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MAJOR ARTICLE

An Outbreak of Legionnaires Disease Caused by Long-Distance Spread from an Industrial Air Scrubber in Sarpsborg, Norway

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Background. On 21 May 2005, the Norwegian health authorities were alerted by officials from a local hospital that several recent patients had received the diagnosis of legionnaires disease; all patients resided in 2 neighboring municipalities. We investigated the outbreak to identify the source and to implement control measures.

Methods. We interviewed all surviving case patients and investigated and harvested samples from 23 businesses with cooling towers and other potential infection sources. The locations of the businesses and the patients' residences and movements were mapped. We calculated attack rates and risk ratios among people living within various radii of each potential source. Isolates of *Legionella pneumophila* were compared using molecular methods.

Results. Among 56 case patients, 10 died. The case patients became ill 12–25 May, resided up to 20 km apart, and had not visited places in common. Those living up to 1 km from a particular air scrubber had the highest risk ratio, and only for this source did the risk ratio decrease as the radius widened. Genetically identical *L. pneumophila* serogroup 1 isolates were recovered from patients and the air scrubber. The air scrubber is an industrial pollution-control device that cleans air for dust particles by spraying with water. The circulating water had a high organic content, pH of 8–9, and temperature of 40°C. The air was expelled at 20 m/s and contained a high amount of aerosolized water.

Conclusions. The high velocity, large drift, and high humidity in the air scrubber may have contributed to the wide spread of *Legionella* species, probably for >10 km. The risk of *Legionella* spread from air scrubbers should be assessed.

Legionnaires disease (LD) was first recognized in 1976 during an outbreak of severe pneumonia among delegates to the 1976 American Legion convention in Philadelphia [1]. The disease was caused by *Legionella pneumophila*, a bacterium often inhaled because of contaminated aerosols. LD affects mainly adults. Cigarette smoking, long-term lung disease, advanced age, and immunosuppression are important risk factors [2]. In Europe, *Legionella* species are the causative organisms in 1.9% of all community-acquired pneumonia cases, in 4.0% of those hospitalized, and in 7.9% of those requiring admission to intensive care units [3]. Outbreaks have previously been linked to a variety of aerosol-producing devices, such as cooling towers, evaporative condensers, air-conditioning systems, mist machines in grocery stores, and whirlpool spas [4–11]. There has been special concern about cooling towers because of their potential to spread contaminated aerosols over wide geographical areas and thus possibly affect a large number of people [8, 12].

In Norway (population, 4.5 million people), LD has been a rare disease, with <25 cases reported annually since the 1980s; about one-half of those cases have been acquired during travel abroad [13]. Previously, only 1 outbreak of LD was reported in Norway [14]. General practitioners are mandated to immediately report single

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cases and outbreaks to the municipal medical officer, who is mandated to report to the Norwegian Institute of Public Health (NIPH).

On 21 May 2005, the 24-h on-call service at the NIPH received a report from the medical department at the hospital in Fredrikstad: an unusual high number of patients ($n = 18$) had been admitted for pneumonia during the previous few days, and, at the time of reporting, 3 of them had received a confirmed diagnosis of LD. Preliminary interviews revealed no common exposures. The municipal medical officer was informed the same day and immediately initiated an outbreak investigation. We describe here the investigation of the largest outbreak of LD in Norway and the identification of an air scrubber as a new source of LD.

METHODS

Setting. Østfold Hospital Trust provides clinical and medical microbiological services to the 250,000 inhabitants of Østfold County, including the 120,000 inhabitants of the industrial twin cities Fredrikstad and Sarpsborg. The municipal medical officers of these 2 cities formed a joint multidisciplinary outbreak-control team that also included epidemiologists from the NIPH. The team coordinated the investigation and public health measures.

Environmental investigation. All cooling towers included in the municipal registers and similar potential sources in the affected area were inspected. The following information was collected from the owners on site: operation and management procedures, cleaning and disinfection routines, environmental conditions (including pH and temperature of circulating water), and irregular events during the previous 2 months, such as seasonal start of operation, accidents, or problems with equipment operation. On the basis of this information and facility location, we assessed the likelihood of the source of the outbreak for each facility. Some facilities were excluded from further investigation. Announcements in the media urged companies with unregistered cooling towers to contact the municipal authorities. The public and the media also reported suspect facilities. All reported facilities were assessed and were followed up if considered relevant. The location of all potential sources was then entered into a geographical information system (GIS).

