

Epidemiologic Investigation of a Restaurant-Associated Outbreak of Pontiac Fever

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This case-control study investigated a cluster of respiratory illness among patrons of a restaurant. Of 173 patrons interviewed, 117 (68%) were ill. Symptoms included myalgias (93%), headache (87%), and fatigue (79%). The mean incubation period was 49 h and the mean duration of illness was 71 h. Patrons aged >15 years were more likely to have been ill than younger patrons (odds ratio [OR], 2.96; $P = .002$); 58% of persons who were ill sat near a large fountain, compared with 18% of respondents who were not ill (OR, 7.5; $P = .005$). *Legionella anisa* was cultured from water samples obtained from the fountain pool. Of 22 individuals who were ill, 11 (50%) had a ≥ 4 -fold increase in the titer of antibody to that strain of *L. anisa* from acute-phase to convalescent-phase serum samples; 3 others (14%) had persistently elevated titers of ≥ 512 ; of a group of 20 individuals who had not been exposed to the restaurant, none had titers of >128 . Pontiac fever should be considered as a diagnosis during acute outbreaks of influenza-like illness with a high attack rate and no other identified etiology.

Most outbreaks of disease in restaurants are due to food-borne gastroenteritis, and restaurant-associated Pontiac fever has never been reported. Legionellosis can present as a severe pneumonia (i.e., legionnaires disease), or it can present as a mild, self-limited illness characterized by fever and influenza-like symptoms without pneumonia (i.e., Pontiac fever). It has been estimated that 8000–18,000 persons are hospitalized in the United States each year with legionellosis [1], most of which cases are attributable to legionnaires disease. Outbreaks of Pontiac fever are seldom reported.

One species of *Legionella*, *Legionella pneumophila*, is responsible for 90% of reported cases of legionellosis in the United States [2, 3]. Although >20 species of

Legionella have been associated with human disease, infections with strains other than *L. pneumophila* are generally not detected by commonly available diagnostic tests. Because Pontiac fever is usually a mild, self-limited illness with nonspecific symptoms and with no widely available confirmatory laboratory test, it is possible that many cases of the disease are undiagnosed. Because outbreaks of Pontiac fever can have an explosive onset, have very high attack rates, and may be associated with cases of legionnaires disease [4], better understanding the epidemiology and prevention of Pontiac fever is important.

A county health department (Nashville-Davidson County Metropolitan Health Department) was notified that 13 persons out of a group of 14 who had eaten at a local restaurant on 19 April 2002 had become ill within 2 days after the meal. Initial reports indicated that the illness included fever, headache, nausea, vomiting, and diarrhea, with symptoms resolving within 2–3 days after onset, and a routine food-borne disease outbreak investigation was initiated. The next day, another report was received by the health department,

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stating that 12 of 15 persons attending a birthday party at the restaurant on the same day as the previous group had also become ill. On the third day, yet another group called to report that 5 of a group of 6 persons eating at the restaurant were ill. By that time, it appeared that the predominant symptoms among these groups were fever, chills, and myalgias, and that gastrointestinal symptoms were experienced by few persons. No common food had been identified among patrons who were ill. An expanded epidemiologic investigation was undertaken to determine the etiology and mode of transmission of the outbreak and to identify preventive interventions.

PATIENTS AND METHODS

Persons eating at restaurant A from 14 April to 28 April 2002 were identified by reservation lists, credit card receipts, and self-report to the county health department. A questionnaire was administered in person or by telephone by health department staff to ill and healthy patrons, as well as to employees of the restaurant. Questions were asked regarding illness, potential exposures, and demographics. Members of the case group were defined as persons with self-reported fever and ≥ 1 additional symptom (headache, cough, myalgias, vomiting, or diarrhea) occurring within 5 days after visiting restaurant A (an initial apparent incubation period of 1–2 days was reported). Members of the control group were defined as restaurant patrons who reported no illness within 5 days after visiting the restaurant. Statistical analyses were performed using the χ^2 test and Fisher's exact tests, using Epi Info 6.0 software [5].

