An Outbreak of Travel-Associated Legionnaires Disease and Pontiac Fever: The Need for Enhanced Surveillance of Travel-Associated Legionellosis in the United States

Andrea L. Benin,[1,2,a] Robert F. Benson,[2] Kathryn E. Arnold,[3] Anthony E. Fiore,[3,a] Patricia G. Cook,[2] L. Keoki Williams,[3,a] Barry Fields,[2] and Richard E. Besser[2]

[1]Epidemic Intelligence Service and [2]Respiratory Diseases Branch, Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention, and [3]Georgia Division of Public Health, Atlanta

Travel-associated outbreaks of legionnaires disease (LD) and combined outbreaks of LD and Pontiac fever (PF) are rarely identified. During one travel-associated combined outbreak at a hotel, a cohort study of potentially exposed persons and an environmental investigation were performed. Two LD and 22 PF cases were identified. *Legionella pneumophila* serogroup 6 (Lp6) isolates from the index patient and the hotel whirlpool spa were found to be identical by amplified fragment–length polymorphism typing. Disease occurred in 10 of 26 guests who were exposed to the spa versus 2 of 29 guests who were exposed only to the pool area (38% vs. 7%; $P = .005$). Immunoglobulin M (IgM) antibody to the outbreak Lp6 strain was more common among persons with PF (4 of 9) than among non-ill persons (2 of 32) (44% vs. 6%; $P = .02$). Spa exposure correlated with disease ($P = .001$) and IgM seropositivity ($P = .007$). New laboratory techniques facilitate outbreak investigation; to expedite outbreak interruption and measure the impact of travel-associated legionellosis, surveillance must be improved.

It is estimated that 8000–18,000 cases of community-acquired legionellosis occur each year in the United States [1]; the total burden of legionellosis is even higher. Cases of legionellosis take 1 of 2 recognized forms, legionnaires disease (LD) or Pontiac fever (PF) [2–6]. Most reported cases are sporadic; common-source outbreaks are rarely detected.

When outbreaks of legionellosis are detected, most involve LD or PF exclusively; combined outbreaks of both LD and PF are unusual [7–9]. Although LD was first identified as an outbreak among travelers (legionnaires at the 1976 American Legion convention in Philadelphia [6]), outbreaks that occur in travelers are particularly difficult to detect. The number of travel-associated outbreaks is unknown, and reports of travel-associated combined LD and PF outbreaks are limited to 1 published case series [7], 1 large outbreak [8, 10], and 1 outbreak at a convention in Iowa that was reported to the Centers for Disease Control and Prevention (CDC) (D. B. Jernigan, unpublished observations). In general, the burden of travel-associated legionellosis in the United States is not defined.

We investigated a travel-associated LD and PF outbreak that was identified only because the index cases occurred in an extended family from 2 states and because the family members, who had stayed at a Georgia hotel, brought their illness to the attention of public health officials. This outbreak investigation highlighted the challenges associated with detecting and investigating travel-associated disease, documented a combined LD and PF outbreak, used enhanced laboratory techniques for molecular subtyping and serologic testing, and demonstrated how new techniques of molecular characterization may be important in the development of new surveillance methods.

Background

In late May 1999, the Georgia Division of Public Health (GDPH) received a report of a case of LD occurring in a visitor to the state. The patient, a 61-year-old woman, was hospitalized with pneumonia in New York in early May. She required mechanical ventilation, and *Legionella pneumophila* serogroup 6 (Lp6) was isolated from her sputum. Concurrent with the onset of her illness, 4 of her family members in Massachusetts and a

Received 24 May 2001; revised 14 August 2001; electronically published 18 December 2001.

Presented in part: 37th annual meeting of the Infectious Diseases Society of America, Philadelphia, November 1999 (abstract 187); Epidemic Intelligence Service Conference, Atlanta, April 2000 (abstract P3).

Informed consent was obtained from patients or from their parents or guardians. The guidelines of the US Department of Health and Human Services were followed in the use of human specimens.

Financial support: The laboratory portion of this research received support from an appointment to the Emerging Infectious Diseases Fellowship Program administered by the Association of Public Health Laboratories and funded by the Centers for Disease Control and Prevention.

[a] Present affiliations: Robert Wood Johnson Clinical Scholars Program, Yale University School of Medicine, New Haven, Connecticut (A.L.B.); Division of Viral Hepatitis, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia (A.E.F.); Division of General Medicine, University of Michigan Health System, Ann Arbor (L.K.W.).