Epidemiological investigation. We intensified the surveillance and case finding by informing all physicians in the area of the need for vigilance and liberal use of urinary antigen tests. We set up a direct link between the local department of microbiology and the outbreak-control team. Physicians elsewhere in the country and in Europe were informed and encouraged to ask their patients with LD about travels to the affected area [15].

A case patient was defined as a patient who had (1) confirmed pneumonia, (2) laboratory evidence of *L. pneumophila* serogroup 1 (Lp 1) infection, (3) onset of illness between 1 April and 1 July 2005, and (4) lived in or visited Sarpsborg or Fredrikstad in the 2 weeks preceding the date of his or her illness onset.

All case patients or their relatives were interviewed regarding movements and transportation during the 2 weeks before illness onset. The daily movements and the place of residence of each case patient were plotted in the GIS, which also contained information from the National Population Register with the exact location of residences of all inhabitants in Fredrikstad and Sarpsborg.

We performed a retrospective cohort study of the disease risk associated with exposure to each of several potential sources, using proximity of residence to the source as a proxy for exposure. Only cases registered during May were included. We hypothesized that the attack rate would be higher for people living close to the source and would gradually diminish with increasing distance. Around each potential source, we made 5 zones, each with an increasing radius, for a total range of 1000–

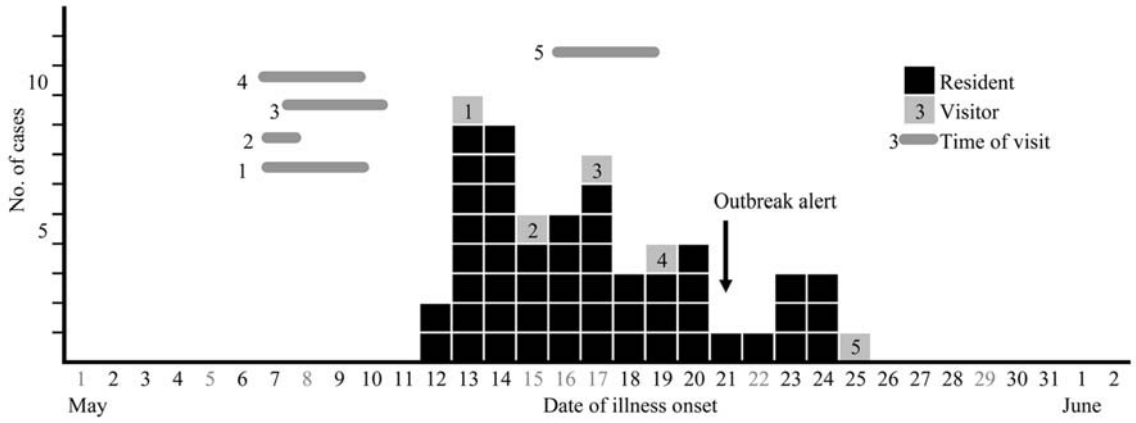

Figure 1. Cases of legionnaires disease ($n = 56$) by date of illness onset, in Sarpsborg and Fredrikstad, Norway, May 2005

Figure 2. Place of residence for case patients with legionnaires disease *(red circles)* and location of cooling towers or other potential sources *(triangles);* most-relevant sources marked with letters A–H), Sarpsborg (northeast) and Fredrikstad (southwest), Norway, May 2005. Orange areas indicate residential areas.

10,000 m. We calculated attack rates for residents living within and outside each zone of increasing radius, and we calculated risk ratios for all 5 zones around each potential source. In addition, we compared each "doughnut-shaped" ring formed by these circles with a reference rate defined as the attack rate among residents living >10,000 m from the potential source. We assumed that only for the true source would the risk ratio diminish gradually with the distance from the source.