Nasopharyngeal swab specimens were obtained from patrons who were ill and were cultured for enteroviruses, adenovirus, respiratory syncytial virus, influenza virus, parainfluenza virus, and herpesvirus. Urine specimens were obtained and tested for *L. pneumophila* serogroup 1–soluble antigen via EIA. Serum specimens were obtained during the initial investigation from persons who were ill, and follow-up serum specimens were obtained ~ 5 weeks later. Serum specimens were not available from healthy patrons who responded to the case-control questionnaire. Therefore, for comparison, at the time convalescent serum specimens were obtained from members of the case group, single serum specimens were obtained from residents of the same city who had not eaten at restaurant A during the epidemic period. Serum samples were tested at the Centers for Disease Control and Prevention (CDC; Atlanta, GA) for combined IgG, IgM, and IgA by means of a polyvalent indirect fluorescent antibody test against a *Legionella anisa* strain isolated from samples of water from environmental sources at restaurant A. For this study, a positive result was defined as a 4-fold rise in antibody titer between the acute phase and convalescent phase serum samples to a titer of ≥ 128 [6].

Environmental specimens were obtained from fountains, wa-

ter pools, and misters within the restaurant. Four 250-mL samples were obtained from large water pools in sterile plastic bottles containing a 10% thiosulfate solution. Swab specimens were obtained from pool edges and decorative aerosol-generating nozzles.

Environmental specimens were tested at the CDC. Water and swab samples were plated directly onto buffered charcoal yeast extract medium. Some samples were acid-treated before plating, with and without supplemental antibiotics. Plates were incubated at 35°C and examined at intervals for the presence of *Legionella*. Suspect colonies were selected and were inoculated onto biplates containing buffered charcoal yeast extract with and without L-cysteine. Cultures that required L-cysteine for growth were subcultured and tested with specific rabbit antisera to identify the *Legionella* species and serogroup [7].

RESULTS

Restaurant A had numerous fountains, decorative pools, and misting machines interspersed throughout the dining area. Dioramas with jungle animals activated intermittently in various areas of the restaurant, including the waiting area, with decorative misting and sounds that attracted observers. Other decorative features included a waterfall with a large pool at the base, water dripping from the ceiling into a long trough within the dining area, and several large fish tanks. During the outbreak period, the restaurant served a mean of ~ 1300 meals per day, with >2300 meals served on 20 April 2002. Approximately 280 employees worked at the restaurant. The restaurant was situated in a large indoor shopping mall. No clusters of illness were reported among patrons or employees of other establishments within the mall. No similar outbreaks were identified at other restaurants in the same chain during this period.

Questionnaires were administered to 173 restaurant patrons. Of these, 117 met the case definition. The mean age of members of the case group was 37 years (range, 4–76 years; SD, 15.6; median, 38 years). The most common symptoms included fever, myalgias, and headache (table 1). The mean incubation period was 49 h (range, 4–120 h) with a mean duration of illness of ~ 71 h (range, 4–192 h). Of persons who were ill, 29 (25%) of 117 sought medical care for this illness, and 1 person was hospitalized. Persons who were ill reported missing a mean of 2 days of work or other routine activities because of this illness (range, 0–8 days). Of persons who were ill, 102 (87%) reported eating in the restaurant from 18 April to 21 April, with most persons reporting onset of symptoms between 20 April and 23 April (figure 1). Among a cohort of 89 persons without previous knowledge of their disease status who were contacted by the health department, 53 were ill (attack rate, 60%), and 88% of those who were ill had eaten at the restaurant on either 19 April or 20 April. No secondary cases were re-

Table 1. Symptoms of 117 patrons reporting illness after eating at restaurant A.

