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Legionellosis linked with a hotel car park – how many were infected?

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SUMMARY

An outbreak of legionellosis associated with a hotel in Sydney, Australia, and the subsequent epidemiological and environmental investigations are described. Four cases of Legionnaires' disease were notified to the Public Health Unit. A cross-sectional study of 184 people who attended a seminar at the hotel was carried out. Serological and questionnaire data were obtained for 152 (83%) of these. Twenty-eight (18%) respondents reported symptoms compatible with legionellosis. Thirty-three subjects (22%) had indirect fluorescent antibody (IFA) titres to *Legionella pneumophila* serogroup 1 (Lp-1) of 128 or higher. The only site which those with symptoms of legionellosis and IFA titre $\geqslant 128$ were more likely to have visited than controls was the hotel car park (adjusted odds ratio [OR] 14·7, 95% confidence interval [CI]: 1·8–123·1). Those with symptoms compatible with legionellosis, but whose IFA titres were < 128 were also more likely to have visited the hotel car park (adjusted OR 4·4, 95% CI: 1·5–12·9). Seroprevalence of Lp-1 antibodies was higher in those who attended the seminar than in a population sample of similar age. Findings suggested that the 4 cases represented a small fraction of all those infected, and highlighted difficulties in defining illness caused by Lp-1 and in interpreting serology.

INTRODUCTION

Since 1976 when the first reported outbreak of Legionnaires' disease occurred [1], numerous outbreaks, both community acquired and nosocomial, have been reported worldwide [2–5]. They include outbreaks associated with a hot spa [6], fountains [7], a grocery mist machine [8], hot water systems [9], whirlpool baths on a cruise ship [10] as well as evaporative condensers and cooling towers [11, 12].

New South Wales (NSW), one of the eastern states of Australia, covers an area of about 800000 km^2. It has a population of *c.* 6 million people, 3·5 million of whom live in the capital city, Sydney [13]. Three major outbreaks (81 cases) of Legionnaires' disease have occurred in NSW since 1987 [14, 15]. All occurred in the month of April, and were associated with cooling towers or evaporative condensers. *L. pneumophila* serogroup 1 (Lp-1) was identified as the causative micro-organism in each outbreak. Under the NSW Public Health Act (1991), legionella infection is a notifiable disease, and artificial habitats of legionellae, such as cooling towers, evaporative condensers and warm water systems must be registered with the local

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government authority. Detailed requirements for installing, operating and maintaining these systems are provided [16].

Outbreak

Between 22 and 27 April 1993, the Western Sector Public Health Unit (PHU) in western Sydney was notified of 4 cases of Legionnaires' disease. *L. pneumophila* serogroup 1 (Lp-1) was cultured from sputum specimens from all 4. All had onset dates of 13 or 14 April. PHU staff interviewed cases or their relatives to determine cases' movements in the 10 days prior to the onset of illness. Two cases had attended an investment seminar for retired people at a Sydney hotel on the afternoon of 7 April. Another case had eaten at a takeaway food shop 15 m from the hotel on the same day. The fourth case, a 40-year old male smoker, who was in good health, had walked in the same area that day. He reported visiting a building 500 m from the hotel, but had not apparently come any closer.

As required by the NSW Public Health Act 1991, the local council keeps a register of all evaporative condensers and cooling towers in the area. Environmental health officers inspected and took water samples from all known cooling towers and evaporative condensers within 150 m of the hotel between 26 and 29 April.

Both of the hotel's cooling towers were the forced draft counterflow type. Environmental health officers reported that access to these towers was difficult, hindering inspections, maintenance and internal cleaning. They also observed algae in the flexible duct of the exhaust system, suggesting inadequate cleaning. Maintenance records indicated that both towers had been drained, flushed and refilled 3 weeks, and disinfected 2 weeks before the seminar. Prior to this, the cooling towers had apparently been maintained in accordance with NSW Health Department guidelines.