Microbiological investigation. Laboratory evidence of LD consisted of either isolation of Lp 1 or a positive *Legionella* urinary antigen test (NOW Legionella Urinary Antigen Test; Binax). Samples from survivors' expectorate and lung tissue specimens from deceased patients were collected, and cultures were performed on buffered charcoal yeast extract–α agar plates. *Legionella* isolates were serotyped (Legionella Latex Test; Oxoid) and were genetically characterized.

At the time of inspection, beginning 23 May, environmental samples were collected from relevant cooling towers and similar installations, local lakes and rivers, and other possible sources. Water samples were taken from the circulation water; in addition, wet surfaces were swabbed. The samples were investigated at local laboratories, with use of standard procedures for *Legionella* isolation and total heterotrophic plate count [16]. *Legionella* isolates were sent to NIPH for serotyping and genotyping, for comparison with patient isolates.

Two different genotyping methods were used for characterization of both patient and environmental isolates: randomly amplified polymorphic DNA (RAPD; Ready-To-Go RAPD Analysis Kit; Amersham Biosciences) with use of 2 different primers and restriction-fragment–length polymorphism with use of the enzyme *Hae*III. Control strains were included [17].

Aerosol-dispersion investigation. We used AirQUIS (http://www.airquis.com/) and its Gaussian puff model INPUFF [18, 19] to describe the transport and dispersion of aerosols emitted from potential sources for the relevant time period, using hourly meteorological information, including wind direction and velocity, outdoor temperature at a height of 25 m, and atmospheric temperature stability between 8 m and 25 m. We assumed that the particle size of the aerosols were 2.5 μm, pipe diameter was 1 m, output velocity was 3 m/s, and emission rate was 100 g/s. The model results were projected onto 1-km

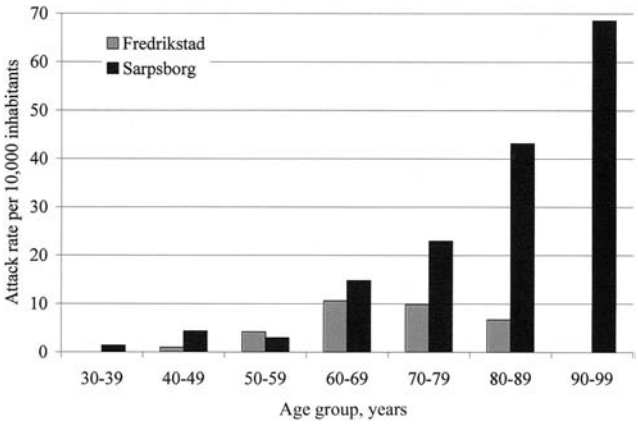

Figure 3. Age-specific attack rates of legionnaires disease (per 10,000 inhabitants), Fredrikstad and Sarpsborg, Norway, May 2005 (n = 51).

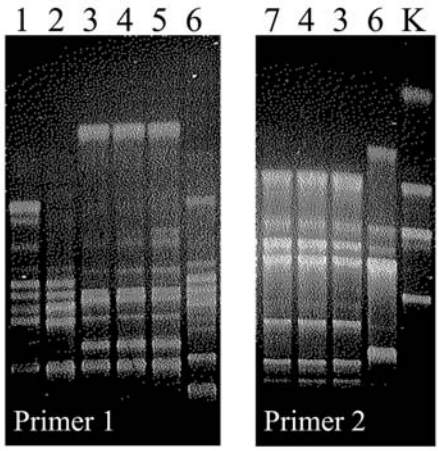

Figure 4. Randomly amplified polymorphic DNA analysis of isolates from patients and environment. *Lane 1,* Control L63; *lane 2,* control P1; *lane 3,* patient 1; *lane 4,* air scrubber company F; *lane 5,* patient 3; *lane 6,* cooling tower company E; *lane 7,* Glomma River; *lane K,* size markers.

square grids, which gave the average relative concentrations within that grid. The zones were calculated hourly and were combined in the GIS for the relevant time period. The focus time period was back-calculated to be 7–11 May, on the basis of the midtime of illness onset (16 May) and a 7-day incubation period [20].

