Outbreak of Leptospirosis among Triathlon Participants and Community Residents in Springfield, Illinois, 1998

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We investigated an outbreak of leptospirosis among athletes and community residents after a triathlon was held in Springfield, Illinois. A telephone survey was conducted to collect clinical information and data on possible risk factors, community surveillance was established, and animal specimens and lake water samples were collected to determine the source of the leptospiral contamination. A total of 834 of 876 triathletes were contacted; 98 (12%) reported being ill. Serum samples obtained from 474 athletes were tested; 52 of these samples (11%) tested positive for leptospirosis. Fourteen (6%) of 248 symptomatic community residents tested positive for leptospirosis. Heavy rains that preceded the triathlon are likely to have increased leptospiral contamination of Lake Springfield. Among athletes, ingestion of 1 or more swallows of lake water was a predominant risk factor for illness. This is the largest outbreak of leptospirosis that has been reported in the United States. Health care providers and occupational and recreational users of bodies of freshwater in the United States should be aware of the risk of contracting leptospirosis, particularly after heavy rains.

In mid-July 1998, public health authorities from the Wisconsin Department of Health (WDOH) were notified of 3 Wisconsin athletes who had been hospitalized with an acute febrile illness. The 3 athletes had participated in a triathlon (competitive swimming, cycling, and running events) held in Springfield, Illinois, on 21 June 1998. All 3 athletes presented with headache, myalgias, elevated liver enzyme levels, and hematuria. Two

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had thrombocytopenia, and 1 had laboratory evidence of acute renal failure. Because of leptospirosis awareness among WDOH authorities, serum samples were obtained from 2 of the 3 hospitalized patients and were immediately tested for leptospirosis at the Centers for Disease Control and Prevention (CDC; Atlanta). One serum sample tested positive for leptospirosis by ELISA [1, 2]. After test results were obtained, the Illinois Department of Public Health and the CDC issued a media advisory to alert the 876 triathlon participants and their health care providers of a possible leptospirosis outbreak. In addition, the Springfield Department of Public Health (SDPH) posted health advisories to discourage recreational use of Lake Springfield.

We subsequently conducted a telephone survey to characterize the illness and to identify its risk factors. Because Lake Springfield is heavily used for recreational

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purposes, surveillance systems were established to identify community residents with leptospirosis. An environmental investigation was undertaken to identify the source for and the extent of leptospiral contamination of Lake Springfield.

PATIENTS AND METHODS

Telephone survey. Contact information for and race times of the triathlon participants were obtained from the triathlon organizer. Athletes who resided in 44 US states, Canada, and Austria were contacted by telephone and were also queried, by use of a standardized questionnaire, about presence of a flulike illness, triathlon preparation activities, use of various competition-related products, sources of food and water ingested during the triathlon, quantity of lake water ingested during the triathlon swimming event, presence of abrasions, and triathlon participation before or after 21 June 1998. Participants were encouraged to seek medical care for illness, and arrangements were made to have their clinical specimens sent to the CDC for leptospirosis testing.

We defined a "suspected case" of leptospirosis as onset of fever that occurred from 21 June through 31 July 1998 in a participant of the Springfield triathlon who had ≥ 2 of the following symptoms or signs: headache, chills, myalgias, diarrhea, red eyes, or eye pain. All serum samples obtained from triathlon participants were screened by IgM ELISA (PanBio) [3]; samples with a positive ELISA result (regardless of whether they were acute- or convalescent-phase samples) underwent a microagglutination test (MAT) [4, 5]. In addition, serum samples obtained from athletes whose signs and symptoms met the definition of a suspected case of leptospirosis were also tested by an MAT, regardless of ELISA screening results. We defined a "laboratory-confirmed case" of leptospirosis as a suspected case for which 1 or more positive results were obtained by use of the following laboratory tests: ELISA, MAT, culture, or immunohistochemical (IHC) analysis of tissue [6, 7].

