

“Lifeguard Lung”: Endemic Granulomatous Pneumonitis in an Indoor Swimming Pool

ABSTRACT

Objectives. Two sequential outbreaks of respiratory disease among lifeguards at an indoor swimming pool with water spray features were investigated.

Methods. Questionnaires were administered to recreation center employees following each outbreak. Respondents reporting 2 or more pool-related symptoms were offered clinical evaluation, including bronchoscopy with bronchoalveolar lavage and transbronchial biopsy. Pool air and water were sampled for fungi, bacteria, amoebae, endotoxin, and respirable particulates.

Results. Thirty-three lifeguards had noncaseating granulomas on biopsy and/or bronchoalveolar lavage lymphocytosis. Attack rates for the outbreaks were 27% and 65%. Case patients had higher cumulative hours of work and tended to work more hours per week. Analyses indicated increased levels of endotoxin in pool air and water (relative to control pools) and gram-negative bacterial colonization of water sprays. Use of water spray features generated a 5.2-fold increase in the number of respirable particles and up to an 8-fold increase in air endotoxin levels.

Conclusions. Lifeguards in this indoor swimming pool developed granulomatous lung disease associated with endotoxin-containing respirable bioaerosols from water spray features, which ventilation system improvements did not prevent. (*Am J Public Health.* 1998;88:1795-1800)

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Aerosolized water contaminants can cause febrile respiratory illness and hypersensitivity pneumonitis,¹ but these diseases have not been associated with swimming pools. In this article, we report 2 sequential outbreak investigations of granulomatous pneumonitis among lifeguards in an indoor swimming pool in which extensive water spray features disseminated pathogenic bioaerosols.

In March 1989, a 24-year-old lifeguard developed persistent cough, chest tightness, progressive dyspnea on exertion, eye irritation, and headache without fever. His symptoms worsened toward the end of his work shift, persisted through the evening, resolved by the next morning, and recurred several hours after he returned to work. In August 1989, physical examination revealed bibasilar end-inspiratory crackles. Chest radiograph showed subtle, diffuse interstitial opacities. Pulmonary function, including lung volume and diffusing capacity, was normal. However, the patient had significant exercise-induced hypoxemia. A marked T-helper (CD4+) predominant lymphocytosis was present in bronchoalveolar lavage. Transbronchial biopsies yielded multiple noncaseating granulomas. Special stains and cultures for acid fast bacteria and fungi were negative. Shortly after this patient was evaluated, 3 other lifeguards from the same pool were found to have similar symptoms, signs, and pathologic results. The pool was closed for further investigation on October 1, 1989.

The indoor pool where the lifeguards worked was located in a large municipal recreation center. The swimming area consisted of 3 separate pools joined by two 4-foot (120-cm) waterfalls. The pool area contained 3 wall spouts, 4 fan sprays, 4 bridge sprays, a large and a small water slide, a levitator pump, a “bubbler,” and a “mushroom” fountain. Two hot tubs located in an alcove

behind the lap pool were disinfected with hydrogen peroxide and a bromine solution. Pool water was disinfected with chlorine and recirculated through the water spray features. Discussion with aquatics supervisors revealed persistently increased combined chlorine levels and alkaline pool water, but review of logs showed that water chemistry parameters consistently met current standards. Water spray features ran continuously when the pool first opened in November 1986. Within several months, the guards complained of oppressive humidity when water spray features were in use, and an hourly rotation system was devised to keep some of the features on at all times and others on intermittently. Despite this system, the lifeguards frequently turned off the water spray features in an effort to improve air quality. Employee health records indicated that at least 10 lifeguards had experienced pool-related respiratory and systemic symptoms during the 3 years since the pool had opened. A number of lifeguards had quit as a result of these symptoms.

Following extensive ventilation system and engineering improvements, the pool reopened in May 1990. Within 3 months, we recognized a second outbreak of granulomatous lung disease among both newly hired and

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previously healthy rehired lifeguards, leading to pool reclosure.

Methods

Our investigation included administration of a supplemented American Thoracic Society respiratory disease questionnaire to all current recreation center employees and former pool employees who could be located by the Colorado Health Department; clinical evaluation of respondents with pool-related symptoms; and industrial hygiene and ventilation system assessment of the pool.