Because the emission rates of aerosols from the various sources were not available, we interpreted the concentrations presented in the model on a relative basis. Also, the model did not represent the initial dispersion of the plume, because of effects of exhaust rates and building-induced turbulence.

The modeled plume distributions were included as layers in the GIS. We then measured the proportion of patients who would have been exposed to each of the sources by either living or visiting within the dispersal region of the aerosol plume during the incubation period.

RESULTS

Descriptive epidemiology. In total, 56 LD cases were diagnosed during the outbreak. The case patients became ill between 12 May and 25 May, with a peak during 13–17 May (figure 1). The mean age of the patients was 69 years (range, 35–94 years; median, 70 years). There were 33 men and 23 women. Fifty of the patients had an underlying disease, of which pulmonary conditions (especially long-term obstructive pulmonary disease), hypertension, and heart conditions were the most common. Ten patients died (case-fatality rate, 17.8%), all of whom were elderly persons (range, 68–94 years; median, 80 years) with underlying medical conditions. Thirty-two of the case patients resided in Sarpsborg, and 19 resided in Fredrikstad (figure 2), for attack rates of 6.4 and 2.7 per 10,000 inhabitants, respectively. Five of the case patients were visitors; 4 during 6–10 May, and 1 during 16–18 May. The age-specific incidence rates increased with age among Sarpsborg residents, whereas, in Fredrikstad, it peaked among those who were 60–69 years of age (figure 3).

Environmental investigation. Twenty-three companies or institutions with 41 aerosol-spreading installations were assessed: 31 cooling towers, 6 air scrubbers, 3 dry coolers with spraying devices, and 1 biological treatment plant. Fifteen of the 23 companies/institutions were considered unlikely to be a source of the outbreak, either because they were not operating during the outbreak, because their management and control regimen was considered of a high standard, or because they were located far from the residency of the majority of the case patients. The last 8 were considered to be potential sources and were evaluated further. Five cooling towers that were not operating according to the national guidelines were immediately closed.

Microbiological results. Lp 1 was cultured from 10 patients; 6 isolates were from lung tissue from deceased patients, and 4 were from surviving patients' expectorate. All isolates showed identical patterns with use of 1 of the primers, whereas all but 2 had identical patterns with use of the second primer. These 2 isolates presented with a single additional PCR fragment with use of the second primer. Restriction-fragment–length polymorphism analysis with *Hae*III showed that the same 2 isolates were missing 1 large restriction fragment, although their patterns were otherwise identical to the remaining isolates. These 2 slightly different isolates were recovered from 1 individual who visited Fredrikstad on 7–9 May and 1 who lived in Sarpsborg.

During 23–26 May, a total of 76 environmental samples were taken from cooling towers and other potential sources in 15 companies or institutions in the 2 municipalities. Lp 1 was isolated from a cooling tower at company E, from an air scrubber at company F, and from a water sample from the river downstream of company F. Only the isolates from the air scrubber and the river proved, by genotyping, to be identical to the patient samples (figure 4).

Cohort study. In the retrospective cohort study, we found that for only 1 of the sources—the air scrubber at company F—did the attack rate and risk ratio increase for each concentric circle and doughnut-shaped zone closer to the source (table 1).

Aerosol-dispersion investigation. During the period of the outbreak, the average daily temperature varied from 6°C to 10°C, with relative air humidity of 50%–80%. The wind direction varied along a northwest-southeast axis, with an average speed of 3–6 m/s [21]. The plume modeled for the air scrubber at company F gave the best fit with the distribution of the cases (table 2 and figure 5).

The source. Company F uses various components of wood to produce wood-based chemicals such as cellulose, lignin-

Table 1. Attack rates (ARs) per 100,000 persons and risk ratios (RRs) for legionnaires disease among residents within circular areas of increasing distance around each potential source, compared with those of residents living outside the zones.