Community investigation. Passive surveillance for leptospirosis among community residents involved the collection of information, by telephone, from residents or from physicians reporting febrile illness consistent with leptospirosis. Active surveillance involved contacting physicians and infection control nurses from the emergency departments of all 3 area hospitals and requesting that persons with possible cases of leptospirosis be referred to the SDPH. The SDPH personnel administered a standardized questionnaire to symptomatic residents; serum specimens that were submitted were screened by ELISA and then underwent MAT. We defined a "suspected community case" of leptospirosis as fever that occurred in a resident of Springfield, Illinois, who had exposure to Lake Springfield in June or July 1998 and who also had ≥ 2 of the following symptoms or signs: headache, chills, myalgias, diarrhea, red eyes, or eye pain. Residents with a "laboratory-confirmed case" were defined as residents of Springfield who had lake exposure during June or July 1998 and who received 1 or more positive results of leptospirosis testing by ELISA, MAT, culture, or IHC analysis of tissue.

Laboratory methods. All serum samples obtained from humans were tested at the CDC. IgM antibody that is detectable by ELISA is usually present within 7–10 days after the onset of illness [3]; the test result for a sample was positive if \geq 10 PanBio units were detected. The CDC confirmatory MAT test for samples obtained from humans uses live *Leptospira* organisms to identify antigen-antibody agglutination across a panel of 21 different serovars [5]. For humans, a single serum sample with a titer of \geq 1:400 or a 4-fold increase in the titer measured in acute- and convalescent-phase serum samples was considered a positive test result.

Blood and urine samples obtained for culture were inoculated in specific media [4, 5] and were held at the CDC for a minimum of 6 months. Isolates recovered from humans were then identified by PCR and mapped restriction-site polymorphism analysis performed by the Zoonotic Diseases Research Unit of the US Department of Agriculture (USDA; Ames, Iowa). To determine the pathogenic species of the genus Leptospira by PCR, the primers G1/G2 and B64-I/B64-II were used [8]. For mapped restriction-site polymorphism analysis, target DNA was amplified using primers 16S-1507/16S-11 [9], which amplify a portion of the 16S rRNA gene of Leptospira organisms. Amplified products were sequenced at the DNA Sequencing Facility of Iowa State University in Ames. Consensus sequences for each isolate were compared with the mapped restriction sites in the 16S rRNA genes for various pathogenic Leptospira species.

IHC staining of gallbladder tissue was performed by use of silver staining techniques with a mixture of reference rabbit polyclonal antisera that were reactive with 16 leptospiral strains [6, 7]. Leptospiral antigens were seen as intact *Leptospira* organisms, threadlike filaments, and granular forms [10].

Environmental investigation and animal diagnostic testing. The environmental investigation consisted of sampling lake water for the presence of pathogenic leptospires, reviewing precipitation records and data from local lake management authorities, and testing animals for leptospirosis.

Lake Springfield, a man-made lake of 17.5 billion gallons of water, was created by the dam on the Sugar Creek; it has a surface area of ~4000 acres and 57 miles of shoreline. The lake is located in the heavily used agricultural area of Illinois and receives runoff from many local livestock farms. It supplies water for Springfield and surrounding towns and is used for public recreation. Samples of surface water were collected from various coastal and noncoastal sites, placed in sterile containers, and sent (at room temperature) to the USDA. Water samples

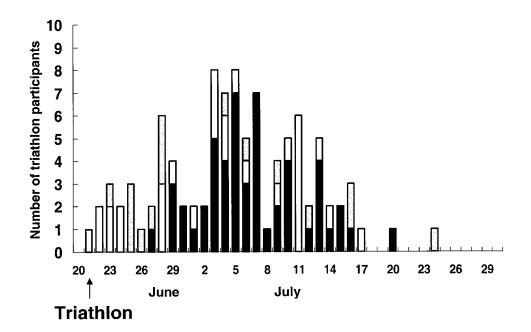


Figure 1. Onset of illness among triathlon participants who met the definition for a suspected case of leptospirosis, by laboratory tests results, Springfield, Illinois, 21 June 1998. Black bars denote laboratory-confirmed cases (n = 52). White bars denote cases that were not laboratory confirmed and for which 0–1 sample was tested (n = 30). Gray bars denote cases that were not laboratory confirmed and for which 2 samples were tested (n = 16). See the Patients and Methods section of the text for case definitions.

were processed for PCR with probes for pathogenic leptospires [8]. Water samples were also injected into hamsters; these animals were subsequently killed, and samples of their blood were collected for culture for leptospira.