Symptom	No. (%) of patrons
Fever	117 (100)
Myalgia	109 (93)
Chills	108 (92)
Headache	102 (87)
Fatigue	93 (79)
Backache	64 (55)
Nausea	64 (55)
Cough	52 (44)
Dizziness	44 (38)
Cramps	39 (33)
Diarrhea	35 (30)
Vomiting	19 (16)

ported. No patients received a diagnosis of pneumonic legionnaires disease. No employees of the restaurant reported illness during the outbreak period.

No common foods were identified among patrons who were ill. Few patrons who were ill reported smoking (9%), asthma (3%), other chronic lung disease (0%), or immunodeficiency disorder (2%), and they did not differ statistically from healthy respondents in these respects. Both ill and healthy respondents reported having been in the restaurant for a median of 90 min. The proportion of case group members who were male (37%) did not differ significantly from the proportion of control group members who were male (41%). The mean age of patrons who were ill was 37 years, compared with a mean age of 28 years for healthy patrons ($P = .002$); 96 patrons who were ill (82% of the case group) and 34 members (61%) of the control group were >15 years old. Patrons 16 years of age or older were significantly more likely to have been ill than patrons under the age of 16 (OR, 2.96; $P = .002$). Among respondents who indicated what area of the restaurant they ate in, 58% of persons who were ill sat near the side of the facility for which environmental cultures were positive for *L. anisa*, compared with 18% of healthy respondents (OR, 7.5; $P = .005$).

Nasopharyngeal swab specimens were obtained from 8 patients; viral cultures of all specimens were negative. *Legionella* urine antigen testing was performed on samples obtained from 24 patients in the case group, all of which had negative results. Forty-five environmental water and swab specimens were obtained from 26 sites within the restaurant. Two water samples and a swab specimen from a large ornamental pool in the dining area were culture-positive for a blue-white fluorescent *Legionella* species. The isolates were identified as *L. anisa* by slide agglutination, and this was confirmed by sequencing of the *mip* gene. Serum specimens were tested for antibody against this

strain of *L. anisa*. Of 22 persons who were ill for whom acute phase and convalescent phase serum samples were available, 11 patients (50%) had ≥ 4 -fold rise in antibody titer, to ≥ 256 , and an additional 3 patients (14%) had persistent titers of ≥ 512 . None of 20 single specimens obtained from city residents who were unexposed to the restaurant had titers of >128 .

The restaurant facilities were inspected by health department personnel. The ornamental water handling system was elaborate, with each large ornamental feature having a separate continuously recirculating water system. For each ornamental feature, conventionally chlorinated municipal water was circulated through a pump, sand-filtered, and disinfected with bromine before reentry to the feature. Major water features were reportedly drained and cleaned at least monthly, and pH and bromine levels were checked almost daily (with pH values of 7.5–8 and bromine levels of 2–5 ppm; recommended ideal levels for public spas and hot tubs are a pH of 7.4–7.6 and a bromine level of 3–5 ppm [8]). On 19 April and 20 April, the recorded pH of the pool from which *L. anisa* was later isolated was 7.6, and the bromine level was 4 ppm. On inspection, it was noted that pools drained directly into the sewer system, with no mechanism for backflow prevention. In addition, several “dead spots” were found in pools where circulation of water and disinfectant may not have occurred. The restaurant’s air conditioning system was inspected and found to be well-maintained and functioning properly, with little pooling condensate. Cultures of 8 water samples and swab specimens from 3 air-handling units were negative for *Legionella*.

DISCUSSION

To our knowledge, this is the first reported outbreak of restaurant-associated Pontiac fever. Because the outbreak occurred in a restaurant, it was initially suspected to be a food-borne disease outbreak, although the mechanism of transmission ultimately was demonstrated to be unrelated to food. An epidemiologic investigation identified the suspected etiology and the source of the outbreak, and it guided the environmental and laboratory investigation which ultimately confirmed the hypothesis. Only 18 outbreaks of Pontiac fever have been reported in the medical literature, of which 9 have been associated with whirlpools or hot tubs, 2 with building air-handling systems, and 5 with industrial operations (table 2) [4, 9–25]. One outbreak was associated with a decorative fountain in a hotel lobby and was the sole previous report of Pontiac fever due to *L. anisa* [19].