Reprints or correspondence: Dr. Richard Besser, Centers for Disease Control and Prevention, MS C23, 1600 Clifton Rd. NE, Atlanta, GA 30333 (rbesser@cdc.gov).

The Journal of Infectious Diseases 2002;185:237–43

0022-1899/2002/18502-0013$02.00

family friend in New York (2 adults and 3 children) became ill with fever, diarrhea, vomiting, and malaise. All ill persons had recently traveled to Georgia to attend a wedding and became ill the day of their return. They had not traveled together; their only common exposures were attendance at the wedding and a stay in Hotel A from 23 through 25 April. No other wedding guests became ill, and no other wedding guests had stayed at Hotel A. While staying at Hotel A, all ill family members had used the hotel's whirlpool spa. The GDPH and the CDC initiated an investigation to determine the extent of the illness and the source of transmission.

Methods

Case Finding and Case Definitions

To identify case patients and determine the magnitude of the outbreak, we used hotel records to obtain the names of guests registered at any time from 1 April through 11 May 1999. Health officials from the CDC and the GDPH contacted guests by telephone and administered a standardized 2-page questionnaire to elicit information regarding possible illness and exposure to potential sources of *Legionella* transmission.

A confirmed case of outbreak-associated LD was defined as physician-diagnosed pneumonia and laboratory evidence of *Legionella* infection in a hotel guest who visited the hotel at some point from 1 April through 11 May 1999. Onset of illness must have occurred within 10 days of residence in the hotel. Laboratory evidence consisted of isolation of *Legionella* species from respiratory secretions or a 4-fold rise in Lp6 antibody titers in paired acute- and convalescent-phase serum specimens.

A case of PF-like illness was defined as subjective fever and at least 1 other symptom (diarrhea, vomiting, myalgia, or cough) in a hotel guest who visited the hotel at some point from 1 April through 11 May 1999. Onset of illness must have occurred within 10 days of exposure at the hotel. Laboratory confirmation was not included in the case definition for PF-like illness.

Environmental Investigation

We collected 1-L water samples and biofilm swab specimens from multiple sites at Hotel A, including the swimming pool, the swimming pool filter, the swimming pool skimmer buckets, the whirlpool spa, the whirlpool spa filter, the showerhead in the indoor pool room, all hot-water heaters, and selected hotel guest room water faucets and showerheads. To each water sample, we added 0.5 mL of 1 *N* sodium thiosulfate to neutralize any halogen disinfectant present. We measured bromine concentrations and pH in the swimming pool and whirlpool spa and reviewed hotel records to evaluate past bromination and maintenance.

Cohort Study of Guests Exposed to the Indoor Pool Area

To determine risk factors for disease and to evaluate the use of IgM testing for PF and LD, we performed a cohort study involving all hotel guests who had been exposed to the indoor pool area during the index weekend (23–25 April, the weekend during which the index patient stayed at the hotel). A standardized questionnaire was developed to collect data on the amount of exposure to the pool areas, the swimming pool, and the whirlpool spa and other water sources, as well as recent illness and medical history. Patients' private physicians and local health departments obtained serum samples for antibody studies and urine samples for antigen testing. Specimens were collected from 27 May through 23 June 1999.

Laboratory Methods

Environmental samples. Water samples were filtered and treated with acid. Water and swab specimens were plated onto buffered charcoal yeast extract (BCYE) medium with and without supplemental antibiotics, using standard plating techniques [11]. Inoculated plates were incubated at 35°C and examined at intervals for the presence of colonies resembling *Legionella* species. Suspect colonies were picked and inoculated onto biplates containing BCYE medium with and without L-cysteine. Cultures that required L-cysteine for growth were subcultured and tested with specific antisera to determine the *Legionella* species and serogroup.

Human serum and urine samples. Serum samples were tested for combined IgG, IgA, and IgM (IgGAM), as well as for IgM alone, using an indirect immunofluorescence test. A positive result of testing for IgGAM was defined as a 4-fold rise in levels between acute- and convalescent-phase samples to a titer $\geq 1:128$. A positive result of testing for IgM was defined as a titer $\geq 1:64$. Urine samples were concentrated 25 times and tested by ELISA (Binax) for Lp1.