Serum samples were obtained from swine, horses, and cattle residing on farms located in the watershed of Lake Springfield and from some of the animals (e.g., goats, llamas, zebu, and donkeys) found in a small zoo located on a stream that feeds into the lake. When dogs living in the community of Springfield were seen by veterinarians for any reason, blood samples were obtained from the dogs. All blood, urine, and tissue samples obtained from animals, except for those obtained from dogs, were convenience samples. At the USDA, serum samples from animals were tested by an MAT using a panel of 6 serovars (C.A.B., personal communication). An MAT titer ≥ 100 was considered a positive result; however, for animals who had previously been vaccinated against leptospirosis, these titers were considered "protective" and were not indicative of active infection.

In addition to testing samples obtained from domestic and exotic animals, the USDA (1) performed MAT on serum samples obtained from such trapped wild animals as raccoons, opossums, muskrats, and deer, and (2) performed fluorescent antibody-antigen detection assays on tissue samples obtained from the same types of wild animals. If the animal was killed or if the sample was obtained from a road-killed animal, kidney and urine samples were also obtained.

Statistical analysis. Univariate RRs were calculated by use

of the SAS procedure GENMOD (version 6.12, SAS Institute). The SAS procedure LOGISTIC was used to perform stepwise unconditional logistic regression. Neither significant interactions nor multicollinearity was found among the variables included in our final multivariable models.

RESULTS

Telephone survey. We interviewed 834 (95%) of 876 participants in the Springfield triathlon; 98 athletes met the definition for a suspected case (attack rate, 12%) (figure 1). The median age of patients with a suspected case was 35 years; 98% were white, and 82% were men. No differences in median age, race, or sex were found between patients with suspected cases and athletes who were not ill. For patients with suspected cases and patients with laboratory-confirmed cases, the median incubation time from lake water exposure to onset of fever was 14 and 15 days, respectively (range, 1–34 days and 6–29 days, respectively).

Among patients with suspected cases of leptospirosis and those with laboratory-confirmed cases, the most common symptoms associated with fever were chills, headache, and muscle aches (table 1). Seventy-five (77%) of 98 patients with suspected cases sought medical care, as did 45 (6%) of 736 athletes who were not ill. Twenty-one (40%) of 52 patients with laboratory-confirmed cases were hospitalized; medical records were available for 13 of the hospitalized patients. At admission, all 13 had elevated levels of liver enzymes or bil-

Table 1. Symptoms and signs of leptospirosis, by suspected case definition and laboratory confirmation, in participants in a triathlon held in Springfield, Illinois, on 21 June 1998.

	No. (%) of athletes			No. (%) of athletes			
Symptom or sign	With a suspected case ^a $(n = 98)$	Who were not ill (n = 736)	RR (95% CI)	With a laboratory- confirmed case ^b (n = 52)	Without infection ^c (n = 193)	RR (95% CI)	
Chills	89 (91)	18 (2)	37.1 (23.4–58.8)	51 (98)	4 (2)	47.3 (17.9–124.9)	
Headache	78 (80)	51 (7)	11.5 (8.6–15.2)	47 (90)	16 (8)	10.9 (6.8–17.6)	
Myalgias	74 (76)	42 (6)	13.2 (9.6–18.2)	42 (81)	15 (8)	10.4 (6.3–17.2)	
Eye pain	41 (42)	29 (4)	10.6 (6.9–16.2)	26 (50)	9 (5)	10.7 (5.4–21.5)	
Diarrhea	50 (51)	88 (12)	4.3 (3.2–5.6)	23 (44)	28 (15)	3.1 (1.9–4.8)	
Red eyes	28 (29)	27 (4)	7.7 (4.8–12.6)	19 (37)	8 (4)	8.8 (4.1–19.0)	

^a Triathlon participants with onset of fever during the period from 21 June through 31 July 1998 plus the presence of ≥2 of the following symptoms or signs: headache, chills, myalgias, diarrhea, red eyes, or eye pain.