Radius from source (m) and findings	Company							
	A	B	C	D	E	F[a]	G	H
1000								
AR	0.0	98.3	0.0	0.0	0.0	271.6	78.8	0.0
RR	0.0	2.6	0.0	0.0	0.0	**7.3**	2.0	0.0
Cases	0	6	0	0	0	5	2	0
Population	1152	6104	1966	1943	2295	1841	2537	2537
1500								
AR	0.0	113.2	24.6	0.0	0.0	188.9	176.9	0.0
RR	0.0	3.5	0.6	0.0	0.0	**5.8**	5.5	0.0
Cases	0	14	1	0	0	12	13	0
Population	2449	12,367	4065	3877	3845	6352	7350	5440
3000								
AR	108.6	79.8	63.7	11.5	12.7	111.7	120.8	25.1
RR	3.7	2.5	1.7	0.3	0.3	**4.7**	5.6	0.6
Cases	19	18	10	2	2	26	28	5
Population	17,502	22,570	15,708	17,318	15,708	23,270	23,186	19,910
5000								
AR	76.2	72.5	72.8	23.8	35.9	74.9	75.7	23.0
RR	3.4	2.9	2.6	0.5	0.8	**3.4**	23.3	0.4
Cases	31	29	25	11	15	32	31	11
Population	40,663	40,000	34,335	46,157	41,747	42,749	40,948	47,764
10,000								
AR	48.3	55.7	45.4	47.5	46.6	47.6	46.7	49.0
RR	1.6	2.3	2.8	3.0	3.3	**1.6**	1.7	3.5
Cases	35	35	46	45	46	35	38	45
Population	72,468	62,821	101,358	94,835	98,770	73,515	81,318	91,848

NOTE. In Sarpsborg and Fredrikstad, Norway, May 2005 (case patients, 49; population, 120,171). Bold type indicates diminishing risk ratio with increasing radius around the source at company F.

[a] All companies have cooling towers except F, which has an industrial air scrubber.

based binding, and dispersing agents; yeast products; bioethanol; and chemical vanilla. To protect the environment from pollution, several measures were put in place to reduce emissions to air and water, including air-treatment plants (air scrubbers) and biodegradation treatment plants for waste water.

The incriminated air scrubber was used to clean the process air coming from a spray dryer used in lignin production. The process air was mixed with fresh air from outside the plant before entering the scrubber, where it was sprayed with water (figure 6). When the mixture condensed, pollutants and dust particles in the air stayed in the water, while gas containing some evaporated water was released. The temperature of the process air going into the incriminated air scrubber was high (80°C–90°C), and the water used for spraying was circulated in the scrubber. Because there was a large drift of aerosolized water through the pipe, that water had to be continuously replaced by cold water (at 10°C–12°C, at the time of the outbreak). This continual replacement contributed to keeping the temperature in the water at ~40°C. The amount of circulating water in the air scrubber was ~4 m^3. The water was circulated by a pump, had a pH of 8–9, and had a high organic content. The air scrubber expelled >4 m^3 water per hour as aerosol, with an air flow of 60,000 m^3/h and a velocity of ~20 m/s. The

Table 2. Effect of proximity to zone covered by the plume modeled for each potential source of legionnaires disease.

Company	No. of case patients	
	Who reside in zone	Who neither reside in nor visited zone
A	42	2
C	38	6
D, E, and H	43	1
F	**44**	**0**
G	41	4

NOTE. The plume is modeled for 7–11 May 2005, in Sarpsborg and Fredrikstad, Norway. Companies D, E, and H are so close to one another that 1 combined plume was modeled for them. No. of case patients, 49.

