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Potable Water as a Source for Legionellosis

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Several lines of evidence have been examined in attempts to implicate potable water as a source for legionellosis. Success has been mixed. The strongest evidence has been the similarity of strains recovered from patients and from potable water and the cessation of outbreaks following institution of measures to eradicate Legionella from potable water systems. Epidemiologic efforts to identify the effective mode of exposure to water (e.g. ingestion) have been remarkably unsuccessful. Although *L. pneumophila* can clearly be acquired on occasion from potable water, the proportion of cases traceable to this source is unknown, as is the role of potable water as a source of infection by other Legionellae. Hyperchlorination, raising hot water temperatures to >55°C, and replacing rubber gaskets are useful methods for controlling outbreaks of legionellosis traced to potable water systems but are not yet justified as routine preventative methods in the absence of such an outbreak.

Introduction

Since the discovery of *Legionella pneumophila* in 1976 (*1*), much has been learned about a remarkable group of bacteria—the Legionellae—that cause pneumonia in humans and are well suited for survival in water, especially warm water. The discovery by Tobin that *L. pneumophila* could be found in potable water (*2*) set off an intense search for evidence that potable water was a source of legionellosis and to determine the manner by which people may become infected from Legionella-contaminated water. Considerable progress has been made but it did not come easily, in large part because the epidemiology and bacteriology of the Legionellae are remarkably different from those of most waterborne bacteria.

The usual scheme for demonstrating that drinking water is a source of infection in humans involves several steps. First, the clinical and epidemiologic features of the illness are reviewed for evidence of similarity with other waterborne diseases. Second, evidence is sought that the people who became ill had a particularly intense exposure to potentially contaminated potable water; this phase involves comparison of water exposure among cases and noncases, a search for evidence of a dose–response relationship, and confirmation that exposure preceded illness by an appropriate interval. Third, studies are done to demonstrate that the offending bacterium was in fact present in the implicated water, and in concentrations appropriate to explain the pattern of disease occurrence. Fourth, if the cases under study comprised a temporal cluster, an explanation is sought for the beginning of the cluster. Fifth, control measures are instituted that are appropriate for waterborne infections, and the results of those measures are closely analyzed for evidence that the effect on disease occurrence is what would have been predicted from the hypothesis that the disease was waterborne.

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Clinical and Epidemiologic Features

On the face of it, legionellosis is not typical of waterborne diseases. The fact that the most common manifestation of legionellosis is pneumonia is particularly unusual. Most infections caused by ingestion of bacteria-contaminated water are characterized by primary involvement of the gastrointestinal tract, although typhoid, for example, can include pneumonia. Many waterborne infections have remarkably high attack rates—perhaps because of the high dose and widespread exposure that comes with contamination of a water supply. The attack rate of Legionella pneumonia is typically low, however—usually less than 5%. Most outbreaks of other waterborne infections are traced to inadequately maintained water supplies, exogenous contamination of which causes infection because of inadequacies in water treatment. Legionellosis outbreaks on the contrary have most commonly been observed in hospitals and hotels fed by well-maintained municipal water supplies, although exceptions have been noted in which clusters occurred in buildings that receive untreated water (*3*,*4*). In searching for an explanation for the striking differences between legionellosis and other waterborne infections, in these regards it may be important to consider that Legionellae may not be acquired in most cases by ingestion and that the epidemiology of organisms adapted to warm water may be very different from that of organisms more usually associated with cold water.

Historical Fit of Exposure and Illness

Considering the intensity of the search for an association between risk of legionellosis and exposure to

potentially contaminated potable water, remarkably little epidemiological evidence has been found. Ingestion as a means of exposure was suggested by the investigation of the outbreak of Legionnaires' disease in Philadelphia in 1976: 45 of 69 convention delegates who became ill had drunk water at the headquarters hotel as compared to 469 of 976 delegates who remained well ($p<0.01$) (*5*). However, 38% of the ill Legionnaires recalled drinking no water in that hotel, suggesting that at least many of the cases had not been acquired by ingestion of water. Jones and colleagues observed that patients who used antacids were 4.9 times as likely as those who did not to develop illness in an outbreak of nosocomial legionellosis in Connecticut (*6*). However, they had difficulty separating antacid use from steroid therapy and respiratory therapy as risk factors. If antacid use does indeed increase risk of legionellosis it might be by allowing ingested Legionellae to survive passage through the stomach, as gastric acid is an effective defense against many waterborne bacteria. Others who have looked for a direct association between drinking water and legionellosis have been unable to find one (*7*).