^b Triathlon participants with a suspected case plus ≥1 positive result on one of the following laboratory assays: ELISA, microagglutination test, tissue immunohistochemistry, or culture.

^c Triathlon participants who did not meet the definition of a suspected case and for whom 2 negative ELISA results were obtained.

irubin, 11 (85%) had thrombocytopenia, 9 (69%) had proteinuria, 8 (62%) had hematuria, and 4 (31%) had elevated creatinine values (up to 6.1 mg/dL). In addition, 7 underwent lumbar puncture, 1 underwent kidney biopsy, and an additional 2 underwent cholecystectomy.

According to univariate analysis, compared with athletes who were not ill, athletes with suspected cases were more likely to have had a swim time of >42 min (RR, 1.8; 95% CI, 1.0–3.5; P = .04) and were also more likely to report having ingested 1 or more swallows of lake water during the swimming event (RR, 3.8; 95% CI, 1.8–7.9; P < .001). Starting position for the swimming event, presence of abrasions, having taken a shower after the race to cool down, and having worn a wet suit or goggles while swimming were not associated with illness (for all, $P \ge .3$). According to multivariable analysis, having ingested "a swallow or more" of lake water was the only variable that was independently associated with illness (table 2).

Of the 98 athletes who met the definition for a suspected case of leptospirosis, 32 (33%) submitted 1 serum sample and 53 (54%) submitted 2 serum samples (table 3). Of these 85 athletes, 52 (61%) had signs, symptoms, and test results that met the definition of a laboratory-confirmed case. The *Leptospira* serovars *grippotyphosa*, *bratislava*, and *djasiman* consistently had the highest MAT reactivity. Of the 736 athletes who were not ill, 192 (26%) submitted 1 sample for testing and 197 (27%) submitted 2 samples. Four athletes who were not ill each submitted 2 samples; although these samples all had positive results by ELISA, none of the corresponding MAT results were positive. The 2 patients with suspected cases who underwent cholecystectomy had positive results on IHC analysis [10].

Community investigation. The SDPH received reports of 293 Springfield residents who met the definition for a suspected community case of leptospirosis. Of the 248 residents (85%)

who had serologic testing performed, 12 (5%) had laboratoryconfirmed leptospirosis (table 3). Reports were also received of 73 individuals whose illness did not meet the definition of a suspected case and who had serologic testing performed; 2 (3%) had laboratory-confirmed illness. The median age of the 14 residents with laboratory-confirmed leptospirosis was 15 years (range, 15–52 years); 13 (93%) were male. Thirteen of 14 residents had infection confirmed by MAT. For 12 of these residents, MAT serovar reactivity was similar to that seen among the triathlon participants (*Leptospira* serovars grippotyphosa, djasiman, and bratislava). Samples obtained from 1 resident had reactivity to *Leptospira* serovar canicola only. The 14th case patient, who had received penicillin intravenously at the time of the onset of symptoms, had leptospirosis confirmed by culture, and all serologic results were negative.

Residents with laboratory-confirmed cases were more likely to be male, to seek medical care, to report having "red eyes" or "eye pain" and "dark urine," and to be hospitalized for their illness, compared with all residents who tested negative for leptospirosis (for all, P < .05). Six of the 14 patients with laboratory-confirmed cases were hospitalized; 2 had documented Jarisch-Herxheimer reactions after having received penicillin intravenously.

The Lake Springfield exposure locations were known for 13 of the residents with laboratory-confirmed illness; 11 (85%) reported having had contact with water near the area where the triathlon swimming event was held. However, residents' exposures to water were widely distributed among locations all over the lake. The predominant water exposures were swimming, jet skiing, and water skiing.