Preparation of bacterial DNA for amplified fragment–length polymorphism testing (AFLP). The patient isolate, environmental isolate, and 10 strains of Lp6 from the CDC archives (Chicago 2, Johannesburg 5, Albany 1, Oxford 1, SRP39, Denver 3, Sydney 1, LD82-683, ED38, and Vasteras 57/1) were inoculated onto BCYE medium and incubated at 35°C for 24–48 h. These strains represent 10 of the 11 electrophoretic types of Lp6 described by McKinney et al. [12]. Cells were lysed, and DNA was extracted by use of a DNA purification kit (MasterPure; Epicentre Technologies).

Adapters AX1, AX2, and PX-G were constructed as described by Mazurek et al. [13]. In addition, adapters PS1 and PS2 were prepared as described by Riffard et al. [14]. All oligonucleotides were obtained from the Biotechnology Core Facility of the CDC. Adapters were prepared by mixing of equal molar amounts of AX1 and AX2 or PS1 and PS2 in 1% polymerase chain reaction (PCR) buffer (PE Biosystems) and allowed to anneal as the mixture was cooled from 80°C to 4°C over the course of 1 h in a thermocycler [13]. Oligonucleotides PS1 and PX-G were used as primers in the PCR.

A portion of the extracted DNA was digested with 40 U each of *Pst* I and *Xba* I in 1× SuRE/Cut buffer H (Roche Molecular Biochemicals) for 1.5 h at 37°C, followed by incubation at 65°C for 20 min to inactivate the restriction enzymes. All enzymes were obtained from Boehringer Mannheim. The *Xba* I (AX1/AX2) and *Pst* I (PS1/PS2) adapters were ligated onto the restricted DNA sample by use of T4 DNA ligase. The samples were incubated at 16°C for 1 h and then at 65°C for 20 min to inactivate the T4 DNA ligase. The samples were treated with 10 U each of *Xba* I and *Pst* I at 37°C

for 15 min to cleave any restriction sites that were reformed by ligation.

DNA samples with ligated adapters were amplified by use of 25-μL reaction volumes containing 5 μL of sample, 0.5 U of Taq polymerase (PE Biosystems), 200 μM of each dNTP (PE Biosystems), 1 μM each of PS1 and PX-G primers, 1× PCR buffer, 0.5 mM $MgCl_2$, and distilled water. Amplification was done in a GeneAmp PCR System 2400 (PE Biosystems) with a profile consisting of an initial denaturation of 5 min at 94°C, 30 cycles of denaturation at 94°C for 30 s, primer annealing at 60°C for 30 s, and extension at 72°C for 90 s, followed by holding at 4°C.

The PCR products were loaded into wells with a 3.5% MetaPhor agar gel (FMC) containing ethidium bromide in 1× Tris-borate–EDTA buffer and electrophoresed at 90 V. After electrophoresis, the gel was photographed with UV illumination. In addition to gel electrophoretic analysis, a fluorescence-labeled fragment analysis method (ABI Prism 310; PE Applied Biosystems) was used, according to the protocol described by Benson et al. [15].

Statistical Analysis

Data entry and analyses were done with Epi Info software (version 6.04; CDC) and SAS software (version 6.12; SAS Institute). Odds ratios and 95% confidence intervals were calculated to assess categorical exposure variables. Dose-response relationships were compared using the χ^2 test for trend analysis. Age-group differences and water exposure times were compared using the Wilcoxon rank sum test. For all statistical tests, $P \leq .05$ was considered to be significant; all P values reported are 2-sided.

Results

Epidemiologic Investigation

Hotel. Hotel A is located in a suburb of Atlanta and has 143 rooms. Seventy percent of the hotel guest rooms were occupied during the index weekend. The hotel swimming pool is located partially indoors and partially outdoors. The indoor section is located in the indoor pool room, which also contains the whirlpool spa, an open shower area, a water fountain, and several chairs and tables. The indoor pool room measures 19.2 × 8.1 m. In the far corner of the indoor pool room, a door leads to a maintenance room that contains filtration units for the pool and whirlpool spa. Doors and windows of the indoor pool room are kept closed at all times; the only ventilation is in the maintenance room. The whirlpool spa has a capacity of 1893 L. There are no cooling towers on the hotel or on any buildings in the immediate area; hotel guest rooms are cooled by closed-unit window air conditioners.