The NSW Health Department's Division of Analytical Laboratories, cultured bacteria from water samples from cooling towers, and carried out Restriction Fragment Length Polymorphism (RFLP) typing on clinical and environmental isolates. Lp-1 was isolated from the samples taken on 26 April from both the first floor and rooftop cooling towers of the hotel. These yielded counts of $2{\cdot}8 \times 10^7$ and $3{\cdot}4 \times 10^6$ colony forming units (c.f.u.)/l, respectively, of Lp-1. One other cooling tower from a nearby building had an Lp-1 count of $4{\cdot}5 \times 10^5$ c.f.u./l. Lp-1 was not detected in samples from any other site.

Lp-1 isolates cultured from the 4 cases and the cooling towers of the hotel were all of the same restriction fragment length polymorphism (RFLP) type [17]. No other cultures from environmental samples matched those from the cases.

Although the prevalence of the particular RFLP type isolated from the environmental samples is unknown, investigations implicated the cooling towers at the hotel as the most likely source of infection. To identify previously unrecognized cases of legionellosis and to confirm the site of environmental exposure, we conducted a cross-sectional study of people who attended the investment seminar at the hotel on the afternoon of 7 April 1993.

METHODS

Cross-sectional study

We contacted, by mail, all of the 184 people who were registered as attending the seminar and asked them to attend a clinic on 3 or 4 June 1993. At the clinic, subjects completed a self-administered questionnaire about possible risk factors for legionellosis, including age, sex, occupation, chronic illnesses, medications, smoking history and alcohol intake. Using a standardized questionnaire, interviewers recorded details of subjects' movements at the hotel and around the local area on 7 April and of any illness between 8 April and 26 April. To aid recall and minimize bias, we asked subjects about illness in the period between the seminar and the Anzac Day Public Holiday on 27 April, an important public holiday in Australia, and a period which included the Easter holiday. A 10 ml blood sample was collected from each subject, to be tested for antibodies to *L. pneumophila*.

Indirect fluorescent antibody (IFA) testing, as described by Wilkinson [18], was performed for *L. pneumophila* serogroups 1–6 (Lp1–6) using American Type Culture Collection (ATCC) strains and a clinical isolate from one of the index patients. Twofold dilutions of sera from 1:64 to 1:256 were screened using the polyvalent antigen. Sera in which total antibody titre were $\geqslant 128$ were titred to end point with total immunoglobulin and antihuman IgM $F(ab')_2$ fragment conjugates. In addition, sera were tested using the same range of dilutions with the monovalent antigens and total immunoglobulin to determine serogroup reactivity.

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We defined clinical illness compatible with legionellosis as illness characterized by fever and/or two or more of the following symptoms: cough, shortness of breath, chest pain, muscle aches and pains, severe headache, dizziness and diarrhoea. We classified subjects into 4 groups according to the presence or absence of clinical illness compatible with legionellosis, and titres to Lp-1 of < 128 or $\geqslant 128$.

The Bureau of Meteorology supplied data (daily minimum and maximum temperatures, wind direction and speed, and rainfall) from the 2 closest meteorological stations.

Data were entered in a database [19] and analysed using Epi Info [20] and Statistix [21] software. Data were cross-tabulated and crude odds ratios (ORs) for exposure variables and possible risk factors, with their 95% confidence intervals (CI), were calculated. The Yates corrected χ^2 statistic or, where an expected cell was less than 5, Fisher's exact test, was used [20]. We used unconditional logistic regression [22] to evaluate the independent effects of exposure variables and risk factors. Variables used in the logistic model were selected according to biological plausibility and our findings from cross tabulations.

Population seroprevalence to Lp-1

To compare seroprevalence of Lp-1 antibodies in the seminar attendees with the general population, we tested sera from 434 randomly selected subjects of similar age (50–80 years). These subjects lived in a semi-urban area on the fringe of Sydney and were enrolled in a population-based cohort study. Similar laboratory testing methods were used.

RESULTS

Of the 184 people contacted, 152 (83%) participated, comprising 92 males and 60 females. All lived in the greater Sydney area. They were in the age range 43–79 years (mean age 61 years). Eight (5%) were current smokers, and 54 (36%) were past smokers. Eighty-nine (59%) reported they drank alcohol regularly. The most common chronic medical conditions reported were hypertension (33%) and arthritis (26%). Thirteen (9%) reported a chronic respiratory disease such as chronic bronchitis, emphysema or asthma, and 15 (10%) reported angina or heart failure. Thirty (20%) reported no risk factors.