Clinical isolates. Leptospira organisms were isolated from blood samples obtained from 1 athlete and from 2 residents; PCR amplification of target DNA revealed *Leptospira kirshneri.*

Table 2. Multivariable analysis of risk factors identified in a telephone survey of athletes with suspected or laboratory-confirmed leptospirosis who had competed in a triathlon in Springfield, Illinois, on 21 June 1998.

	Percentage of athletes				Percentage of athletes			
Risk factor	With a suspected case ^a $(n = 98)$	Who were not ill (n = 736)	RR (95% CI)	<i>P</i> value	With a laboratory- confirmed case ^b (n = 52)	Without infection ^c (n = 193)	RR (95% CI)	<i>P</i> value
Abrasions ^d	13	9	1.2 (0.7–2.1)	.4	10	10	1.0 (0.4–2.3)	1.0
Swim time >42 min ^e	81	74	1.4 (0.8–2.4)	.3	83	80	1.0 (0.5–2)	1.0
Swallowed lake water more than once	65	45	2.0 (1.3–3.2)	.002	65	48	2.0 (1.1–3.5)	.02

^a Triathlon participants with onset of fever that occurred from 21 June through 31 July 1998 plus the presence of ≥2 of the following symptoms or signs: headache, chills, myalgias, diarrhea, red eyes, or eye pain.

^b Triathlon participants with a suspected case plus ≥1 positive result on one of the following laboratory assays: ELISA, microagglutination test, tissue immunohistochemistry, or culture.

² Triathlon participants who did not meet the definition for a suspected case and for whom 2 negative ELISA results were obtained.

^d Presence or acquisition of abrasions or lacerations during the swimming event of the triathlon.

^e Swim time was divided into quartiles; 42 min denotes the cutoff point between the first quartile (baseline) and the remaining 3 quartiles. Analysis of swim time was performed with time as a linear model.

Mapped restriction-site polymorphism analysis of the 16S rRNA gene confirmed that the *Leptospira* serovar was *grippo-typhosa*. The digestion pattern of the athlete's isolate was identical to that of the isolate of one resident; the pattern of the other resident's isolate showed minor differences.

Environmental investigation. Within 40 days after the triathlon, lake water samples were collected on 3 different days from locations around the triathlon swimming event site, from sites located at the extreme opposite end of the lake, and from sites located between these points. Of the 27 lake water samples that were analyzed, only 1 sample, which was obtained from the shoreline adjacent to the starting point of the triathlon, had a PCR reaction (G1/G2) that was positive for a pathogenic *Leptospira* species; however, no organism was isolated. Two samples were culture positive; however, both isolated organisms were saprophytic *Leptospira* species.

Serum samples were obtained from 205 domestic and exotic animals (128 cattle, 15 pigs, 29 horses, 8 sheep, 6 dogs, and 19 zoo animals) for MAT analysis. Eighty-four (66%) of 128 cattle tested and all 15 pigs had previously received a veterinary *Leptospira* vaccine that contained antigens for the *Leptospira* serovars grippotyphosa, canicola, icterohaemorrhagiae, pomona, hardjo, and bratislava. In the vaccinated animals, all titers >1: 100 corresponded to vaccine serovars. Vaccination status was not available for the dogs; however, only 1 dog had a titer of 1:100 against serovar pomona. None of the horses, sheep, animals from the zoo, or 44 remaining cows had been immunized. Thirty-three (33%) of these 100 unvaccinated animals had MAT titers >1:100; *Leptospira* serovars hardjo, icterohemorrhagiae, and bratislava had the highest reactivity.

Biologic samples (serum, urine, and tissue) were obtained from 60 wild animals (raccoons, muskrats, opossums, and deer); no cultures were positive for *Leptospira* organisms, and no serum samples had titers >1:50. Of the 46 tissue samples examined, 2 kidney tissue specimens (1 from a raccoon and 1 from a deer) were found to be positive for *Leptospira* organisms by IHC analysis.