The possibility that bathing or indirect exposure to showers might lead to legionellosis has generated considerable interest, but only recently has epidemiologic support been found. Hanrahan and colleagues found in a study of nosocomial legionellosis case-patients had rooms located nearer to the communal showers than did controls (1.5 vs. 4.1 room-lengths on the average; $p<0.05$) (*8*). However, a confounding effect of length of hospital stay could not be ruled out. Bartlett and colleagues in the study of a hotel-based outbreak in Spain found that, among roommates, the ill person tended to use the washing facilities first in the morning (*4*). In light of some evidence that Legionellae proliferate in plumbing dead-ends this association might suggest that the Legionellae were acquired during morning bathing; however, the association was not statistically significant. It must be noted also that Helms and colleagues found that case-patients in a nosocomial outbreak in Iowa City showered significantly less long than did controls (*7*). Taken as a whole, the epidemiologic evidence that bathing or showering is a risk factor in legionellosis is pretty weak.

Arnow and colleagues found strong evidence that respiratory therapy devices that produce aerosols of tap water can lead to legionellosis (*9*). In a nosocomial cluster in Chicago, all 5 cases and only 4 of 69 controls had used such devices. Because all of the cases and many fewer of the controls had also received high-dose immunosuppressive therapy, it was important to rule out a confounding effect. This they did by showing that among cases and controls who received corticosteroid therapy and respiratory therapy with devices that produce aerosols of either tap water or sterile water, three of three cases had been exposed only to tap water and three of three controls only sterile water ($p = 0.05$). The study by Jones and colleagues supports the suggestion that aerosolized tap water from respiratory therapy equipment can be a source of nosocomial legionellosis (*6*).

In some circumstances it seems that recreational whirlpool baths that use potable water can be a source of legionellosis. Outbreaks of both Legionnaires' disease and Pontiac fever have been linked epidemiologically to use of such whirlpools (*10*).

Anecdotal evidence suggests that legionellosis may on occasion be spread via inoculation of water. Arnow and colleagues reported a case of pneumonia in an immunosuppressed woman 3 to 5 days after she had received a series of six tap water enemas that resulted in perirectal erosions (*11*). *L. pneumophila* was subsequently cultured from a perirectal abscess. *L. pneumophila* infection of a hemodialysis fistula has also been observed (*12*).

One reasonable interpretation of the difficulty in finding much epidemiologic evidence to implicate potable water is that few cases in fact are caused by potable water. Other possibilities remain, however. It may be that few people are susceptible and that susceptibility is associated with the absence of effective exposure to contaminated water—as might occur if immunosuppressed patients were both susceptible to legionellosis and less likely to shower. Alternatively, exposure to contaminated potable water may be by a route or mechanism (such as aerosols generated by flushing of toilets) not being measured reliably or at all.

Presence of Legionella and Illness

It has become increasingly clear that in many instances the same strain of *L. pneumophila* that has caused a case of human illness can be found in the potable water supply where the illness was acquired. The earliest evidence was based on similarities of serogroups of clinical isolates and those from plumbing apparatus (*2,13*). More recently several techniques have been used to subtype strains of *L. pneumophila*, including serotyping with monoclonal (*14*) or absorbed polyclonal (*15*) antibodies, plasmid analysis (*16*), and gas–liquid chromatography (*17*). Plouffe and colleagues reported the plasmid contents of strains of *L. pneumophila* serogroup 1 collected in the course of investigation of a nosocomial outbreak involving two adjacent hospital buildings (*16*). Of the 20 cases, 19 occurred in building UH (1.03 case/1000 discharges) and 1 in building RH (0.08 case/1000 discharges). The potable water system of the two buildings were equally contaminated with *L. pneumophila* (28% and 27% of cultures from hot water taps positive in the two buildings, respectively). The only clinical isolate and all environmental isolates from building RH contained 40 and 85 Mdalton plasmids. The 15 clinical isolates from building UH included 12 with no plasmid and 3 with only a 40 Mdalton plasmid; 95% and 4%, respectively, of the potable water isolates from building UH had these plasmid patterns and fewer than 1% had both plasmids.