Twenty-eight subjects (18%) reported symptoms compatible with legionellosis in the period 8–26 April 1993. The duration of illness range from 2–63 days (median 12 days).

Table 1. *Serological results to Lp-1* for 152 subjects who attended the seminar on 7 April 1993, and for a population sample of 434 randomly selected adults from another study*

IFA titre to Lp-1* (total antibody)	Number (%)	
	Seminar attendees	Population sample
< 128†	119 (78)	416 (96)
128	12 (8)	17 (4)
256	12 (8)	0 (0)
512	4 (3)	0 (0)
$\geqslant 1024$	5 (3)	1 (0·2)
Total	152 (100)	434 (100)

* *Legionella pneumophila* serogroup 1.

† Statistically significantly difference between seminar attendees and population sample ($\chi^2 = 41{\cdot}5$, $P < 0{\cdot}0001$).

Table 2. *Subjects who participated in cross-sectional study, grouped by serological results and symptoms reported*

Group	Males	Females	Total no.
A*	6	3	9
B†	9	10	19
C‡	19	5	24
D§	58	42	100
Total	92	60	152

* A, symptoms compatible with legionellosis and IFA titre $\geqslant 128$.

† B, symptoms compatible with legionellosis and IFA titre < 128.

‡ C, IFA titre $\geqslant 128$ and no symptoms compatible with legionellosis.

§ D, IFA titre < 128 and no symptoms compatible with legionellosis.

Thirty-three subjects (22%) had IFA titres to Lp-1 $\geqslant 128$ (Table 1). Of these, 21 (64%) had titres $\geqslant 256$ and 9 (27%) were $\geqslant 512$. One person had an IFA titres of 256 to *L. pneumophila* serogroup 4 and a titre to Lp-1 of < 128. Five of the 33 with total antibody titres $\geqslant 128$ had IgM titres $\geqslant 64$. Two had titres of 64, 1 a titre of 128, and 2 had titres of 512. All reactive sera responded at similar titres to both the ATCC and clinical strains.

Nine (6%) subjects (Group A) had illness compatible with legionellosis and IFA titres to Lp-1 $\geqslant 128$ (Table 2). Nineteen (13%) subjects (Group B)

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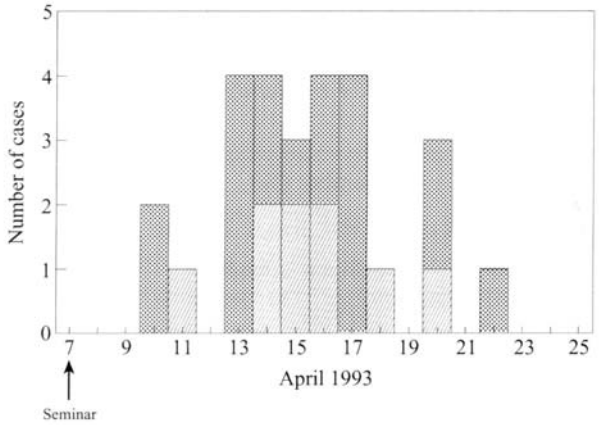

Fig. 1. Date of illness onset for 27 subjects with illness compatible with legionellosis (not reported by 1 subject), western Sydney, April 1993. ▨, Group A (titres ⩾ 128); ⊡, group B (titres < 128).

Table 3. *Frequency of symptoms compatible with legionellosis in Group A and Group B subjects*

	Group A* ($n = 9$)		Group B† ($n = 19$)		Total ($n = 28$)	
Symptom	No.	(%)	No.	(%)	No.	(%)
Muscle aches or pains	5	(56)	16	(84)	21	(75)
Fever‡	8	(89)	7	(37)	15	(54)
Cough	5	(56)	10	(53)	15	(54)
Severe headache	3	(33)	11	(58)	14	(50)
Shortness of breath	3	(33)	9	(47)	12	(43)
Chest pain	2	(22)	8	(42)	10	(36)
Dizziness	2	(22)	7	(26)	9	(32)
Diarrhoea	2	(22)	5	(37)	7	(25)

* A, symptoms compatible with legionellosis and IFA titre ⩾ 128.