Precipitation records for the Springfield area in 1994–1998 were reviewed. Amounts of precipitation for the periods from January to June in 1994-1997 were combined, and the mean precipitation (3 inches) was compared with the mean precipitation for the same months in 1998 (5 inches). May was the month of peak precipitation in 1994-1997 (mean, 6 inches). June was the month of peak precipitation in 1998 (10 inches), with more than double the mean for the months of June in 1994-1997 (4 inches) and 5 times more than that for June 1997 (2 inches). Fecal and total coliform counts for Lake Springfield (84 and 800 coliforms per 100 mL of lake water, respectively) were obtained before the 1998 triathlon; these counts were higher than the average counts obtained during the same time period in 1997-1994 (65 and 70 coliforms per 100 mL lake water, respectively). For the month of June 1998, the average water pH, temperature, and raw turbidity were 8.07, 24.6°C, and 20.3 NTU, respectively, compared with values of 8.11, 23.6°C, and 16.3 NTU, respectively, for the months of June in 1994-1997.

DISCUSSION

This investigation documents the largest recognized outbreak of leptospirosis in the continental United States. Before outbreak awareness was established, none of the ill athletes who sought medical care was suspected of having leptospirosis. As a result, many lumbar punctures, 1 kidney biopsy, and 2 cholecystectomies were performed. This not only reflects the lack of familiarity of US health care providers with the symptoms

Table 3. Results of leptospirosis testing performed on participants in a triathlon held on 21 June 1998 in Springfield, Illinois, and on community residents who had \ge 1 serum sample tested.

Test	Athletes with a suspected case ^a (n = 85)	Athletes who were not ill (n = 389)	Residents with a suspected community case ^b (n = 248)	Residents without a suspected community case (n = 73)
Single-sample ELISA	32 (34)	192 (0)	110 (3)	54 (0)
Two-sample ELISA	53 (51)	197 (2)	138 (3)	19 (0)
Single-sample MAT	40 (58)	18 (0)	58 (7)	35 (0)
Two-sample MAT	41 (56)	12 (0)	139 (5)	20 (10)

NOTE. Data are no. of persons tested (% with a positive test result). MAT, microagglutination test.

^a Triathlon participants with onset of fever during the period from 21 June through 31 July 1998 plus the presence of ≥ 2 of the following symptoms or signs: headache, chills, myalgias, diarrhea, red eyes, or eye pain.

^b A resident of Springfield, Illinois, who had fever that occurred after exposure to Lake Springfield in June or July 1998 plus the presence of ≥2 of the following symptoms or signs: headache, chills, myalgias, diarrhea, red eyes, or eye pain.

and epidemiology of leptospirosis but, also, a lack of familiarity with the morbidity associated with this infection

Leptospirosis is a zoonotic disease caused by spirochetes of the genus Leptospira [11, 12]. Many wild and domestic animals are reservoirs for Leptospira organisms, and they shed these organisms in their urine, thereby contaminating freshwater, mud, and soil. Leptospirosis has a worldwide distribution; however, it is more common in tropical climates, where conditions for transmission are favorable [13, 14]. Large epidemics have been recognized in Latin America and the Caribbean after hurricanes, tropical rainstorms, and flooding [6, 7, 15–17], and, as in those previously reported outbreaks, heavy rainfall preceded the outbreak associated with the triathlon in Springfield reported here. Presumably, flooding elevates the water table, allowing the soil to be saturated by subsurface leptospires, preventing the evaporation of contaminated animal urine and fostering the passage and survival of leptospires in surface waters [18].

In the continental United States, leptospirosis is usually detected in veterinarians and farmers. In addition, small outbreaks have been documented in individuals who swim in watering holes, streams, or ponds contaminated with infected urine [19, 20]. By concentrating the leptospires present in water, drought or dry weather conditions may contribute to outbreaks that involve small bodies of water. In contrast, Lake Springfield is a large lake that may have been contaminated by lake runoff during an unusually rainy season. Despite epidemiologic evidence of widespread leptospiral contamination of the lake, an animal reservoir with the epidemic strain was not identified.