Epidemiology

Following recognition of the first outbreak, we distributed questionnaires for case ascertainment to 67 current or former pool employees and 71 current nonpool employees of the recreation center. When the pool reopened in May 1990, the questionnaire was distributed to all 23 lifeguards as part of a baseline medical evaluation. Three months later, 18 lifeguards and 12 nonpool employees completed the questionnaire as part of the second outbreak investigation.

Recreation center employees who reported at least 2 of 4 symptoms (frequent cough, recurrent wheezing or chest tightness, dyspnea, or fever) occurring on days or evenings after working at the recreation center were offered further diagnostic evaluation. We obtained informed consent for bronchoscopy with bronchoalveolar lavage and transbronchial biopsies.

We defined a definite case of pool-related lung disease as involving a person with 2 work-related symptoms and both of the following: (1) bronchoalveolar lavage lymphocytosis greater than 33% (based on the 95% confidence interval for percentage lymphocytes in young, healthy nonsmokers)² and (2) transbronchial biopsies showing macrophage aggregates, noncaseating granulomas, bronchiolitis with perivascular lymphocyte accumulations, or interstitial fibrosis. We defined a probable case as involving a symptomatic individual with either bronchoalveolar lavage lymphocytosis or an abnormal biopsy, with the intent of addressing misclassification of disease due to transbronchial biopsy sampling error. We combined definite and probable cases for many of the analyses, and we defined nonpatients as asymptomatic lifeguards and clinically evaluated lifeguards who did not meet the case definition criteria. Symptomatic lifeguards not undergoing evaluation ($n = 14$) were not included in analyses. Smokers were defined as those who had smoked at least 20 packs of cigarettes or at

least 1 cigarette per day for at least 1 year, and ex-smokers were defined as those who had stopped smoking more than 1 month before evaluation.³ Latency was defined as the interval between date of hire at the pool and onset of pool-related symptoms. We calculated each individual's cumulative exposure by multiplying the number of hours worked per week by the total number of weeks worked at the pool.

Industrial Hygiene

Ventilation system and particle size analysis. We examined the pool ventilation system by direct visualization, fiber-optic inspection of ducts not easily accessed, and passing of cable-mounted television cameras through ductwork. Smoke testing and tracer gas analysis were used to assess air movement and ventilation efficiency. We measured the effect of pool water features on respirable aerosol levels with a Las-X 16-channel forward light scattering spectrophotometer (Particle Measuring Systems, Boulder, Colo) in September 1990 after the pool had closed the second time. The Las-X was set to average the number of particles counted over a 10-minute sampling period in the size range of 0.1 to 7.5 μm diameter. During the sampling periods, water feature activity and ventilation system settings were varied to assess their relative contributions to aerosol levels at a fixed instrument location.

Microbial sampling. We tested air and water from the hot tub and multiple pool areas for microbial contaminants that have been associated with outbreaks of hypersensitivity pneumonitis and other febrile respiratory illnesses, including gram-negative bacilli, legionella, thermophiles, fungi, and amoebae. Details of sampling methods, media, and incubation temperatures and times are available on request. In the case of both outbreaks, samples were obtained from the same sites several times before and after the pool closed.

Fungi. Air, water, and swab samples were obtained from a variety of sites in the recreation center, including the pool, classrooms, and gymnasium, and these samples were shipped at room temperature via overnight courier to the University of Michigan Allergy Research Laboratory for analysis. We obtained duplicate air samples from outdoors and from numerous areas throughout the building, including the hot tub area, office, locker rooms, slide area, basement pump room, and wading pool, using the sixth stage of 2 Andersen microbial air samplers (Andersen Samplers Inc, Atlanta, Ga) with malt extract/streptomycin agar plates for impaction. Samples were taken at a calibrated airflow of 28.3 L per minute, with a sampling duration from 1 to 2 minutes.

Fungi recovered were identified to the level of genus. Two personal Burkard spore traps (Burkard Manufacturing Co Ltd, Hertfordshire, England), calibrated to sample at a flow rate of approximately 10 L per minute, were used to collect paired samples for analysis of nonviable fungi. Swab samples were obtained from the ventilation system and from environmental surfaces in the pool. Water samples from the deep pool, lap pool, wading pool, hot tubs, and water spouts were sent as bulk samples, plated, and identified to the level of genus.