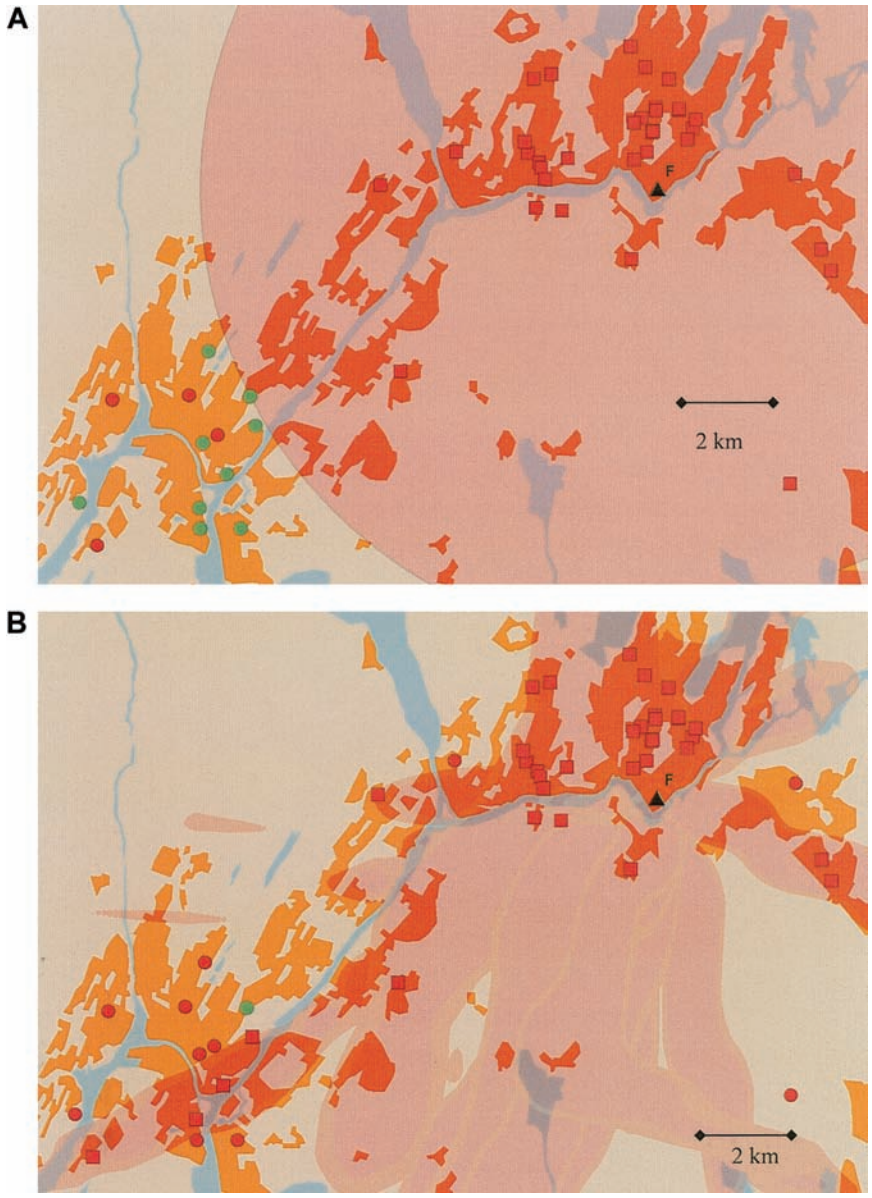

Figure 5. *A,* Residence locations of case patients with legionnaires disease who lived within *(red squares),* visited *(red circles),* and neither lived within nor visited *(green circles)* a 10-km radius of *(A)* or the modeled aerosol-distribution plume from *(B)* the air scrubber at company F *(black triangle)* for the time period of 7–11 May 2005, based on actual wind directions and velocities.

tank of the air scrubber was routinely cleaned with high-pressure hot water every 3–4 weeks; the last time before the outbreak was in late April, but no disinfection was performed. The circulating water was transferred to a storage tank during the cleaning process and was then returned to the air scrubber. The pump and pipes had not been manually cleaned. Both the tank and the pipes had layers of solid scale, which supported biofilm formation.

On 8 June, the air scrubber was closed, and new routines for cleaning and disinfection were implemented. When the air scrubber was restarted, sampling and *Legionella* cultures were performed to assess the treatment. New national regulations were put in place to cover all aerosol-producing installations that could facilitate *Legionella* growth and dispersion.