Assays to determine the presence of pathogenic leptospires in bodies of water are labor intensive and are of limited use. Failure to detect pathogenic leptospires in a body of water provides no valid information about water safety; in addition, the public health risk of a positive result is not known, because infection depends on the dose and type of exposure. Thus, screening large bodies of freshwater for leptospires should not guide public health authorities in making decisions regarding the safe recreational use of water.

A disrupted skin barrier is the classically acknowledged route of transmission; however, our survey of athletes found that ingestion of lake water was the prominent risk factor for illness. Previous studies have also found that ingestion of leptospirecontaminated water and food was associated with disease [16, 20–23]; in addition, the finding of leptospires in mesenteric lymph nodes (Dr. Sherif Zaki, personal communication) suggests that the stomach may be an important portal of entry.

The clinical presentation of leptospirosis varies markedly [11] and may include nonspecific symptoms that are easily mistaken for such common febrile illnesses as influenza. Unless clinical suspicion for leptospirosis is high, laboratory diagnosis is rarely pursued. Serologic confirmation usually requires acute-phase and convalescent-phase samples, which can be tested by a variety of assays, none of which is readily available in hospital or clinic laboratories. The gold standard, MAT, is available only in reference laboratories, is labor intensive and costly, lacks sensitivity for many species, and requires extensive technical expertise [4, 5]. In addition, results are not available until long after treatment decisions have been made.

The results of this investigation call for increased leptospirosis awareness among health care providers and recreational or occupational users of bodies of freshwater in the continental United States, especially after heavy rainfalls. Also, with the increase in "ecotourism," during which exposure to bodies of freshwater potentially contaminated with leptospira may frequently occur, the numbers of travelers who return from their travels with leptospirosis may increase [16, 24]. Empiric therapy with doxycycline, ampicillin, amoxicillin, or penicillin [11] should be considered for patients suspected of having leptospirosis after exposure to any outdoor freshwater source. Primary chemoprophylaxis has been shown to be effective [25,

26]; however, few data exist regarding prophylactic treatment after high-risk exposure [27]. Given that the current methods of freshwater testing cannot reliably furnish information that can be translated into effective public health measures, heightened awareness and development of sensitive, rapid, and inexpensive diagnostic assays for leptospirosis are needed.

STUDY GROUP MEMBERS

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References

- Centers for Disease Control and Prevention. Outbreak of acute febrile illness among athletes participating in triathlons—Wisconsin and Illinois, 1998. MMWR Morb Mortal Wkly Rep 1998; 47:585–8.
- Centers for Disease Control and Prevention. Update: leptospirosis and unexplained acute febrile illness among athletes participating in triathlon—Illinois and Wisconsin, 1998. MMWR Morb Mortal Wkly Rep 1998; 47:673–6.
- Winslow WE, Merry DJ, Moira LP, Devine PL. Evaluation of a commercial enzyme-linked immunosorbent assay for detection of immunoglobulin M antibody in diagnosis of human leptospiral infection. J Clin Microbiol 1997; 35:1938–42.
- Sulzer CR, Jones WL. Leptospirosis: methods in laboratory diagnosis. Revised ed. HEW publication (CDC) 80-8275. Washington, DC: US Department of Health, Education, and Welfare, 1978.
- Weyant RS, Bragg SL, Kaufmann AF. *Leptospira* and *Leptonema*. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Yolken RH, eds. Manual of clinical microbiology. 7th ed. Washington, DC: American Society for Microbiology, **1999**:739–45.
- Zaki SR, Spiegel RA. Leptospirosis. In: Nelson AM, Horsburgh CR Jr, eds. Pathology of emerging infections 2. Washington, DC: American Society for Microbiology, 1998:73–91.
- 7. Zaki RS, Shieh W-J. Leptospirosis associated with outbreak of acute febrile illness and pulmonary haemorrhage, Nicaragua, 1995. The Ep-

idemic Working Group at Ministry of Health in Nicaragua. Lancet **1996**; 347:535–6.