It will be intriguing to see whether this striking association will be seen elsewhere as well documented sets

of related clinical and potable water specimens (and appropriate controls) are subtyped. Some evidence that the association is not exact comes from the study by Saravolatz and colleagues that showed *L. pneumophila* serogroup 3 and *L. dumoffii* in the water supply of a hospital with 20 cases of nosocomial legionellosis none of which apparently was caused by those strains (*18*).

The study by Plouffe and colleagues is useful, not only because it shows that the two strains that caused most of the cases are present in the water supply of the building where the cases occurred, but also because it demonstrated that the cases occurred where the rate of environmental contamination with a particular strain was relatively high (*16*). With the exception of studies of control procedures (discussed below), little evidence of this sort has been published. Stout and colleagues did show that with the waning of a hospital outbreak of *L. pneumophila* pneumonia the proportion of water sampling sites that were positive for *L. pneumophila* dropped from 10 of 10 to 6 of 10, but the data presented in that paper do not suggest that the concentrations of *L. pneumophila* in the positive specimens dropped correspondingly (*19*). A study of *L. micdadei* nosocomial infections by Rudin and colleagues showed that *L. micdadei* was found in the hospital's potable water only rarely despite the continuing occurrence of cases (*20*).

The Beginnings of Clusters

In several instances manipulations of plumbing systems preceded outbreaks of legionellosis by intervals that suggested a causal relationship. At the Wadsworth Medical Center in Los Angeles *L. pneumophila* pneumonia had occurred at a rate of about 4 cases per month after the opening of the hospital in 1977, but 22 cases occurred in March 1980 (*21*). The ensuing investigation revealed that on February 27 an emergency water pump had been tested by turning off the exit valves in the water holding tanks at the hospital. The pump failed, causing water pressure to drop precipitously. For several weeks thereafter hospital personnel complained that hospital water was discolored. Testing of the *L. pneumophila* concentration in hospital water before and after reproducing the pressure shock showed that the brown water had 30 times more *L. pneumophila* than the clear water, further evidence that the potable water had indeed been the source of the March 1980 cluster. Discolored water was also noted after a reserve heating unit was put into service at a hospital in Kingston a year after an outbreak of *L. pneumophila* pneumonia. A man admitted to the hospital on the day the new unit was installed developed legionellosis; >108 *L. pneumophila*/L were isolated from the sediment in the heater (*22*). Instances of other work on plumbing systems shortly before clusters have also been reported although a causal relationship has not seemed certain (*3*).

Effect of Control Measures

Perhaps the most impressive evidence that legionellosis is spread from potable water is found in several observations that outbreaks have ceased after steps have been taken to eradicate *L. pneumophila* from the systems or markedly to reduce its concentration. In the Wadsworth Medical Center outbreak supplemental chlorination to maintain a free residual chlorine concentration of 3 mg/L led to a sudden and sustained drop in the incidence of cases (*21*). In the Kingston outbreak, cases, which had been occurring at a rate of 2 per month, ceased when chlorination (to 2 mg/L) and raising the temperature of the hot water (to 55°C) were instituted (*22*). When cases recurred a year later, *L. pneumophila* was eradicated from the plumbing system and cases ceased only when all black rubber washers were replaced with washers that would not, in laboratory testing, contribute to the growth of *L. pneumophila* (*23*).

At the hotel in Benidorm, Spain, cases of legionellosis had been noted in visitors every year from 1975 through 1980 (*4*). In 1980 the cold water system was chlorinated to 60 mg/L for 30 min and the water in the hot water tanks was heated to 90°C for 30 min. Subsequently hot- and cold-water systems were treated so as to maintain free residual chlorine concentrations of >2 mg/L and hot-water temperatures of 50 to 60°C at all taps. No cases of legionellosis were recognized in hotel guests in the subsequent three years.

Best and colleagues used intermittent increases in the temperature of potable hot water to control a recurrent problem of nosocomial legionellosis in a Pittsburgh Veterans Administration hospital (*24*). They found that, with some consistency, an increase in culture positivity to 30% or more of surveillance sites was associated with subsequent cases of *L. pneumophila* or *L. micdadei* pneumonia and that *L. pneumophila* culture-positivity and case incidence were markedly reduced after 72 hr of raising the hot water tank temperature to 60 to 77°C. The evidence of a direct effect was more impressive for *L. pneumophila* than for *L. micdadei*, perhaps because cases of pneumonia caused by the latter were fewer. However, Rudin and colleagues found that the incidence of cases of *L. micdadei* pneumonia in a nearby Pittsburgh hospital increased at a time when the concentration of *L. pneumophila* in the hospital water supply dropped markedly because of an increase in the hot water temperature to 54°C (*20*). It may be either that *L. micdadei* is not regularly transmitted from potable water or that it is more heat-resistant than *L. pneumophila*.