Bacteria. We obtained air samples for bacteriologic analysis using the sixth stage of 2 Andersen microbial air samplers, a 28.3 L per minute flow rate, 1- or 2-minute sampling times, and nutrient, MacConkey, or tryptic soy (for thermophiles) agars. In addition, air was passed through sterile glass impingers containing 0.85% saline and 10% sodium thiosulfate at a calibrated flow rate of 1 L per minute via SKC universal flow sampling pumps (SKC Inc, Eighty Four, Pa). We obtained duplicate water samples for bacteriologic analysis by immersing sterile polycarbonate bottles containing 2 mL of 10% sodium thiosulfate solution directly into pool water or under the outflow stream of the pool water features. Within 18 hours, aliquots of 100 mL, 10 mL, 1 mL, 0.1 mL, and 0.01 mL were filtered by vacuum suction through 0.45- μm cellulose acetate filters (47 mm diameter; Gelman Sciences, Ann Arbor, Mich). Filters were then placed on petri dishes with nutrient, MacConkey, or R2A agar for culture. Isolates of gram-negative bacilli were identified by use of the API20E system (Analytabs Products, Plainview, NY). Water samples for legionella were cultured on buffered charcoal yeast extract and selective buffered charcoal yeast extract agars.

Amoebae. Water samples from the lap pool, wading pool, and hot tub were filtered through 3 layers of sterile cheesecloth and a sterile 5.0- μm cellulose acetate membrane (47 mm diameter), inoculated on nonnutrient agar plates coated with enteric bacteria, incubated at 43°C, and observed microscopically for amoebal growth.

Endotoxin. Air samples for endotoxin analysis were drawn through 37-mm polycarbonate 0.4- μm capillary pore membrane filters at 2 to 4 L per minute by calibrated SKC universal flow sampling pumps for 300 minutes. Simultaneous duplicate or triplicate samples were collected at each sampling point, with most samples being taken from the return air plenum. Air samples from 2 other indoor pools in the Denver metropolitan area and outdoor air samples were obtained as controls. Duplicate water samples were collected in endotoxin-free 25-mL

glass vials held under streams from water spray features or submerged in the pools. Tap water supplied to the pool and water from 5 area pools served as controls. Samples were sent via overnight courier to the Harvard School of Public Health, where they were analyzed by the kinetic Limulus assay with resistant-parallel-line estimation.⁴ Limulus amoebocyte lysate and control standard endotoxins (NP1 or *Escherichia coli* 0113) were obtained from Associates of Cape Cod (Woods Hole, Mass). All results are reported in endotoxin units (EU) of reference standard endotoxin EC5 (United States Pharmacopeia, Rockville, Md).

Statistical Analysis

Endotoxin results were computed as weighted mean log potency and standard error from simultaneous duplicate or triplicate samples.⁴ These results are presented on the arithmetic scale with 95% confidence intervals. All other results are expressed as mean values with ranges. The difference between 2 means for continuous variables was determined by Student's *t* test (2-tailed). We chose a probability of .05 or less as the level of statistical significance.

Results

Epidemiology

Of 138 questionnaires distributed following the initial outbreak, 129 (93%) were returned for analysis (62 by pool employees and 67 by nonpool employees). Of 56 individuals with 2 or more work-related symptoms, 40 (71%) underwent a complete medical evaluation. These 40 individuals included 26 current pool employees, 6 for-

mer pool employees, and 8 nonpool recreation center employees. Eighteen individuals, all lifeguards, had either definite ($n=9$) or probable ($n=9$) cases, for an attack rate of 27% of pool employees (5 of whom did not complete the questionnaire). Two of the lifeguards had been away from the pool for 6 months and 2 years at the time of diagnosis. No cases were identified in the nonpool employee population.

Twenty-three lifeguards were hired when the pool reopened in May 1990. Of these individuals, 16 were newly hired and 7 had previously worked at the pool. None reported symptoms on a baseline questionnaire at the time the pool reopened. Three months later, 18 of these 23 lifeguards reported pool-related respiratory and systemic symptoms on a follow-up questionnaire. We medically evaluated the 18 symptomatic lifeguards, 15 of whom were diagnosed as having definite or probable cases (an attack rate of 65%). Five of the 15 new case patients had worked in the pool before the initial closing. Three of these 5 former employees were hired within 2 weeks of the initial closing. Two of these individuals had reported symptoms and undergone clinical evaluation during the initial outbreak investigation, at which time neither had demonstrable lung disease, including normal transbronchial biopsies and bronchoalveolar lavage. The attack rates for rehired and newly hired employees were comparable.