DISCUSSION

We have identified an industrial air scrubber as the source of the largest LD outbreak in Norway to date, with 56 cases, including 10 deaths. Epidemiological and microbiological investigations, aerosol dispersion modeling, and an assessment of the growth conditions for *Legionella* in the air scrubber all pointed to this novel source of an LD outbreak. Although the use of urinary antigen testing facilitated detection of the out-

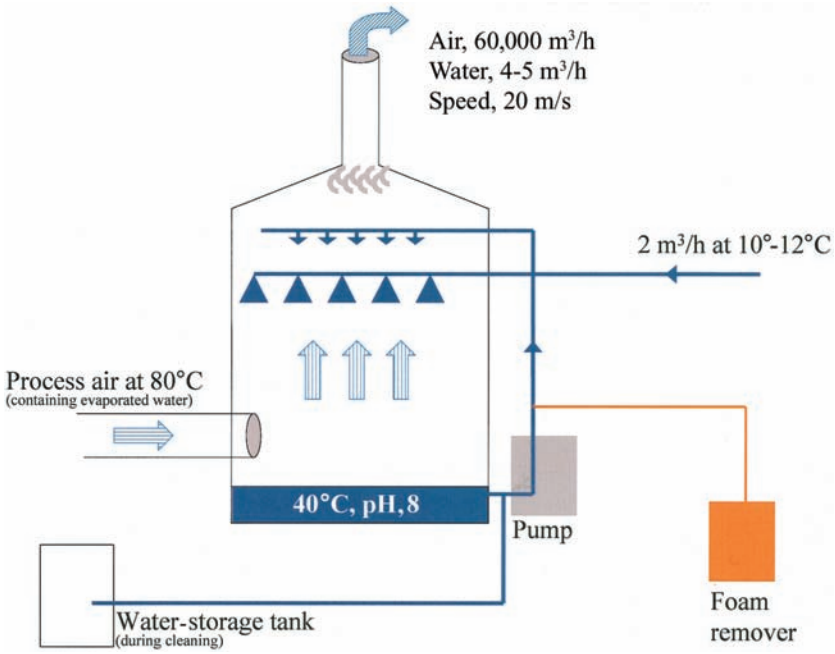

Figure 6. Schematic drawing of the air scrubber at company F

break and the later case finding, we believe that there may have been many more milder cases. After the outbreak alert, empirical therapy to cover for LD was used to treat community-acquired pneumonia in the area.

Until recently, the maximum distance of transmission of *Legionella* was considered to be ~3 km [8]. However, in a cooling tower-related outbreak in Pas-de-Calais, France, in 2003, a distance of 6–7 km was suggested [12]. The present outbreak demonstrates even farther spread. Although an increased risk was clearly demonstrated only for zones up to a distance of 3 km (table 3), 8 case patients stated that they had not been within 10 km of the source, which indicates a larger transmission range from an air scrubber than from cooling towers. Air scrubbers expel air under pressure, which causes a very high velocity. In the incriminated air scrubber, the air velocity was 20 m/s, with a water drift of ~4–5 m^3/h. This would be much higher than the emission velocity and water drift from a cooling tower with a similar amount of circulating water, thus facilitating further spread by the air scrubber. The high emission point and high emission velocity probably also led to low concentration in the immediate vicinity of the scrubber, thus explaining why there were no cases among workers on site.

One could also hypothesize that the long-distance spread was caused by the emission of the same *Legionella* strain from ⩾2 sources in the same period. However, there are several issues that indicate that there was 1 source only: (1) outbreaks of LD are quite rare, and all cases in the whole area occurred during the same short time frame, with no difference between the 2 municipalities; (2) the attack rate increased with age only in 1 of the municipalities—the 1 where the scrubber was located; (3) only for this source did the attack rate gradually increase with closeness to the source; and (4) *Legionella* isolates with the outbreak profile were recovered from only 1 of the sampled environmental sites. Therefore, we consider it unlikely that several sources were involved.

We used a retrospective cohort design to find out which of the several cooling towers and similar installations was the source. The exact time and duration of the exposure was unknown, but we reasoned that older patients would have spent most of the time at home, so we could use residence as proxy for exposure. Furthermore, the information about movements was more prone to errors and bias than was the home address. The method relied on a GIS with complete information on location of each individual's residence. The method may be used in other outbreaks of LD when several sources are under suspicion. If a complete register of residences is not available, home addresses of the case patients and a sample of the population can be collected for a case-cohort approach.