Helms reported efforts to control the occurrence of *L. pneumophila* pneumonia in an Iowa City hospital where three clusters involving 24 cases had been documented in 1981 (*7*). After shock chlorination to >15 mg/L for 12 hr, free chlorine levels of >3 mg/L were maintained, as was the temperature in the hot water heater at 52.2°C. No cases of nosocomial legionellosis were observed in the affected building in the subsequent 13 months.

Baird and colleagues had reasonable success using chlorination alone to control a continuing problem with nosocomial *L. pneumophila* pneumonia at Riverside Methodist Hospital in Columbus (*25*). In the 5 years

before chlorination (to 4 mg free residual chlorine/L) 91 cases of nosocomial and 25 cases of community-acquired legionellosis had been recognized; in the 12 months after chlorination, seven and five cases of nosocomial and community-acquired legionellosis, respectively, were recognized ($p<0.02$). They estimated that a 10-year chlorination program would cost about $4000 per year.

Evidence that elevated temperatures of hot water systems may by themselves diminish exposure to *L. pneumophila* is found in surveys of hotel, hospital and home water systems. Dennis and colleagues showed that in the water systems of a group of British hotels *L. pneumophila* was found more consistently on the hot water side than on the cold water side (*26*). Plouffe and colleagues isolated *L. pneumophila* from hot water taps of four hospital buildings with hot water storage temperatures of 43 to 45°C but not from two adjacent buildings, on the same water supply, with hot water temperatures of 58 to 60°C (*27*). Arnow and Weil, in a survey of 37 Chicago apartment buildings, recovered *L. pneumophila* from water specimens from 19 (37%) of 52 apartments (*28*). The mean temperature of hot water at the time of specimen collection was lower for culture-positive (47.7°C) than for culture-negative (54.9°C) specimens.

In attempting to rid a potable water system of Legionella, the other side of the ecological coin from disinfection and heating may be the removal of conditions that favor the growth of the organisms. The cul de sac appears to be an especially welcome place for Legionella to collect and grow (*23,29,30*). Aerators in faucets have been reported inordinately likely to be culture-positive (*30*). Colbourne and colleagues showed that the first 75 mL drawn from a tap that had been unused overnight had the highest concentration of *L. pneumophila* (*23*). Removing culs de sac and obstructions to the free flow of potable water may be of some help in limiting exposure to the organisms.

Another help may be the replacement of rubber washers that facilitate the growth of Legionella. Colbourne and colleagues showed that certain washers are frequently contaminated with *L. pneumophila* and that, like paraffin wax, they are effective in supporting *L. pneumophila* growth under laboratory conditions (*23*). Replacement of such washers in the water system of one hospital resulted in disappearance of the organism from environmental surveillance cultures. If this success can be repeated consistently, it will represent a major advance in the control of Legionella in potable water systems.

If the Legionella cannot readily be eradicated from the potable water system or sufficiently diminished in numbers, one might consider controlling a problem of legionellosis by limiting exposure of susceptibles to the water. At the Wadsworth Medical Center before potable water was recognized to be the source, the renal transplantation program was discontinued and high risk patients were admitted to a separate building (*21*). Helms and colleagues, besides heating and chlorinating the water, substituted distilled water for and curtailed shower use by hematology-oncology patients (*7*). Such measures may be useful in extraordinary circumstances or until other effective control measures can be instituted, but are sufficiently cumbersome as not to be practical long-term solutions in most instances.

Conclusions

Accumulated evidence clearly implicates potable water as the source of some cases, including some outbreaks, of *L. pneumophila* infection. Such an association has not clearly been shown for the other Legionella species—the data are conflicting for *L. micdadei* and essentially nonexistent for the other species. Even for *L. pneumophila*, however, it is unknown what proportion of cases might be attributable to potable water. Furthermore, although institutional hot water systems have been implicated repeatedly, there is very little information on the possible role of home hot water systems as sources of legionellosis.