The mean age of the affected lifeguards in the first outbreak was 23.7 years, and 67% were male (Table 1). The single current smoker smoked 6 cigarettes a week. The mean latency from time of hire to onset of symptoms was 8.4 months. Thirteen of the 18 affected lifeguards noted an onset of symptoms during the winter and early spring months that they attributed to increased pool

occupancy and decreased fresh air intake. Case patients worked more cumulative hours (mean cumulative exposure: 2253 hours vs 907 hours; $P=.01$) and more hours per week than nonpatients (31 vs 23; $P=.09$).

Nonpatients and patients from the second outbreak were demographically similar (Table 1). Mean latency (which included the duration of brief previous employment for the 5 rehired employees) was 9 weeks (range: 2 to 4 months) for 12 of the 15 patients who recalled the time of onset of their symptoms. As in the first outbreak, mean cumulative exposures were highest for those with definite cases, intermediate for those with probable cases, and lowest for nonpatients.

The majority of affected lifeguards reported work-related cough, dyspnea on exertion, chest tightness, and upper respiratory congestion (Table 2). Among various job duties, guarding on the 10-foot-high "crow's nest" when doors and windows were closed was most frequently associated with work-related symptoms. Systemic symptoms were less common than respiratory symptoms and were never reported in isolation.

Industrial Hygiene

Ventilation system analysis. No visible microbial contamination existed in the ventilation system. Smoke testing following the first outbreak indicated that the design and proximity of the roof exhaust fans and air intake system, along with an enclosed area around the air handler, resulted in approximately 20% reentrainment of exhaust air into the air intake system. Tracer gas analysis revealed deficiencies in ventilation system control, further limiting outdoor air intake. These circumstances resulted in a maximum outdoor air supply of only 60% of the design capacity of 30 000 cu ft per minute. We also

TABLE 1—Demographics, Cumulative Exposure, and Symptom Latency in Pool-Related Disease Investigations: Colorado, 1989/90

Characteristic	First Outbreak			Second Outbreak		
	Definite Cases (n = 9)	Probable Cases (n = 9)	Noncases (n = 25)	Definite Cases (n = 6)	Probable Cases (n = 9)	Noncases (n = 6)
Mean age, y (range)	22.0 (18–25)	26.0 (19–59)	24.7 (16–57)	19.5 (17–30)	21.1 (17–35)	19.3 (16–23)
Sex						
Male	6	6	9	2	5	4
Female	3	3	16	4	4	2
Smoking	1 CS	1 FS	1 FS	All NS	All NS	All NS
Mean cumulative hours in pool (range)	2500** (255–5880)	2005* (619–3590)	907 (60–3243)	674 (550–1480)	537 (96–1598)	526 (315–680)
Mean symptom latency, mo (range)	10.4 (1–26)	6.25 (1–23)	...	2.5 (2–3)	1.9 (0.75–3)	...

Note. CS = current smoker; FS = former smoker; NS = never smoker.

* $P=.01$ (in comparison with noncases).

** $P=.003$ (in comparison with noncases).

TABLE 2—Work-Related Symptom Prevalences, by Group, From First and Second Outbreaks: Colorado, 1989/90

Symptom	First Outbreak			Baseline (May 1990)		Second Outbreak	
	Lifeguard Patients (n = 18)	Pool Nonpatients ^a (n = 44)	Nonpool Employees (n = 67)	Pool Employees (n = 23)	Lifeguard Patients (n = 15)	Lifeguard Nonpatients (n = 3)	Nonpool Employees (n = 12)
Cough ^b	61	46	2
Shortness of breath ^c	56	36	15	0	13	0	17
Chest tightness ^d	83	68	22	17	53	0	8
Wheezing ^e	67	52	9	17	47	17	17
Fever ^f	39	21	6	0	13	0	0
Myalgias ^g	39	9	9	0	0	0	0

^aAll questionnaire responders, including those who did not undergo clinical evaluation.

^bCough was not included in the baseline or follow-up questionnaire surveys of the second lifeguard group.

^cSignifies a positive response to the question "Are you troubled by shortness of breath when hurrying on the level or walking up a slight hill?"

^dSignifies a positive response to the question "Does your chest frequently feel tight?"

^eSignifies a positive response to the question "Does your chest ever sound wheezy or whistling occasionally apart from colds?"

