilities combined with information, education, and communication on safe food and water handling practices.[28] Instituting these activities in dense urban areas where street-vended foods are widely available and regularly consumed by much of the population may help prevent future cholera outbreaks.

Crab was the only food exposure significantly associated with cholera-like illness in multivariable analysis. Shellfish provide a natural reservoir for *V. cholerae* and have been implicated in previous cholera outbreak investigations, particularly inadequately cooked shellfish or shellfish that was cross-contaminated with *V. cholerae* during storage.[29,30] Although not statistically significant, the multivariable matched OR for hot rice was 0.04 (CI 95% [0.002–1.24]), suggesting a protective effect. This finding is consistent with previous studies demonstrating the protective effect of hot foods and the risk associated with eating inadequately heated rice, an effective substrate for promoting *V. cholerae* growth.[31] Ensuring that food is well cooked and served hot, and promoting safe food handling practices to prevent cross-contamination can help decrease the risk of cholera infection.

This investigation was subject to several limitations. First, the case definition was sensitive, and likely captured diarrhea cases caused by organisms other than *V. cholerae*. Of 31 stool samples, only 13 showed PCR evidence of *V. cholerae* infection. Even with the small sample size, Firth's penalized likelihood analysis of the PCR-positive sub-group showed that street-vended water remained a significant risk factor, a primary finding of the full analysis. If the PCR-positive sub-group was larger, other risk factors may have shown statistical significance consistent with the full analysis. Second, the questionnaire consolidated the concepts of unsafe water and untreated water into one question, which made it difficult to determine how the unsafe water question was interpreted by participants. The survey, however, showed that half of the control population treated their drinking water and appropriately identified chlorination or boiling as methods to treat drinking water. This suggests that a substantial portion of the study population may have correctly understood the distinction between safe and unsafe water. Third, because we were only able to obtain a small number of water samples from a convenience sample of households and public sources, our results were not necessarily representative of water quality in Freetown. The water samples, however, were collected from different sources in the study area. Finally, the study was conducted in densely populated urban slums in Freetown. Although these communities were the most highly affected, results may not be representative of other areas within Freetown or populations outside the capital city.

In 2011, the United Nations Population Division estimated that 40% of Africa's population lived in urban areas. By 2030, this number is expected to approach 48%, and by 2050, Africa's growing urban population will reach 58%. Although Sierra Leone's country-wide attack rate was 0.41%, Western area, the most populous urban district that includes Freetown, experienced an attack rate of 0.95% (Sierra Leone Cholera Command and Control Center, unpublished data). The conditions that existed within Freetown—overcrowding, poor water infrastructure, an abundance of street-vended food, and a scarcity of good sanitation and hygiene facilities, are not unique to this West African capital city. The JMP classification system for improved water sources that does not account for microbiological quality may be inadequate in these urban settings where highly accessible public taps are connected to an old and deteriorating water infrastructure that has not kept pace with population growth. Point-of-use chlorination provides an effective short-term solution to the public health problem of contaminated drinking water, but long-term investment in piped, adequately maintained water networks, sewage systems, and safe hygiene practices must be made to prevent future outbreaks of cholera.

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Authors' addresses: Von D. Nguyen, Nandini Sreenivasan, Tracy Ayers, Steven Stroika, Cheryl Bopp, Robert Quick, Eric D. Mintz,

and Joan M. Brunkard, Division of Foodborne, Waterborne, and Environmental Diseases (DFWED), Centers for Disease Control and Prevention (CDC), Atlanta, GA, E-mails: dly1@cdc.gov, vii2@cdc.gov, eyk6@cdc.gov, fru3@cdc.gov, cab4@cdc.gov, rxq1@cdc.gov, edm1@cdc.gov, and feu4@cdc.gov. Eugene Lam, Global Immunization Division, CDC, Atlanta, GA, E-mail: vif4@cdc.gov. David Kargbo, Foday Dafae, Amara Jambai, and Abdul Kamara, Disease Control and Prevention, Sierra Leone Ministry of Health and Sanitation, Freetown, Sierra Leone, E-mails: dkkargbo@yahoo.com, fodaydafae@yahoo.co.uk, amarajambai@yahoo.com, and abdulatkamara@gmail.com. Wondimagegnehu Alemu, WHO Representative, Uganda World Health Organization Country Office, Kololo, Kampala, Uganda, E-mail: alemuw@ug.afro.who.int. M. Sirajul Islam, Environmental Microbiology Laboratory, International Centre for Diarrhoeal Disease Research, Dhaka, Bangladesh, E-mail: sislam@icddrb.org.

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