Case finding. We were able to contact 414 guests who had stayed at the hotel on at least 1 night from 1 April through 11 May 1999; 150 (36%) of those guests stayed at the hotel on at least 1 night during the index weekend. Of the 414 guests, 21% were from Georgia, 9% from Tennessee, 6% from Alabama, 6% from Florida, 6% from Illinois, and ≤5% from each of 21 other states. Case finding identified an additional patient who had LD and 22 persons (5%) who had PF-like illness within 10 days of the last night of the hotel stay. Of the 150 persons who stayed at the hotel during the index weekend, 13 developed PF-like illness within 10 days of the last night of the hotel stay, compared with 9 of 264 guests who did not visit the hotel during the index weekend (9% vs. 3%; $P = .02$). Overall, attack rates for LD and PF-like illness among all persons contacted during the case finding were 11% for those who entered the indoor pool area and 2.6% for those who did not enter the indoor pool area.

Whirlpool spa maintenance records. On the weekend during which the case patients were in the whirlpool spa, bromine concentrations of 2 ppm were recorded, the minimum recommended concentration for whirlpool spas; the ideal bromine concentration is 3–5 ppm [16–18]. During that time, the pH was 7.2, which is also the minimum suggested value. The ideal pH is 7.4–7.6 [16–18]. During the daytime on 22 April, the day before the index weekend, the whirlpool spa water had been emptied and changed, and the whirlpool spa was drained on 26 April and left empty for several days while the underwater lighting fixtures were repaired.

During the 51 days from 22 March through 11 May, the pH was recorded 39 times in the maintenance log book, and the bromine concentration was recorded 40 times. The pH was outside of the ideal range for 22 (56%) of the 39 observations, and the bromine concentration was below the minimum recommended value for 10 (25%) of the 40 observations. In that time, there were 3 instances in which no bromine or pH values were documented for ≥2 days in a row.

Cohort Study

Of 150 hotel guests who stayed at Hotel A for at least 1 night during the index weekend, 64 (43%) reported being in either the outdoor or indoor pool area. Of these, 53 (83%) participated in the cohort study, 41 (77%) submitted serum specimens, and 35 (66%) submitted urine samples.

Clinical symptoms. In addition to the 2 patients with LD already described (in the section "Background" and the section "Case finding" in Results), 10 (18%) of the 53 study participants developed PF-like illness, and 7 of those became ill within 5 days (median, 1 day) of their last night at the hotel. The following symptoms were reported by the guests: diarrhea (6 of 10 guests with PF-like illness), cough (4 of 10), nausea (5 of 10), vomiting (6 of 10), headache (4 of 7), and myalgia (2 of 5) (information was not available for all guests).

Demographic and background information. The median age of ill persons was 20.5 years, the median age for persons with PF-like illness was 12 years (interquartile range [IQR], 5–31 years), and the ages of the 2 persons with LD were 61 and 71 years. The median age of non-ill persons was 37 years (IQR, 11–46 years).

Table 1. Pool area exposures in a cohort of 53 guests who used the indoor pool area of a hotel implicated in an outbreak of legionnaires disease and Pontiac fever.

Exposure parameter	Cohort members		*P*
	Ill ($n = 12$)	Not ill ($n = 41$)	
Guests who bathed in whirlpool spa, no. (%)	10 (83)	16 (39)	.007[a]
Time in indoor pool area, median min (IQR)	61 (61–150)	60 (15–180)[b]	.46
Time in whirlpool spa, median min (IQR)			
All guests	60 (12.5–60)	0 (0–10)	.0008
Only guests who used the whirlpool spa	60 (60–60)[c]	30 (7.5–60)[d]	.039

NOTE. IQR, interquartile range.
[a] Relative risk, 5.19 (95% confidence interval, 1.26–21.47).
[b] Of the 41 persons who did not become ill, information about time spent in the indoor pool area was available for 35.
[c] Of the 12 persons who became ill, only 10 entered the whirlpool spa.
[d] Of the 41 persons who did not become ill, only 16 entered the whirlpool spa.

Although the 2 LD patients had a history of long-term smoking, there was no statistical difference in smoking status between the group of patients who became ill and the group of patients who did not; 1 of 10 persons with PF-like illness, 2 of 2 persons with LD, and 9 of 41 non-ill persons had a history of smoking of any duration. Similarly, no statistical difference was seen in the presence of underlying medical conditions in the group of patients who became ill and the group of patients who did not. Two of 10 persons with PF-like illness, 0 of 2 persons with LD, and 5 of 41 non-ill persons had an underlying medical condition.