† B, symptoms compatible with legionellosis and IFA titre < 128.

‡ Statistically significant difference between Group A and Group B, $P = 0{\cdot}02$, Fisher exact test.

reported compatible illness but IFA titres < 128. Twenty-four (16%) subjects (Group C) had IFA titres ⩾ 128 but reported no compatible illness. One hundred (66%) subjects (Group D) reported no compatible illness and also had IFA titres < 128.

Incubation period following presumed exposure at the seminar ranged from 3–15 days (median 8 days) (Fig. 1). There was no difference in median incubation period between groups A and B. Fever was the only symptom reported more frequently by group A than group B ($P = 0{\cdot}02$, Fisher exact test) (Table 3). There was no difference between the two groups in reported frequency of other symptoms or in the number of symptoms reported. There was also no significant difference in median duration of illness between groups A and B. However, 6 subjects from group A (67%) and 10 subjects from group B (53%) consulted a doctor.

We compared IFA titres to Lp-1 in groups A and C. The geometric mean titre for group A was 406, which was significantly higher than that of 228 for group C ($P = 0{\cdot}04$). Of the 5 subjects with IgM titres of 64 or higher, 2 were from Group A and 3 from group C.

Using logistic regression, we compared risk factors for each of groups A, B and C separately with group D. Group D was the reference group in each model. Results, for a model including age, sex, current smoking status and use of the hotel car park, and for a model with use of car park lift and car park ramp, to gain entrance to the hotel, replacing use of car park per se are shown in Table 4. The only site which subjects in group A were more likely to have visited than group D was the hotel car park. Respondents in group A were over 14 times more likely to have used the car park than those in group D. When use of either the car park ramp or the car park lift, replaced car park in the model, subjects in group A were 14 times more likely to have used the car park ramp than those in group D. They were no more likely to have used the car park lift. Those in group A were also 29 times more likely to be current smokers than those in group D. Those in group B were over 4 times more likely to have used the hotel car park than those in group D, but they were no more likely to have used the car park ramp or lift than group D. There was no difference in age, sex and current smoking status between members of groups B and D. We found no difference in age, current smoking status, or geographic sites visited between members of groups C and D. Although there was no statistically significant difference in sex distribution between these groups, 19/24 (79%) of group C were males, compared with 58/100 (58%) of group D.

Other than the hotel car park, no other sites within the hotel or elsewhere were more likely to have been visited by groups A or B than those in group D. The hotel car park was an indoor underground car park of three levels. Access to the car park was from the street adjacent to the hotel entrance. Cars entered via a ramp to the first level, and either parked on this level or proceeded down to the lower levels. A lift located at the entrance end of the car park operated between

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Table 4. *Adjusted odds ratios for risk factors for subjects reporting symptoms compatible with legionellosis and/or with IFA titres to Lp-1 of ⩾ 128*

	Adjusted odds ratios* and (95% CI)		
Risk factor	Group A†	Group B†	Group C†
Age	1·1 (1·0–1·2)	1·0 (0·9–1·1)	1·1 (1·0–1·1)
Male sex	1·0 (0·2–5·0)	0·6 (0·2–1·8)	2·6 (0·9–7·6)
Current smoking	29·6 (2·1–424·4)	2·9 (0·3–33·2)	1·2 (0·1–12·3)
Car park	14·7 (1·8–123·1)	4·4 (1·5–12·9)	0·9 (0·3–2·3)
Car park lift‡	1·0 (0·2–5·9)	2·5 (0·9–7·3)	0·3 (0·1–1·3)
Car park ramp‡	13·6 (2·5–73·1)	2·9 (0·9–9·6)	2·0 (0·5–7·5)

* Adjusted using logistic regression for age, sex, current smoking history and use of car park (or use of carpark ramp and lift).