Ignorance of three critical features of Legionella infection has markedly hampered efforts to understand in detail under what conditions Legionella may be acquired from potable water. The first is the virulence of the organism. Recent studies suggesting variations in strain virulence indicate that this may be an exceedingly worthwhile area for further study. The second is the degree of exposure of the host to Legionella. Crude measures such as proximity to showers or amount of water imbibed have proved so far to be of little help, perhaps because they fail to account for such potentially critical variables as Legionella concentration and aerosol particle size and density. We would likely benefit a good deal from the development and deployment of gentle and sensitive air sampling techniques to measure exposure to Legionella in air or aerosols. The third is the determination of host susceptibility to Legionella. The occurrence of low attack rates of Legionnaires' disease in common source outbreaks among populations with low prevalences of elevated titres of specific antibody as measured by indirect immunofluorescence suggests that such antibody is at best an insensitive measure of immunity. If we had a method accurately to distinguish susceptible from resistant persons epidemiologic studies might be far more precise in identifying the particular exposures that are likely to lead to infection.

Although several methods for controlling spread of Legionella have been identified none seems ready for general use, except as a control measure when there is evidence of a legionellosis outbreak. In the face of an outbreak, raising the temperature of the hot water to >55°C or chlorinating so as to achieve a free residual chlorine level of > 2mg/L is likely to be effective and justified. Without an outbreak, however, the risks of scalding or of exposure to trihalomethane and the cost may more than offset the theoretical advantages of preventing legionellosis (*31*). If removal of rubber gaskets proves generally effective in eradicating Legionella from

hot water systems, this may some day prove to be a practical general control measure (*23*).