Exposure to the pool area. The median time of exposure to the indoor pool area was similar for ill and non-ill persons (table 1). The attack rate for those exposed to the indoor pool area was 22%, and, for those who entered the whirlpool spa water, it was 38%. The attack rate for those who entered the pool areas but did not enter the whirlpool spa water was 7% (relative risk, 5.6; $P = .0005$). Persons with LD or PF-like illness were significantly more likely to have bathed in the whirlpool spa. Ill persons reported spending a significantly longer period of time in the whirlpool spa than non-ill persons ($P = .007$; table 1).

Laboratory results. There was a dose-response relationship between duration of whirlpool spa exposure and LD or PF-like illness ($P = .001$) and IgM seropositivity ($P = .007$) (figure 1). There was no dose-response relationship between time spent in the indoor pool area and disease or IgM seropositivity.

The number of ill persons who had positive results of testing for IgM (titer $\geq$1:64) was significantly higher than the number of non-ill persons with positive results. Of the 41 persons tested for IgM, 4 of 9 individuals with PF-like illness and 2 of 32 non-ill persons had positive results (44% vs. 6%, $P = .02$). A 4-fold rise in IgGAM levels was detected in 1 of 9 persons with PF-like illness and in 1 of 32 non-ill persons ($P = .4$).

All 35 of the urine samples that were submitted yielded negative results on testing for Lp1 urine antigen.

Environmental Investigation

Lp6 was isolated from 1 of the whirlpool spa biofilm swab samples. No legionellae were isolated from any other environmental specimens. The Lp6 isolates from the whirlpool spa and from the infected patient were identical on AFLP analysis. The outbreak Lp6 strain was distinct on AFLP analysis from all available previously found Lp6 strains (figure 2).

Discussion

We describe one of the few recognized travel-associated combined outbreaks of LD and PF. Epidemiologic and laboratory data from this investigation indicated that the source of transmission was a poorly maintained whirlpool spa. Not only were attack rates significantly higher among persons exposed to the whirlpool spa, but the environmental isolate from the whirlpool spa matched the index patient isolate on molecular analysis and was found to be a unique Lp6 strain. A dose-response relationship existed between exposure to the whirlpool spa and both illness and serologic response.

Travel-associated outbreaks of LD are difficult to detect for many reasons: attack rates for LD are low, meaning that no clinician is likely to see >1 case; the incubation period is long, allowing infected persons to leave the area in which they acquired the infection; and surveillance is poor, making it difficult to link cases epidemiologically. Appropriate surveillance for travel-associated illness should be timely and sensitive, and it should contain an epidemiologic component that links exposure to hotels or other travel-associated sites and a laboratory component so that isolates can be matched. Implementation of detailed surveillance for travel-associated legionellosis is necessary for

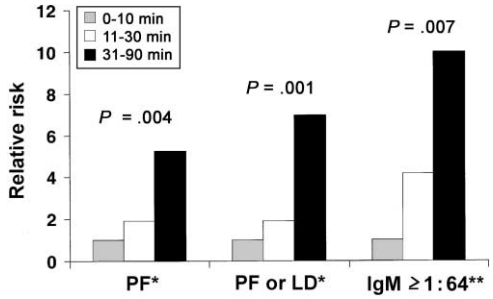

Figure 1. Dose-response relationship between duration of exposure to a whirlpool spa and both disease and IgM seropositivity (the χ^2 test was used to analyze the trend). The 0–10-min category was used as the reference group (relative risk, 1). For Pontiac fever (PF), 95% confidence intervals (CIs) were 0.23%–15.3% for 11–30-min exposures and 1.53%–17.9% for 31–90-min exposures. For PF or legionnaires disease (LD), 95% CIs were 0.23%–15.3% for 11–30-min exposures and 2.18%–22.3% for 31–90-min exposures. For IgM titers $\geq$1:64, 95% CIs were 0.3%–57.5% for 11–30-min exposures and 1.27%–78.9% for 31–90-min exposures. *$n = 53$; **$n = 41$.