† Compared with Group D (no clinical symptoms compatible with legionellosis and IFA titre < 128).

‡ These variables replaced car park in the second model.

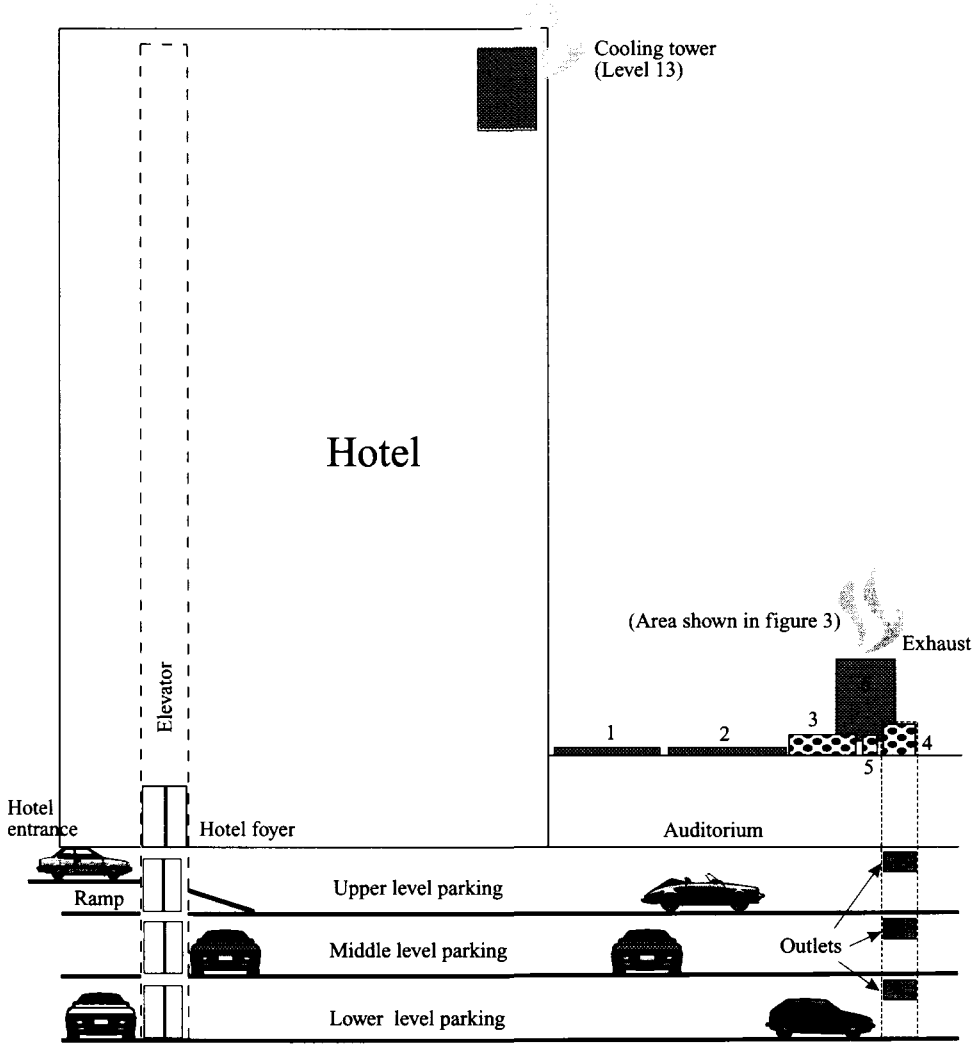

Fig. 2. Cross-sectional view of the hotel and car park. 1, Sundeck; 2, Swimming pool; 3, Cooling tower air intake; 4, Car park air intake; 5, Auditorium air intake; 6, Cooling tower (Level 1).

the car park and the hotel foyer. Access to the hotel could also be gained by walking up the ramp out of the car park to the street (Fig. 2).