Given the ubiquity of *Legionella* in the environment, it is remarkable that only 18 outbreaks of Pontiac fever have been reported in >3 decades. It is possible that many cases of Pontiac fever may go unrecognized, given that it is a relatively mild and self-limited disease with nonspecific symptoms. Even if the disease is suspected, there are no readily available laboratory

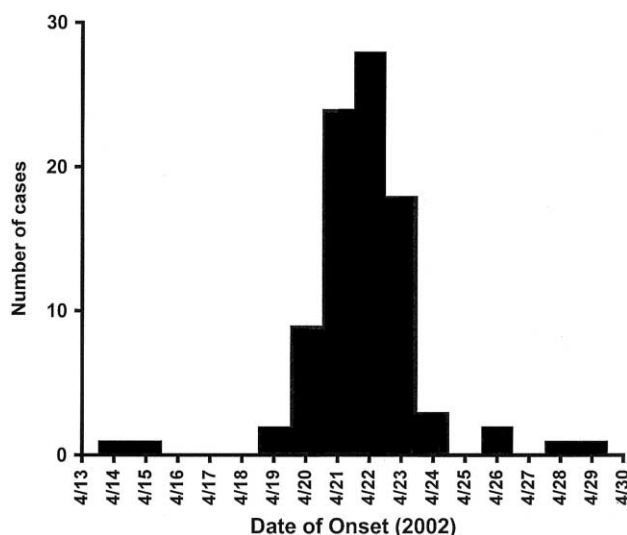


Figure 1. Epidemic curve showing dates of onset of illness among those patrons of restaurant A included in the Pontiac fever case group.

tests to confirm the diagnosis, as evidenced by the fact that the CDC laboratory performed the testing of specimens from at least 11 of the 13 outbreaks reported in the United States. Serologic testing for Pontiac fever is not practical except during recognized outbreaks. It is impossible to know how many cases of Pontiac fever may go unrecognized and be dismissed as “viral syndromes.” It is notable that, in this outbreak, several groups of patrons called a local health department to report clusters of influenza-like illness with a suspected association with a restaurant. Such claims are rare even in the midst of a severe influenza epidemic and can be very difficult to investigate satisfactorily during periods of widespread viral illness in a community.

The genus *Legionella* currently includes 48 species with 70 distinct serotypes, of which ~50% have been associated with human disease [2]. *L. pneumophila* causes ~90% of reported cases of legionellosis in the United States, and 79% of all culture-confirmed or urine antigen-confirmed cases are caused by *L. pneumophila* serogroup 1. Many illnesses due to other serotypes may go undiagnosed because urinary antigen testing is specific for *L. pneumophila* serogroup 1. Diagnosis by culture is the gold standard for confirmation of legionnaires disease. In contrast, cultures for patients with Pontiac fever usually are negative for *Legionella* species. It may often be difficult to collect specimens for culture from individuals with mild nonpneumonic illness. Among all the reports of Pontiac fever, only a single colony of *Legionella* has ever been cultured from a sample obtained from a patient [22]. Similarly, urinary antigen testing often has negative results for patients with Pontiac fever, even when the etiologic agent is *L. pneumophila* serogroup 1 [17, 22].

Most reported outbreaks of Pontiac fever have been diagnosed by isolation of *Legionella* from environmental specimens

and by confirmation of a ≥ 4 -fold increase in the serum titer of antibodies to the environmental strain among patients. PCR has also been used to identify *L. pneumophila* in environmental samples during a Pontiac fever outbreak [21]. Even in culture-confirmed cases of legionnaires disease, a 4-fold rise in antibody titer is detected in only 70%–80% of patients, and seroconversion may not occur for up to 2 months after onset of illness [2]. Reported rates of seroconversion in outbreaks of Pontiac fever have been quite variable (table 2). Of note, 4 (22%) of 18 reported outbreaks of Pontiac fever also included patients who received a diagnosis of legionnaires pneumonia [4, 13, 18, 20], and ≥ 2 reported outbreaks of legionnaires disease included patients with nonpneumonic symptoms consistent with Pontiac fever [26, 27].