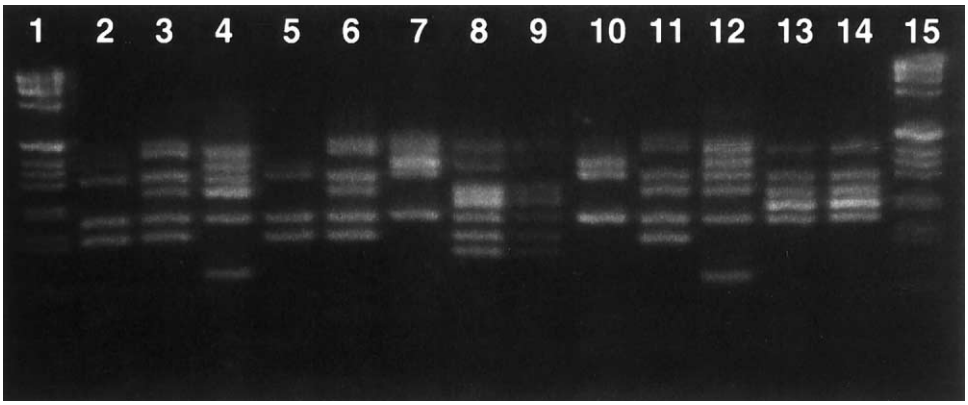

Figure 2. Amplified fragment–length polymorphism testing of standard *Legionella pneumophila* serogroup 6 (Lp6) isolates and the outbreak isolates. The outbreak Lp6 isolates from the whirlpool spa *(lane 13)* and the index patient *(lane 14)* were identical and demonstrated a pattern different from those of all previously isolated Lp6 strains. *Lanes 1* and *15,* Molecular weight standards; *lane 2,* Sydney 1; *lane 3,* Albany 1; *lane 4,* LD82-683; *lane 5,* Vasteras 57/1; *lane 6,* Chicago 2; *lane 7,* Denver 3; *lane 8,* SRP39; *lane 9,* SRP39; *lane 10,* ED38; *lane 11,* Johannesburg 5; *lane 12,* Oxford 1.

interruption of *Legionella* transmission from ongoing outbreak sources, for better understanding of travel-associated disease, and for implementation of preventive measures.

Whirlpool spas have been identified as sources of both LD and PF and have been associated with outbreaks in hotels and on cruise ships [7, 8, 10, 16, 19–24]. Because spas are common features of hotels and cruise ships, they may have a significant role in travel-associated legionellosis. Publicly used whirlpool spas may have much heavier use than whirlpool spas in private sites, and a heavy bather load can compromise halogen concentrations [17, 18]. Review of the maintenance records at Hotel A indicated that whirlpool spa conditions were frequently below optimum during the time period that we examined. Whirlpool spa–related disease is highly preventable; appropriate halogenation and filter disinfection practices can minimize the potential for *Legionella* growth and transmission [16–18]. Hotels and cruise ships must take responsibility for properly monitoring their facilities and for maintaining appropriate water conditions.

Laboratory testing is necessary for confirmation of legionellosis outbreaks. Culture followed by molecular typing is essential for matching environmental isolates to patient isolates for identification of the source of transmission. To be useful in an outbreak, methods of organism identification not only must allow comparison of patient and environmental isolates but also must be rapid. An available commercial urine antigen test provides rapid testing for infection with Lp1. Because IgM testing has the potential to provide rapid diagnosis for all strains of *Legionella* during an outbreak, it is important to standardize this tool, which is newly available for use in outbreak investigations. Like urine antigen testing, it does not yield an isolate. Therefore, although antigen and IgM testing are complements to legionellosis investigations that give rapid results, culture and molecular typing remain essential for tracing a patient isolate to an environmental source.

Although serologic studies do not result in isolation of an organism, they do have the ability to fine-tune case definitions of PF and LD. For this outbreak investigation, we adapted new IgM testing for use with disease caused by a non-Lp1 strain to confirm the diagnosis of legionellosis and establish the case status for both PF and LD. Serologic results confirmed that PF and LD were occurring concurrently during the outbreak and allowed us to document a dose-response relationship between exposure to the whirlpool spa and both disease and seropositivity. Although LD has been shown to have a dose-response relationship to duration of exposure to whirlpool spa water [21], no association between either PF or seropositivity and duration of exposure has been reported.