The fresh air intake for the car park was *c.* 30 m from the hotel first floor cooling tower, and on the same level (Fig. 3). Air from this intake was not

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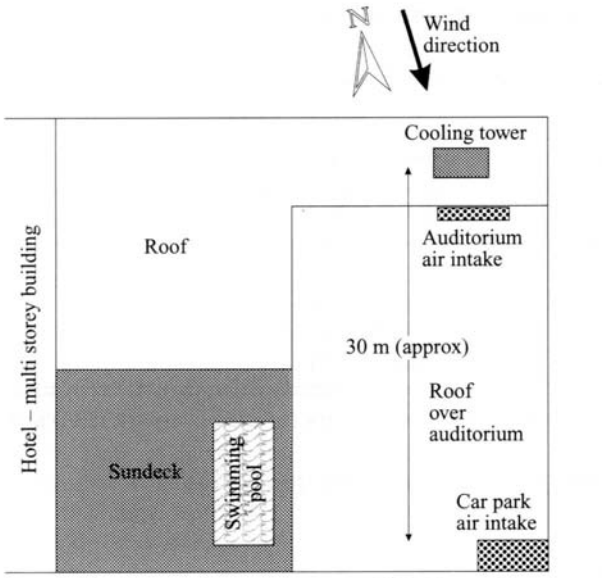

Fig. 3. Birds' eye view of the rear of the hotel, Level 1.

filtered and was ducted around, and released into the car park. An engineering inspection indicated that it was possible for contaminated plume to have entered the car park through the fresh air intake. Wind on the afternoon of 7 April was from the north, at 7–9 km/h which would have directed plume from the tower towards the car park air intake. The air intake for the auditorium where the seminar was held was situated directly below the cooling tower. Air entering through this intake was filtered before being ducted through the auditorium.

Those who attended the seminar were significantly more likely to have IFA titres of $\geqslant 128$, than the population sample ($\chi^2 = 41{\cdot}5$, $P < 0{\cdot}0001$) (Table 1).

DISCUSSION

The finding that 2 of the 4 cases of Legionnaires' disease initially recognized in this outbreak had attended the same seminar allowed us to identify a group of people who were potentially also exposed to Lp-1. Our cross-sectional study of seminar attendees suggested that the 4 cases represented only a small fraction of those infected. It highlighted difficulties in defining illness caused by Lp-1 and in interpreting serology. Despite this, it provided strong epidemiological evidence that the hotel car park was an important site of exposure to Lp-1.

Infection with Lp-1 has been described as causing 2 distinct clinical syndromes: Pontiac fever (an acute self-limited influenza-like condition without pneumonia) and Legionnaires' disease (a severe bacterial pneumonia typically occurring in elderly or immunocompromised individuals) [23, 24]. Lp-1 is also known to cause asymptomatic infection [24]. Host, rather than bacterial or other exposure-related factors appear to determine the clinical expression of infection, since cases of both Pontiac fever and Legionnaires' disease have frequently resulted from a common source of exposure [1, 3, 4, 12, 25, 26]. It seems likely, and our results suggest, that Lp-1 infection does not cause 2 distinct syndromes. Rather, legionellosis is a continuum of clinical signs, ranging from asymptomatic infection through mild respiratory illness and Pontiac fever, to Legionnaires' disease.

Case definitions used in outbreak investigations vary [2, 7, 27]. In our cross-sectional study, 18% subjects reported symptoms compatible with a sensitive clinical case definition. Our case definition was chosen to identify as many cases of Lp-1 infection as possible, but as a result may have included some people with illnesses due to other causes. General Practice Sentinel Surveillance figures suggest that there were low levels of influenza-like illness (*c.* 8/1000 consultations) in the community at that time (Western Sector Public Health Unit, unpublished data).

Incubation periods for legionellosis reported in the literature vary. Our subjects reported illness onset up to 15 days following presumptive exposure at the seminar. As symptoms of legionellosis are often vague and general, their onset may be difficult to determine and subject to recall bias. Our findings suggest that investigation of cases' movements should not be restricted to the 10 days prior to reported onset, as was then recommended in New South Wales [28].