In addition to *L. pneumophila* serogroups 1 and 6, Pontiac fever outbreaks have been associated with *Legionella micdadei* [2, 22, 24, 28], *L. anisa* [19], and *Legionella feeleyi* [15]. There do not appear to be distinct clinical or epidemiologic differences between outbreaks caused by different species. In the absence of diagnostic tests that will readily detect different species, it is impossible to determine the true incidence of disease attributable to particular strains of *Legionella*.

None of the employees of restaurant A admitted to having had recent Pontiac fever-like illness. Unfortunately, we were unable to obtain any specimens from employees for serologic testing. Results of testing of volunteers unexposed to the restaurant indicated that antibodies to *L. anisa* did not appear to be prevalent in the surrounding community. It is interesting that in only 1 of 10 Pontiac fever outbreaks associated with fountains, hot tubs, or whirlpools was a single employee of the implicated facility noted to be ill [24]. Although a variety of explanations could be proposed, it is not unreasonable to assume that some staff in these facilities may have been exposed to the etiologic agent for at least as long as the reported patients. In 8 reported outbreaks of Pontiac fever, antibody to *Legionella* were detected in healthy persons who had been exposed to the etiologic agent [4, 14, 19, 24]. In an outbreak associated with a fountain in a hotel lobby, 42% of the hotel employees (none of whom were ill) had elevated titers of antibody to the implicated pathogen. Unfortunately, we were unable to test the hypothesis that employees of this restaurant may have developed protective antibodies to *L. anisa* as a result of past asymptomatic or mildly symptomatic exposure to the organism.

It has been theorized that Pontiac fever may be caused by hypersensitivity to a cellular component of *Legionella* or to a protozoan host of the bacteria [29]. It is possible that endotoxins from dead or live legionellae may cause the symptoms of Pontiac fever [23]. Such a hypothesis could explain the failure to culture organisms from patients with acute Pontiac fever and the marked differences in epidemiology when Pontiac fever is compared with legionnaires disease.

Table 2. Summary of epidemiologic information on reported outbreaks of Pontiac fever.

Year	No. of patients	LD	Location	Source of isolate and/or outbreak setting	<i>Legionella</i> organism isolated ^a	Seroconversion, ^b % of patients	Reference(s)
1949	12	No	Richmond, VA	Steam condenser cleaners	<i>L. pneumophila</i>	NA	[9]
1968	144	No	Pontiac, MI	Air conditioning system	Lp1	84	[10, 11]
1973	10	No	James River, VA	Compressed air used to clean steam turbine	Lp1	56	[12]
1980	2	Yes	Vermont	Employees cleaning a cooling tower	<i>L. pneumophila</i>	100	[13]
1981	34	No	Vermont	Whirlpool	Lp6	67	[14]
1981	317	No	Windsor, Canada	Water-based coolant in engine assembly plant	<i>L. feeleii</i>	64	[15]
1982	14	No	Michigan	Whirlpool	Lp6	86	[16]
1984	86	No	New York City	Air conditioner cooling tower for office building	Lp1	42	[17]
1988	170	Yes	Scotland	Whirlpool	<i>L. micdadei</i>	60	[18]
1988	34	No	California	Decorative fountain, hotel lobby	<i>L. anisa</i>	63	[19]
1991	6	Yes	Vermont	Hot tub	<i>L. pneumophila</i>	100	[20]
1992	13	No	Colorado	Indoor hot tub	Lp6	64	[21]
1995	13	No	Denmark	Whirlpool	<i>L. micdadei</i> , Lp1	67	[22]
1997	5	No	Denmark	Sewage treatment plant	Lp1	80	[25]
1998	45	No	Wisconsin	Whirlpool	<i>L. micdadei</i>	33	[23]
1999	29	No	Sweden	Whirlpool	<i>L. micdadei</i>	NA	[24]
1999	24	Yes	Georgia	Whirlpool	Lp6	11	[4]
2002	117	No	Tennessee	Restaurant with fountains	<i>L. anisa</i>	50	...