Because it has no aerosol module, our dispersion model was not designed to allow for the interaction among different components of the particles, formation of secondary aerosols, and size change of particles by condensation, evaporation, and coagulation processes. Thus, the model results display only the general characteristics of the aerosol plume, the dilution of the plume, and the direction and extent of the transport. The purpose was to relate the plumes for each source to the patients' residences and not to estimate the actual concentration level.

Table 3. Attack rates (ARs) per 100,000 persons and risk ratios (RRs) for legionnaires disease among residents within the specified doughnut-shaped zones around each potential source, compared with residents living outside the 10,000-m zone.

	Company							
Zone around source, m	A	B	C	D	E	F[a]	G	H
<1000								
AR	0.0	98.3	0.0	0.0	0.0	271.6	78.8	0.0
RR	0.0	4.0	0.0	0.0	0.0	**9.1**	2.8	0.0
Cases	0	6	0	0	0	5	2	0
Population	1152	6104	1966	1943	2295	1841	2537	2537
1000–1500								
AR	0.0	127.7	47.6	0.0	0.0	155.2	228.5	0.0
RR	0.0	5.2	3.0	0.0	0.0	**5.2**	8.1	0.0
Cases	0	8	1	0	0	7	11	0
Population	1297	6263	2099	1934	1550	4511	4813	2903
1500–3000								
AR	126.2	39.2	77.3	14.9	16.9	82.8	94.7	34.6
RR	4.3	1.6	4.8	0.9	1.2	**2.8**	3.3	2.4
Cases	19	4	9	2	2	14	15	5
Population	15,053	10,203	11,643	13,441	11,863	16,918	15,836	14,470
3000–5000								
AR	51.8	63.1	80.5	31.2	49.9	30.8	16.9	21.5
RR	1.8	2.6	5.0	2.0	3.6	**1.0**	0.6	1.5
Cases	12	11	15	9	13	6	3	6
Population	23,161	17,430	18,627	28,839	26,039	19,479	17,762	27,854
5000–10,000								
AR	12.6	26.3	31.3	69.8	54.4	9.8	17.3	77.1
RR	0.4	1.1	2.0	4.4	3.9	**0.3**	0.6	5.5
Cases	4	6	21	34	31	3	7	34
Population	31,805	22,821	67,023	48,678	57,023	30,766	40,370	44,084
>10,000								
AR	29.3	24.4	15.9	15.8	14.0	30.0	28.3	14.1
RR	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Cases	14	14	3	4	3	14	11	4
Population	47,703	57,350	18813	25,336	21,401	46,656	38,853	28,323

NOTE. In Sarpsborg and Fredrikstad, Norway, May 2005 (case patients, 49; population, 120,171). Ref, reference. Bold type indicates diminishing risk ratio with increasing distance from the source at company F.

[a] All companies have cooling towers except F, which has an industrial air scrubber.

The identification of the same *Legionella* genotype from patient samples and the air scrubber but from no other potential source was strong evidence of the involvement of company F. However, it has been reported elsewhere that cooling towers located close to each other may harbor the same *Legionella* genotype [22]. The microbiological results thus need to be interpreted together with results from the epidemiological and environmental investigation.

It is difficult to explain why the air scrubber, after several years of operation, suddenly caused a large outbreak. The epidemic curve indicates a release lasting a few days at most. The scrubber had a nutrient-rich environment that would facilitate thick biofilm prone to sloughing [23], causing a burst of higher *Legionella* concentrations in the circulating water and released aerosol.

At the premises, there was also a biological treatment plant with an open aeration pond, located ~200 m away from the air intake of the air scrubber. These ponds have been shown to harbor large concentrations of *Legionella* species [23, 24]. The pond may have influenced the microbiological conditions in the air scrubber.

This outbreak emphasizes the importance of considering all potential aerosol-producing devices in an LD outbreak. Industrial air scrubbers may, under certain conditions, be very potent disseminators of aerosols, as shown by the widespread transmission of at least 10 km in this outbreak. This study also

demonstrated the usefulness of GIS as a tool for outbreak investigation.

Acknowledgments

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Potential conflicts of interest. T.J. is an employee of Geodata, a Norwegian company that markets the GIS software used in the investigations. All other authors: no conflicts.

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