^fSignifies a positive response to the question "Do you frequently feel feverish during the day or night?"

^gSignifies a positive response to the question "Do you frequently ache all over during the day or night?"

found poor distribution of inside supply air, with the crow's nest having minimal air movement.

Following the first pool closure, an additional air handling unit was installed, and the amount of fresh air supplied to the pool was increased to 45 000 cu ft per minute. The air intake of the old air handler was relocated to prevent reentrainment of exhaust air. Air distribution throughout the pool was improved with a new duct system.

Aerosol particle analysis. Activation of waterfalls and the mushroom fountain generated a statistically significant 1.4-fold increase in respirable aerosol particles (0.45–7.5 µm diameters). Addition of a water slide resulted in a 2.3-fold increase above background. The number of respirable particles rose 5.2-fold with full water feature use.

Microbial analysis. *Pseudomonas aeruginosa*, *Stenotrophomonas maltophilia*, and other *Pseudomonas* species were detected at the highest levels (greater than 500 000 colony forming units per 100 mL) in first-catch samples from several water features (including the bridge spouts and wall spouts) after several days of disuse. *Acinetobacter calcoaceticus* and various unidentified gram-negative bacilli were also recovered in these

samples. Samples obtained following first-catch samples showed much lower concentrations of viable bacteria (5–31 colony-forming units per 100 mL). Total bacterial counts in water from the spray features were highest when R2A agar was incubated at room temperature for a minimum of 96 hours. Lap pool water and air samples for bacteria were repeatedly low (less than 500 colony forming units per 100 mL), with *Pseudomonas* species being the predominant isolates from air samples. Samples cultured for fecal coliforms, fecal streptococci, thermophilic bacteria, and *Legionella* were negative. We found no evidence of indoor amplification of viable or nonviable fungi before or after either pool closing. A small, encysted hartmanellid amoeba was isolated from pool water following the first outbreak.

Endotoxin analysis. Air endotoxin levels in the pool prior to its initial closing were 27 to 162 times higher than air endotoxin levels at 2 control pools without water spray features and 25 times higher than levels in outside air (Table 3). Lap pool water endotoxin levels were significantly higher (mean = 240 EU/mL, 95% confidence interval [CI] = 120, 420) than samples from 3 control indoor swimming pools (range:

73–86 EU/mL; highest upper confidence limit: 94 EU/mL) and tap water endotoxin levels obtained from the recreation center (3.9 EU/mL, 95% CI = 3.0, 4.9).

Air endotoxin levels had increased to a mean of 450 EU/m³ 2 weeks after the pool closed in October 1989 (with all water features on) (Figure 1). Lap pool water endotoxin levels had tripled (800 EU/mL). Ten weeks after the pool closed (December 1989), we measured water and air endotoxin levels after the water features had been fully on for 24 hours and with outdoor air intake reduced to the minimum. Both air and lap pool water endotoxin levels were greatly reduced in comparison with previous sampling results (4.1 EU/m³ and 0.85 EU/mL, respectively). The only persistently elevated endotoxin level was measured in a first-catch water sample obtained from the bridge spout (570 EU/mL), where the highest levels of *Pseudomonas* species were also detected. Both air and lap pool water endotoxin levels were low shortly after the pool reopened in May 1990 (5.7 EU/m³ and 9.4 EU/mL) but increased to 28 EU/m³ and 150 EU/mL, respectively, with recognition of the second outbreak 3 months later.

Shortly after the pool closed the second time, mean air endotoxin measurements

TABLE 3—Endotoxin Levels in Air and Water in Index and Control Pools: Colorado, 1989/90