NOTE. LD, legionnaires disease associated with outbreak; NA, not available.

^a Lp1, *Legionella pneumophila* serogroup 1; Lp6, *L. pneumophila* serogroup 6.

^b Percentage of those epidemiologically identified patients with Pontiac fever from whom serum samples were obtained during both the acute phase and convalescent phase of disease for whom a ≥ 4 -fold increase in serum titer of antibody against *Legionella* was reported.

This outbreak occurred in a facility that had been in operation for many months, without any previous problems. It is interesting that the outbreak appeared to have subsided before the health department investigated and temporarily shut down the facility's ornamental water fixtures. No specific inciting environmental cause for the outbreak could be identified in retrospect. Many areas in the restaurant had been cleaned before the facility was inspected and before samples could be obtained. It is possible that a transient, unrecognized problem with the water filtration and treatment system could have led to the proliferation and dissemination of the pathogen and that subsequent correction of the problem eliminated the exposure. Several Pontiac fever outbreaks have occurred in facilities with brominated whirlpools and hot tubs [4, 16, 21, 23, 28]. In only one of these outbreaks was a brominator identified as faulty [28]. *Legionellae* are more susceptible to chlorine than to bromine [11]. Recommendations for ideal bromine and pH levels in whirlpool spas have been published [8], and consideration should be given to development of recommendations that include decorative fountains. Recently, data have begun to accumulate suggesting that monochloramine may be a more ef-

fective biocide than chlorine for preventing the spread of *Legionella* through municipal water systems and in health care facilities [2]. The effect of an increase in the use of monochloramine on the incidence of legionellosis outbreaks has yet to be assessed.

Pontiac fever may be more common than is generally appreciated. Improvements in diagnostic testing could lead to a better understanding of the epidemiology and prevention of outbreaks of Pontiac fever. Assiduous care must be given to the maintenance of ornamental water fixtures, such as fountains and misters, and their water treatment systems to prevent outbreaks of legionellosis.