- Gravekamp C, Van de Kemp H, Franzen M, et al. Detection of seven species of pathogenic leptospires by PCR using two sets of primer. J Gen Microbiol 1993; 139:1691–700.
- Ralph D, McClelland M, Welsh J, et al. *Leptospira* species categorized by arbitrarily primed polymerase chain reaction (PCR) and by mapped restriction polymorphisms in PCR-amplified rRNA genes. J Bacteriol 1993; 175:973–81.
- 10. Guarner J, Shieh WJ, Morgan J, et al. Leptospirosis mimicking acute cholecystitis among athletes participating in a triathlon. Hum Pathol **2001**; 32:750–2.
- Tappero JW, Ashford DA, Perkins BA. Leptospirosis. In: Mandell GL, Bennet JE, Dolin R, eds. Principles and practice of infectious diseases. 5th ed. New York: Churchill Livingstone, 1999:2495–501.
- Yasuda PH, Steigerwalt AG, Sulzer KR, Kaufmann AF, Rogers F, Brenner DJ. Deoxyribonucleic acid relatedness between serogroups and serovars in the family Leptospiraceae and proposals for seven new *Leptospira* species. Int J Syst Bacteriol **1987**; 37:407–15.
- Faine S. Leptospirosis. In: Evans AS, Brachman PS, eds. Bacterial infections of humans, epidemiology and control. 3rd ed. New York: Plenum Medical Book, 1998:395–420.
- Ko AI, Galvao Reis M, Ribeiro Dourado CM, Johnson WD Jr, Riley RW. Urban epidemic of severe leptospirosis in Brazil. Lancet 1999; 354:820–5.
- Bruce MG, Sanders EJ, Leake JAD, et al. Leptospirosis among patients with dengue-like illness in Puerto Rico [abstract for poster]. 47th Annual Meeting of American Society of Tropical Medicine and Hygiene. 1998:59.
- Centers for Disease Control and Prevention. Outbreak of leptospirosis among white-water rafters—Costa Rica, 1996. MMWR Morb Mortal Wkly Rep 1997; 46:577–9.
- Trevejo RT, Rigau-Perez JG, Ashford DA, et al. Epidemic leptospirosis associated with pulmonary hemorrhage—Nicaragua, 1995. J Infect Dis 1998; 178:1457–63.
- Faine S. The etiologic agent. In: Faine S, ed. Guidelines for the control of leptospirosis. WHO offset publication 67. Geneva: World Health Organization, 1982:17–36.
- Jackson LA, Kaufmann AF, Adams WG, et al. Outbreak of leptospirosis associated with swimming. Pediatr Infect Dis J 1993; 12:48–54.
- Corwin A, Ryan A, Bloys W, et al. A waterborne outbreak of leptospirosis among military personnel in Okinawa, Japan. Int J Epidemiol 1990; 19:743–8.
- Crawford RP, Heinemann JM, McCulloch WF, et al. Human infections associated with waterborne leptospires, and survival studies on serotype *pomona*. J Am Vet Med Assoc 1971; 159:1477–84.
- Cacciapuoti B, Ciceroni L, Maffei C, et al. A waterborne outbreak of leptospirosis. Am J Epidemiol 1987; 126:535–45.
- Haunz EA, Cardy JD. Canicola fever: report of nine cases in one family. Arch Intern Med 1952; 89:978–93.
- Centers for Disease Control and Prevention. Public health dispatch: outbreak of acute febrile illness among participants in EcoChallenge Sabah 2000—Malaysia, 2000. MMWR Morb Mortal Wkly Rep 2000; 49:816–7.
- Takafuji ET, Kirkpatrick JW, Miller RN, et al. An efficacy trial of doxycycline chemoprophylaxis against leptospirosis. N Engl J Med 1984; 310:497–500.
- Sehgal SC, Sugunan AP, Murhekar S, et al. Randomized controlled trial of doxycycline prophylaxis against leptospirosis in an endemic area. Int J Antimicrob Agents 2000:13:249–55.
- Gonsalez CR, Casseb J, Monteiro FGV, et al. Use of doxycycline for leptospirosis after high-risk exposure in Sao Paulo, Brazil. Rev Inst Med Trop Sao Paulo 1998; 40:59–61.