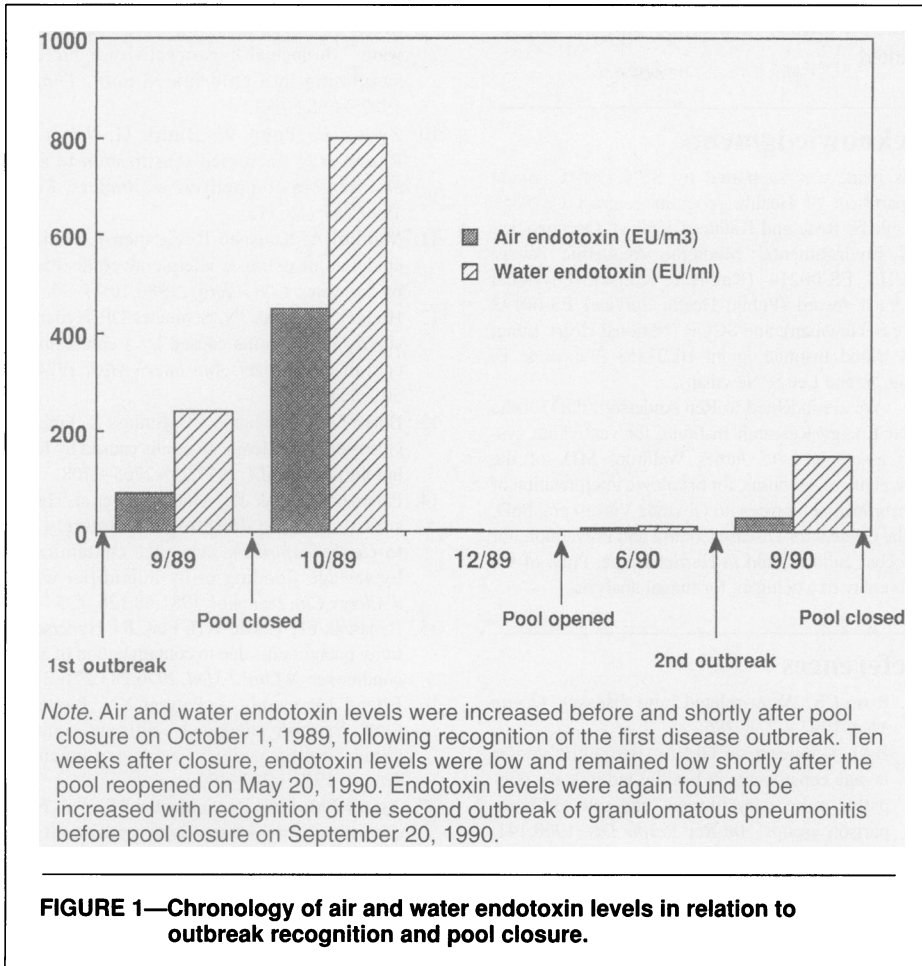
Medium	Index Pool ^a		Control Pools				
	First Outbreak	Second Outbreak	Pool 1	Pool 2	Pool 3	Outside Air	Tap Water
Air, ^b EU/m ³ , mean (95% CI)	76 (53, 110)	28 (12, 69)	2.8 (2.1, 3.7)	0.47 (0.37, 0.60)	...	3.0 (1.6, 5.8)	...
Water, ^c EU/mL, mean (95% CI)	240 (120, 240)	150 (120, 190)	73 (57, 91)	84 (64, 110)	86 (79, 94)	...	3.9 (3.0, 4.9)

Note. CI = confidence interval.

^aLevels obtained shortly before pool closure and with minimal use of water features.

^bMeasured at crow's nest.

^cMeasured at lap pool.



were obtained in conjunction with Las-X particle analysis, with the water spray features turned off (7.8 EU/m³) and then fully operational (59 EU/m³). Full use of water features resulted in a 3.5- to 8-fold increase in mean air endotoxin levels, depending on the location of sampling.

Discussion

Indoor swimming pools have not previously been identified as sources of endemic granulomatous lung disease. Respiratory irritation and bronchial reactivity have been attributed to exposure to volatile organic compounds present in pools,⁵⁻¹⁰ and both hypersensitivity pneumonitis and febrile respiratory illnesses have occurred in association with microbially contaminated tap water,¹¹ humidifiers and cool mist vaporizers,¹²⁻¹⁴ spray humidification and cooling systems,¹⁵⁻¹⁷ saunas,¹⁸ and hot tubs.¹⁹ In the outbreaks described here, the swimming pool was clearly associated with disease found in lifeguards. Only employees who worked in the pool area were found to have granulomatous pneumonitis. The 33 affected lifeguards reported respiratory and systemic symptoms only in relation to working at the pool. This

pool-related symptom pattern was particularly apparent with the second outbreak, since lifeguards were known to have been asymptomatic at the time of preemployment evaluation. We demonstrated an exposure response effect in the first outbreak, with significantly longer mean cumulative pool exposures for case patients than for nonpatients.