Once a *Legionella* species is isolated, molecular characterization can be accomplished by several methods. Established techniques of macrorestriction analysis and PCR-based typing have been limited by problems in reproducibility, standardization, and comparability [25–27]. The recent introduction of AFLP analysis has enabled molecular typing to be done on a level that can be widely applied, making documentation of travel-associated disease more feasible. AFLP techniques can be used to supplement surveillance of multistate travel-associated disease outbreaks; because AFLP patterns are readily computerized by use of fluorescence detection methods, information can be compared electronically across multiple states and locations. This technology may eventually be incorporated into already existing computerized surveillance systems, such as PulseNet, a pulsed-field gel electrophoresis–based surveillance system for enteric diseases [28–30]. The outbreak described in the present study marks the first time that AFLP testing was used on a non–serogroup 1 *L. pneumophila* and suggests that AFLP testing may be readily usable across serogroups and species.

Once cases of legionellosis are identified and confirmed, timeliness in the comparison of case information from many states

and locations is essential to finding and eliminating the source of transmission. As in this outbreak, the movement of travelers presents a challenge to timely surveillance for travel-associated legionellosis. However, through development of computerized systems that integrate AFLP technology, we can maximize the timeliness of any comprehensive surveillance system for travel-associated legionellosis.

Surveillance for travel-associated LD has not been strong in this country; however, in Europe, a travel-associated *Legionella* surveillance system links 31 countries, and in 1999 it detected 26 travel-associated clusters [31]. In the European scheme, travel-associated cases are entered into a computerized database. The system cross-checks patients' travel accommodations to determine whether other patients who stayed in the same accommodations in the previous 6 months are listed, thus making up a cluster. Collaborators in each country or center are notified, and follow-up investigation is initiated. Based on data from this system, it is estimated that 50% of LD cases in England and Wales are travel associated [32, 33].

This investigation demonstrates how the timely identification of a travel-associated outbreak of legionellosis can result in disease prevention. Because of the highly preventable nature of travel-associated LD and the possibly high incidence of disease, surveillance for travel-associated disease in the United States should be improved.

Acknowledgments

We thank Georgia public health officials Laura Gilbert, Tom Bennett, Joy Wells, Laura Axelson, Murl McCall, and Patrick Koffman, for their assistance; we thank Heather Craddock for her hard work on fluorescent amplified fragment–length polymorphism subtyping.