Serological diagnosis of legionellosis usually requires demonstration of a fourfold rise in antibody titre between paired acute and convalescent phase sera [24]. Up to 25% of culture-confirmed cases fail to seroconvert [29, 30]. When seroconversion occurs, it takes place within 4 weeks for 90% of cases, and by 6–10 weeks for the remainder [29]. Factors influencing seroconversion are unknown. We were able to collect only 1 sample from our subjects, 8 weeks following presumptive exposure. Thus we could not readily distinguish recent or past infection. The high prevalence in subjects (22% had IFA titres $\geqslant 128$) compared with the population sample (4%) suggested that the seminar attendees were indeed a group with more exposure to Lp-1. The population sample were likely to have had similar lifetime opportunities for exposure to Lp-1. Although resident in a semi-urban

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area, most had moved there from Sydney when they retired.

We classified subjects into 4 groups based on clinical symptoms and serology. Group A (compatible symptoms and titre $\geqslant 128$) included the 2 seminar attendees diagnosed with Legionnaires' disease during the outbreak. Group B included subjects with compatible symptoms but titres < 128. Thus group B may have included cases of legionellosis who failed to seroconvert, as well as people with illnesses due to other causes. Interestingly, this group included 2 subjects who had been hospitalized with pneumonia at the same time as the outbreak cases, but who were not diagnosed with Legionnaires' disease. Groups A and B showed little difference in frequency and duration of symptoms, indicating that these 2 groups were clinically similar, and supporting the hypothesis that Group B contained cases of legionellosis who did not seroconvert.

Group C comprised subjects with an IFA titre of $\geqslant 128$ but who did not report symptoms compatible with legionellosis. Thus this group may have included subjects with previous seroconversion or those with asymptomatic recent infections. The higher geometric mean titre in group A could indicate that this group were more likely to have had a recent infection or reinfection. Surprisingly, 3 of the 5 subjects with elevated IgM titres fell into group C, including 1 with a total antibody titre of $\geqslant 1024$ and IgM titre of 512. This finding highlights the limited usefulness of IgM titres in the diagnosis of legionellosis.

Misclassification of case status would tend to mask any associations between risk factors and outcome. Despite this, logistic modelling strongly implicated the car park as an important site of Lp-1 infection. Engineering inspections supported this hypothesis. The finding that cases were more likely to have used the car park ramp than the lift could be related to where they parked within the car park. The car park has three levels. Those who parked on road level were perhaps more likely to have used the ramp because it is visible from this level, while those who parked on the lower levels may have been more likely to have used the lift. The road level is also closest to the fresh air intake and contaminated air may have been more concentrated closest to this intake. Further ducting around the car park to the lower levels may have diluted the level of contamination. Unfortunately, we did not ask where subjects parked in the car park.

Two of the original outbreak Legionnaires' disease cases did not attend the seminar or use the hotel car park, suggesting that infection also occurred by inhaling contaminated discharge external to the hotel. One of the cases did not go within 500 m of the hotel. Also, 2 of the 9 subjects in group A, and 7 of the 19 in group B did not use the car park. Legionella infection can occur up to 2 miles from the source [31, 32]. Thus the Legionnaires' disease cases identified during the outbreak and the additional suspected cases found in the cross-sectional study probably represent only a small fraction of all those in the community who were infected. Those who attended the seminar were at greater risk of developing Legionnaires' disease compared with the general population, because of their age. Drift from the cooling tower had the potential to infect local residents, passers-by, hotel staff and other visitors to the hotel; none of these groups was investigated.

Our findings support the notion that passive surveillance is insufficient for detecting small clusters [33, 34]. They imply that when apparently sporadic cases occur, there may in fact be undiagnosed cases in the community, possibly infected from a common source. Our results demonstrate the difficulties in defining illness caused by Lp-1, and in interpreting serology. They suggest that Lp-1 causes illness characterized by fever and respiratory symptoms ranging from mild to severe, and that some cases fail to seroconvert. To identify as many cases as possible, case definitions should be sensitive enough to include this wide spectrum of illness. We were unable to answer the question 'How many were infected?'. We suggest this is likely to be so for most outbreaks of legionellosis.

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