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References

1. Marston BJ, Plouffe JF, File TM, et al. Incidence of community-acquired pneumonia requiring hospitalization: results of a population-based active surveillance study in Ohio. *Arch Intern Med* **1997**;157:1709–18.
2. Fields BS, Benson RF, Besser RE. *Legionella* and Legionnaire's disease: 25 years of investigation. *Clin Microbiol Rev* **2002**;15:506–26.
3. Benin AL, Benson RF, Besser RE. Trends in legionnaires disease, 1980–1998: declining mortality and new patterns of diagnosis. *Clin Infect Dis* **2002**;35:1039–46.
4. Benin AL, Benson RF, Arnold KE, et al. An outbreak of travel-associated legionnaires disease and Pontiac fever: the need for enhanced surveillance of travel-associated legionellosis in the United States. *J Infect Dis* **2002**;185:237–43.
5. Dean AD, Dean JA, Coulombier D, et al. Epi Info version 6: a word processing, database and statistics program for epidemiology on microcomputers. Atlanta: Centers for Disease Control and Prevention, **1994**.
6. Centers for Disease Control and Prevention (CDC). Hospital-laboratory diagnosis of *Legionella* infections. Atlanta: CDC, **1988**.
7. Centers for Disease Control and Prevention (CDC). Procedures for the recovery of *Legionella* from the environment. Atlanta: CDC, **1994**.
8. Centers for Disease Control and Prevention (CDC). Suggested health and safety guidelines for public health spas and hot tubs. Atlanta: CDC, **1985**.
9. Armstrong CW, Miller GB. A 1949 outbreak of Pontiac fever-like illness in steam condenser cleaners. *Arch Environ Health* **1985**;40:26–9.
10. Glick TH, Gregg MB, Berman B, Mallison G, Rhodes WW, Kassanoff I. Pontiac fever: an epidemic of unknown etiology in a health department. 1. Clinical and epidemiologic aspects. *Am J Epidemiol* **1978**;107:149–60.
11. 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.
12. Fraser DW, Deubner DC, Hill DL, Gilliam DK. Nonpneumonic, short-incubation-period legionellosis (Pontiac fever) in men who cleaned a steam turbine condenser. *Science* **1979**;205:690–1.
13. 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.
14. Spitalny KC, Vogt RL, Orciari LA, Witherell LE, Etkind P, Novick LF. Pontiac fever associated with a whirlpool spa. *Am J Epidemiol* **1984**;120:809–17.
15. Herwaldt LA, Gorman GW, McGrath T, et al. A new *Legionella* species, *Legionella feeleii* species nova, causes Pontiac fever in an automobile plant. *Ann Intern Med* **1984**;100:333–8.
16. Mangione EJ, Remis RS, Tait KA, et al. An outbreak of Pontiac fever related to whirlpool use, Michigan, 1982. *JAMA* **1985**;253:535–9.
17. Friedman S, Spitalny KC, Barbaree JM, Faur Y, McKinney R. Pontiac fever outbreak associated with a cooling tower. *Am J Public Health* **1987**;77:568–72.
18. Goldberg DJ, Wrench JG, Collier PW, et al. Lochgoilhead fever: outbreak of non-pneumonic legionellosis due to *Legionella micdadei*. *Lancet* **1989**;1:316–8.
19. Fenstersheib MD, Miller M, Diggins C, et al. Outbreak of Pontiac fever due to *Legionella anisa*. *Lancet* **1990**;336:35–7.
20. Thomas DL, Mundy LM, Tucker PC. Hot tub legionellosis: legionnaires disease and Pontiac fever after a point-source exposure for *Legionella pneumophila*. *Arch Intern Med* **1993**;153:2597–9.
21. Miller LA, Beebe JL, Butler JC, et al. Use of polymerase chain reaction in an epidemiologic investigation of Pontiac fever. *J Infect Dis* **1993**;168:769–72.
22. Luttichau HR, Vinther C, Uldum SA, Moller J, Faber M, Jensen JS. An outbreak of Pontiac fever among children following use of a whirlpool. *Clin Infect Dis* **1998**;26:1374–8.
23. Fields BS, Haupt T, Davis JP, Arduino MJ, Miller PH, Butler JC. Pontiac fever due to *Legionella micdadei* from a whirlpool spa: possible role of bacterial endotoxin. *J Infect Dis* **2001**;184:1289–92.
24. Gotz HM, Tegnell A, De Jong B, et al. A whirlpool associated outbreak of Pontiac fever at a hotel in northern Sweden. *Epidemiol Infect* **2001**;126:241–7.
25. Gregersen P, Grunnet K, Uldum SA, Andersen BH, Madsen H. Pontiac fever at a sewage treatment plant in the food industry. *Scand J Work Environ Health* **1999**;25:291–5.
26. Fraser DW, Tsai TR, Orenstein W, et al. Legionnaires' disease: description of an epidemic of pneumonia. *N Engl J Med* **1977**;297:1189–97.
27. O'Mahony MC, Stanwell-Smith RE, Tillett HE, et al. The Stafford outbreak of legionnaires disease. *Epidemiol Infect* **1990**;104:361–80.
28. Goldberg DJ, Emslie JA, Fallon RJ, Green ST, Wrench JG. Pontiac fever in children. *Pediatr Infect Dis J* **1992**;11:240–1.
29. Rowbotham TJ. Pontiac fever explained? *Lancet* **1980**;2:969.