REFERENCES

1. McDade, J. E., Shepard, C. C., Fraser, D. W., Tsai, T. R., Redus, M. A., and Dowdle, W. R. Laboratory investigation team. Legionnaires' disease: isolation of a bacterium and demonstration of its role in other respiratory disease. N. Engl. J. Med. 297: 1197–1203 (1977).
2. Tobin, J. O'H., Beare, J., and Dunhill, M. S. Legionnaires' disease in a transplant unit: isolation of the causative agent from shower baths. Lancet ii: 118–121 (1980).
3. Bartlett, C. L. R., and Bibby, L. F. Epidemic legionellosis in England and Wales, 1979-1982. Zentralb. Bakteriol. Mikrobiol. Hyd. Abt. 1 A255: 64–70 (1983).
4. Bartlett, C. L. R., Swann, R. A., Casal, J., Canada Royo L., and Taylor, A. G. Recurrent Legionnaires disease from a hotel water system. In: Proceedings of the Second International Symposium on Legionella (C. Thornsberry and J. C. Feeley, Eds.), American Society for Microbiology, Washington, 1984, pp. 235–239.
5. Fraser, D. W., Tsai, T. R., Orenstein, W., Parkin, W. E., Beecham, H. J., Sharrar, R. G., Harris, J., Mallison, G. F., Martin, S. M., McDade, J. E., Shepard, C. C., and Brachman, P. S. The field investigation team. Legionnaires' disease: description of an epidemic of pneumonia. N. Eng. J. Med. 297: 1189–1197 (1977).
6. Jones, E., Checko, P., Dalton, A., Cope, J., Barbaree, J., Klein, G., Martin, W., and Broome, C. Nosocomial Legionnaires' disease associated with exposure to respiratory therapy equipment, Connecticut. In: Proceedings of the Second International Symposium on Legionella (C. Thornsberry and J. C. Feeley, Eds.), American Society for Microbiology, Washington, 1984, pp. 225–227.
7. Helms, C. M., Massanari, R. M., Zietler, R., Streed, S., Gilchrist, M. J. R., Hall, N., Hausler, W. J., Jr., Sywassink, J., Johnson, W., Wintermeyer, L., and Hierholzer, W. J., Jr. Legionnaires' disease associated with a hospital water system: a cluster of 24 nosocomial cases. Ann. Intern. Med. 99: 172–178 (1983).
8. Hanrahan, J. P., Morse, D. L., Scharf, V. B., Debbie, J. G., Schmid, G. P., McKinney, R. M., and Shayegani, M. Community hospital legionnellosis outbreak linked to hot-water showers. In: Proceedings of the Second International Symposium on Legionella (C. Thornsberry and J. C. Feeley, Eds.), American Society for Microbiology, Washington, 1984, pp. 224–225.
9. Arnow, P. M., Chou, T., Weil, D., Shapiro, E. N., and Kretzschmar, C. Nosocomial Legionnaires' disease caused by aerosolized tap water from respiratory devices. J. Infect. Dis. 145: 460–467 (1982).
10. Witherell, L. E., Orciari, L. A., Spitalny, K. C., Peeletier, R. A., Stone, K. M., and Vogt, R. L. Disinfestion of *Legionella pneumophila*-contaminated whirlpool spas. In: Proceedings of the Second International Symposium on Legionella (C. Thornsberry and J. C. Feeley, Eds.), American Society for Microbiology, Washington, 1984, pp. 339–340.
11. Arnow, P. M., Boyko, E. J., and Friedman, E. J. Perirectal abscess caused by *Legionella pneumophila* and mixed anaerobic bacteria. Ann. Intern. Med. 98: 184–185 (1983).
12. Kalweit, W. H., Winn, W. C., Jr., Rocco, T. A., Jr., and Girod, J. C. Hemodialysis fistula infections caused by *Legionella pneumophila*. Ann. Intern. Med. 96: 173–175 (1982).
13. Cordes, L. G., Wiesenthal, A. M., Gorman, G. W., Phair, J. P., Sommers, H. M., Brown, A., Yu, V. L., Magnussen, W. H., Meyer, R. D., Wolf, J. S., Shands, K. N., and Fraser, D. W. Isolation of *Legionella pneumophila* from hospital shower heads. Ann. Intern. Med. 94: 195–197 (1981).
14. Para, M. F., and Plouffe, J. F. Production of monoclonal antibodies to *Legionella pneumophila* serogroups 1 and 6. J. Clin. Microbiol. 18: 895–900 (1983).
15. Zanen-Lim, O. G., Van Den Broek, N. J., Rietra, P. J. G. M., Van Ketel, R. J., and Zanen, H. C. Immunochemical analysis of cell envelopes of *Legionella pneumophila* serogroup 1 strains isolated from patients and water during an epidemic in Amsterdam. In: Proceedings of the Second International Symposium on Legionella (C. Thornsberry and J. C. Feeley, Eds.), American Society for Microbiology, Washington, 1984, pp. 268–269.
16. Plouffe, J. F., Para, M. F., Maher, W. E., Hackman, B., and Webster, L. Subtypes of *Legionella pneumophila* serogroup 1 associated with different attack rates. Lancet ii: 649–650 (1984).
17. Moyer, N. P., Lange, A. F., Hall, N. H., and Hausler, W. J., Jr. Application of capillary gas-liquid chromatography for subtyping *Legionella pneumophila*. In: Proceedings of the Second International Symposium on Legionella (C. Thornsberry and J. C. Feeley, Eds.), American Society for Microbiology, Washington, 1984, pp. 274–276.
18. Saravolatz, L. D., Pohlod, D., Helzer, K., Wentworth, B., and Levin, N. Legionella infections in renal transplant recipients. In: Proceedings of the Second International Symposium on Legionella (C. Thornsberry and J. C. Feeley, Eds.), American Society for Microbiology, Washington, 1984, pp. 231–233.
19. Stout, J., Yu, V. L., Vickers, R. M., Zuravleff, J., Best, M., Brown, A., Yee, R. B., and Wadowsky, R. Ubiquitousness of *Legionella pneumophila* in the water supply of a hospital with epidemic Legionnaires' disease. N. Engl. J. Med. 306: 455–468 (1982).
20. Rudin, J. E., Wing, E. J., and Yee, R. B. An ongoing outbreak of *Legionella micdadei*. In: Proceedings of the Second International Symposium on Legionella (C. Thornsberry and J. C. Feeley, Eds.), American Society for Microbiology, Washington, 1984, pp. 227–229.
21. Shands, K. N., Ho, J. L., Meyer, R. D., and Fraser, D. W. Potable water: possible role in epidemic Legionnaires' disease. Paper presented at 20th Interscience Conference on Antimicrobial Agents and Chemotherapy, September 22-24, 1980; Abstract 501.
22. Fisher-Hoch, S. P., Tobin, J. O'H., Nelson, A. M., Smith, M., Talbot, J., and Bartlett, C. L. R. Investigation and control of an outbreak of Legionnaires' disease in a district general hospital. Lancet ii: 307–310 (1981).