References

1. Marston BJ, Plouffe JF, File TM, et al. Incidence of community-acquired pneumonia requiring hospitalization. Arch Intern Med **1997**;157:1709–18.
2. Kaufmann AF, McDade JE, Patton CM, et al. Pontiac fever: isolation of the etiologic agent *(Legionella pneumophila)* and demonstration of its mode of transmission. Am J Epidemiol **1981**;114:337–47.
3. Breiman RF, Butler JC. Legionnaires' disease: clinical, epidemiological, and public health perspectives. Semin Respir Infect **1998**;13:84–9.
4. Butler JC, Fields BS, Breiman RF. Prevention and control of legionellosis. Infect Dis Clin Pract **1997**;6:458–64.
5. Glick TH, Gregg MB, Berman B, Mallison G, Rhodes WW, Kassanoff I. Pontiac fever: an epidemic of unknown etiology in a health department. I. Clinical and epidemiologic aspects. Am J Epidemiol **1978**;107:149–60.
6. Fraser DW, Tsai TR, Orenstein W, et al. Legionnaires' disease: description of an epidemic pneumonia. N Engl J Med **1977**;297:1189–97.
7. Thomas DL, Mundy LM, Tucker PC. Hot tub legionellosis: legionnaires' disease and Pontiac fever after a point-source exposure to *Legionella pneumophila.* Arch Intern Med **1993**;153:2597–9.
8. Goldberg DJ, Fallon RJ, Green ST, Wrench JG. Pontiac fever in children. Pediatr Infect Dis J **1992**;11:240–1.
9. Girod JC, Reichman RC, Winn WC, Klaucke DN, Vogt RL, Dolin R. Pneumonic and nonpneumonic forms of legionellosis: the result of a common-source exposure to *Legionella pneumophila.* Arch Intern Med **1982**;142:545–7.
10. Goldberg DJ, Wrench JG, Collier PW, et al. Lochgoilhead fever: outbreak of non-pneumonic legionellosis due to *Legionella micdadei.* Lancet **1989**;1:316–8.
11. Centers for Disease Control and Prevention (CDC). Procedures for the recovery of *Legionella* from the environment. Atlanta: CDC, **1994**.
12. McKinney RM, Kuffner TA, Bibb WF, et al. Antigenic and genetic variation in *Legionella pneumophila* serogroup 6. J Clin Microbiol **1989**;27:738–42.
13. Mazurek GH, Reddy V, Marston BJ, Haas WH, Crawford JT. DNA fingerprinting by infrequent-restriction-site amplification. J Clin Microbiol **1996**;34:2386–90.
14. Riffard S, Lo Presti F, Vandenesh F, Forey F, Reyrolle M, Etienne J. Comparative analysis of infrequent-restriction-site PCR and pulsed-field gel electrophoresis for epidemiological typing of *Legionella pneumophila* serogroup 1 strains. J Clin Microbiol **1998**;36:161–7.
15. Benson JM, Ellingsen D, Renshaw MA, Resler AG, Evatt BL, Hooper WC. Multiplex analysis of mutations in four genes using fluorescence scanning technology. Thromb Res **1999**;96:57–64.
16. Centers for Disease Control and Prevention (CDC). Final recommendations to minimize transmission of Legionnaires' disease from whirlpool spas on cruise ships. Atlanta: CDC, **1997**.
17. Centers for Disease Control and Prevention (CDC). Suggested health and safety guidelines for public spas and hot tubs. Atlanta: CDC, **1985**.
18. American Society of Heating, Refrigerating, and Air-Conditioning Engineers. Minimizing the risk of legionellosis associated with building water systems. Atlanta: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, **2000**.
19. Centers for Disease Control and Prevention. Update: outbreak of Legionnaires' disease associated with a cruise ship, 1994. MMWR Morb Mortal Wkly Rep **1994**;43:574–5.
20. Rocha G, Verissimo A, Bowker R, Bornstein N, Da Costa MS. Relationship between *Legionella* spp and antibody titres at a therapeutic thermal spa in Portugal. Epidemiol Infect **1995**;115:79–88.
21. Jernigan DB, Hofmann J, Cetron MS, et al. Outbreak of Legionnaires' disease among cruise ship passengers exposed to a contaminated whirlpool spa. Lancet **1996**;347:494–9.
22. Luttichau HR, Vinther C, Uldum SA, Moller J, Faber M, Jenson JS. An outbreak of Pontiac fever among children following use of a whirlpool. Clin Infect Dis **1998**;26:1374–8.
23. Mangione EJ, Remis RS, Tait KA, et al. Outbreak of Pontiac fever related to whirlpool use, Michigan 1982. JAMA **1985**;253:535–9.
24. Spitalny KC, Vogt RL, Orciari LA, Witherall LE, Etkind P, Novick LF. Pontiac fever associated with a whirlpool spa. Am J Epidemiol **1984**;120:809–17.
25. Lo Presti F, Riffard S, Vandenesh F, Etienne J. Identification of *Legionella* species by random amplified polymorphic DNA profiles. J Clin Microbiol **1998**;36:3193–7.
26. Mamolen M, Breiman RF, Barbaree JM, et al. Use of multiple molecular subtyping techniques to investigate a Legionnaires' disease outbreak due to identical strains at two tourist lodges. J Clin Microbiol **1993**;31:2584–8.
27. Jonas D, Meyer HW, Matthes P, et al. Comparative evaluation of three different genotyping methods for investigation of nosocomial outbreaks of Legionnaires' disease in hospitals. J Clin Microbiol **2000**;38:2284–91.
28. Tauxe RV. Emerging foodborne diseases: an evolving public health challenge. Emerg Infect Dis **1997**;3:425–34.
29. Tauxe RV. Strategies for surveillance and prevention. Lancet **1998**;352(Suppl 4):10.
30. Swaminathan B, Barrett TJ, Hunter SB, Tauxe RV. PulseNet: the molecular subtyping network for foodborne bacterial disease surveillance,

United States. The CDC PulseNet Task Force. Emerg Infect Dis **2001**; 7:382–9.

31. European Working Group for *Legionella* Infections. The European surveillance scheme for travel associated Legionnaires' disease. London: Public Health Laboratory Service Communicable Disease Surveillance Centre, **1999**. Available at http://www.ewgli.org. Accessed on 6 June 2000.
32. Joseph CA, Harrison TG, Ilijic-Car D, Bartlett CLR. Legionnaires' disease in residents of England and Wales: 1996. Commun Dis Rep CDR Rev **1997**; 7:153–9.
33. Joseph CA, Harrison TG, Ilijic-Car D, Bartlett CLR. Legionnaires' disease in residents of England and Wales: 1998. Commun Dis Public Health **1999**; 2:280–4.