Several lines of evidence implicate a pathogenic bioaerosol amplified in and disseminated from pool water features as the cause of the outbreaks. First, the water spray features generated a respirable aerosol proportional in quantity to the number of features in use. Second, samples of water collected from water spray features after periods of disuse (as occurred each night after the pool closed) contained increased numbers of gram-negative bacteria, predominant *Pseudomonas* species. The water spray feature design, which included a separate circuit and pump for each group of water sprays, appeared to promote bacterial growth within the circuits. These circuits were subsequently found to be severely corroded, with some having substantial occlusion of the interior diameter by uncharacterized deposits. Pool chlorination may not have been effective within these circuits during periods of disuse, allowing amplification of bacteria that were then aerosolized in res-

pirable droplets when the sprays were activated. Third, endotoxin levels in pool air and water were elevated during both outbreaks in this pool (relative to control pools) and increased with water feature use. Gram-negative bacteria and bacterial outer membrane fragments contain endotoxin. The presence of endotoxin without demonstration of high levels of viable bacteria in our pool air samples suggests that the microbial contaminants were not present in high enough concentrations to allow detection, did not survive aerosolization, or were not detected owing to the limitations of available culture conditions. Our observation of an association between air and water endotoxin levels and pool-related lung disease suggests that endotoxin may be a marker, an etiologic agent, or an adjuvant enhancing the processes leading to lymphocytic alveolitis and granulomatous pneumonitis.^{20,21}

We did not find one isolated microorganism to which we can attribute these 2 outbreaks of pool-related granulomatous disease, despite using state-of-the-art bioaerosol measurement methods and laboratories for every microbial genera that had been associated with similar respiratory disease. Serum precipitin testing against an extract of material collected on a fiberglass filter for a 24-hour high-volume pool air sample showed precipitins in both pool cases of hypersensitivity pneumonitis and unexposed controls, and no precipitins were present in serum of case patients against a standard commercial battery of hypersensitivity pneumonitis antigens (data not shown).

The disease found in lifeguards in these sequential outbreaks shares histopathologic features with hypersensitivity pneumonitis. Although the high attack rate is unusual, previous reports of hypersensitivity pneumonitis outbreaks in industrial and office buildings have described widely varying attack rates ranging from 1% to 70% of exposed populations.²² The attack rate of 27% in the first pool outbreak undoubtedly underestimates the true rate, since many symptomatic former lifeguards had left pool employment before the outbreak was recognized, and 14 symptomatic lifeguards were not evaluated. The higher attack rate of 65% in the second outbreak, which occurred despite the ventilation system improvements engineered before reopening and the short duration of exposure for most patients, is probably more accurate, because lifeguards were evaluated rapidly after onset of symptoms.

Little information exists concerning the latency between exposure and symptom onset for immunologic lung diseases such as hypersensitivity pneumonitis. The sequential outbreaks we have described here provided a unique opportunity to evaluate symptom

latency. Following the first outbreak, some lifeguards described pool-related symptoms within weeks after beginning employment, while others worked for 2 years at the pool before noticing symptoms. The second outbreak provided a more reliable estimate of symptom latency, because pool exposure began at approximately the same time for all lifeguards and medical evaluations were performed shortly after symptoms were reported. The high attack rate and short duration of exposure preceding symptom onset in the second outbreak suggest extremely effective dissemination of bioaerosols to which a large portion of the exposed employee population could react.

The initial finding of ventilation system deficiencies seemed to support the lifeguards' reports of poor air quality in the pool, especially in the winter months, when outdoor air intake was minimized to conserve heat. However, correction of ventilation deficiencies following the first outbreak did not prevent recurrence of illness after the pool reopened. The second outbreak suggests that source control, rather than dilution ventilation, is necessary to prevent disease. The pool was subsequently reopened without apparent disease recurrence after water feature circuits had been replaced and supplied with water directly from a corona discharge ozone disinfection system and after provision of free residual chlorine in pool water from chlorine gas.

These recurring outbreaks are probably not unique to the indoor swimming pool described here. With the growth in the leisure pool industry worldwide and the use of water spray features in indoor pools, new cases of pool-associated granulomatous pneumonitis are likely to occur. Recognition of other outbreaks of granulomatous pneumonitis will allow further investigations of the microbial ecology and bioaerosol dissemination in indoor swimming pools so that successful primary prevention, surveillance,

and abatement strategies can be implemented. □

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