ANNALS of Internal Medicine

MARCH 1984 • VOLUME 100 • NUMBER 3

Published Monthly by the American College of Physicians

A New *Legionella* Species, *Legionella feeleii* Species Nova, Causes Pontiac Fever in an Automobile Plant

LOREEN A. HERWALDT, M.D.; GEORGE W. GORMAN, B.S.; TERESA McGRATH, M.D.; SANDU TOMA, M.D.; BONNIE BRAKE, M.S.; ALLEN W. HIGHTOWER, M.S.; J. JONES, M.D.; ARTHUR L. REINGOLD, M.D.; PETER A. BOXER, M.D.; PATRICK W. TANG, M.Sc.; C. WAYNE MOSS, Ph.D.; HAZEL WILKINSON, Ph.D.; DON J. BRENNER, Ph.D.; ARNOLD G. STEIGERWALT, B.S.; and CLAIRE V. BROOME, M.D.; Atlanta, Georgia; Toronto and Windsor, Ontario, Canada; and Cincinnati, Ohio

From 15 to 21 August 1981, Pontiac fever affected 317 automobile assembly plant workers. Results of serologic tests were negative for *Mycoplasma, Chlamydia,* respiratory tract viruses, and previously described legionellae. A gram-negative, rod-shaped organism (WO-44C) that did not grow on blood agar, required L-cysteine for growth, and contained large amounts of branched-chain fatty acids was isolated from a water-based coolant. The organism did not react with antisera against other legionellae, and on DNA hybridization the organism was less than 10% related to other *Legionella* species. Geometric mean titers found by indirect fluorescent antibody testing to WO-44C were significantly higher in ill employees than in controls ($p = 0.0001$). Attack rates by department decreased linearly with the department's distance from the implicated coolant system. The etiologic agent apparently was a new *Legionella* species; we propose the name *Legionella feeleii* species nova (AATC 35072). This is the first outbreak of nonpneumonic legionellosis in which the etiologic agent is not *L. pneumophila,* serogroup 1.

PONTIAC FEVER is a severe influenza-like illness characterized by fever, headache, myalgia, and malaise. Unlike Legionnaires' disease (pneumonic legionellosis), which has caused numerous outbreaks and sporadic cases (1-6), Pontiac fever has been recognized only retrospectively in two outbreaks (7, 8). The first outbreak occurred in the Oakland County, Michigan, Health Department in August 1968 and was caused by the airborne spreading of *Legionella pneumophila,* serogroup 1, from a contaminated air-conditioning system. The organism was retrospectively isolated in 1977 from frozen samples of condenser water and from lung tissue of guinea pigs exposed to aerosols of evaporative condenser water (9). In addition, seroconversion to *L. pneumophila,* serogroup 1, was shown in ill persons but not in controls. The second outbreak occurred in 1973 in ten men who used compressed air to clean a steam turbine (8). The etiologic agent was not isolated, but five of the men showed seroconversion to *L. pneumophila,* serogroup 1. In both outbreaks, the mean incubation period was approximately 36 hours, and the attack rates were 95% and 100%, respectively. The illness was self-limited and there were no fatalities. We report here the clinical, epidemiologic, microbiologic, and serologic characteristics of an outbreak of Pontiac fever caused by a previously undescribed *Legionella* species (isolate WO-44C).

▶ From the Division of Bacterial Diseases, Center for Infectious Diseases, Centers for Disease Control, Atlanta, Georgia; Ministry of Labor and Ministry of Health, Toronto, Ontario, Canada; Windsor-Essex County Health Unit, Windsor, Ontario, Canada; and the Hazard Evaluation and Technical Assistance Branch, National Institute for Occupational Safety and Health, Cincinnati, Ohio.

Background

Corporation A operates a large factory in Windsor, Ontario, Canada, that includes two engine assembly plants (plants 1 and 2) that produce V-8 engines from precast iron or aluminum parts. The parts are ground and machined to the proper sizes on the production lines and are assembled into complete engines on the assembly line (Figure 1 Top). The production departments and the assembly line are serviced by several systems that produce aerosols or exhaust humidified air into the plant or on the roof—compressed air lines, parts washers, "wet" air cleaners, and coolant systems (Figure 1 Top). The coolant systems lubricate, cool, and clean the grinding and machining surfaces. The individual coolant systems do not interconnect, and they range in capacity from 1000 to 30 000 gallons. The coolant used is 88% to 99% water and 1% to 12% oil. Caustics are added as needed to keep the coolant's pH between 8.5 and 9.5 (except in systems servicing aluminum machining, for which the pH should not exceed 9), and biocides are added when the bacterial count exceeds 10^5 to 10^6 organisms per millilitre. The coolant, which also contains metal shavings, dirt, and other debris, is mechanically circulated through underground troughs from a main tank, to the machines, and back to the main tank. As the coolant is applied to the grinding or machining surface, large drops of coolant splash on and around the machine and a very fine aerosol—oil mist—becomes suspended in the air.

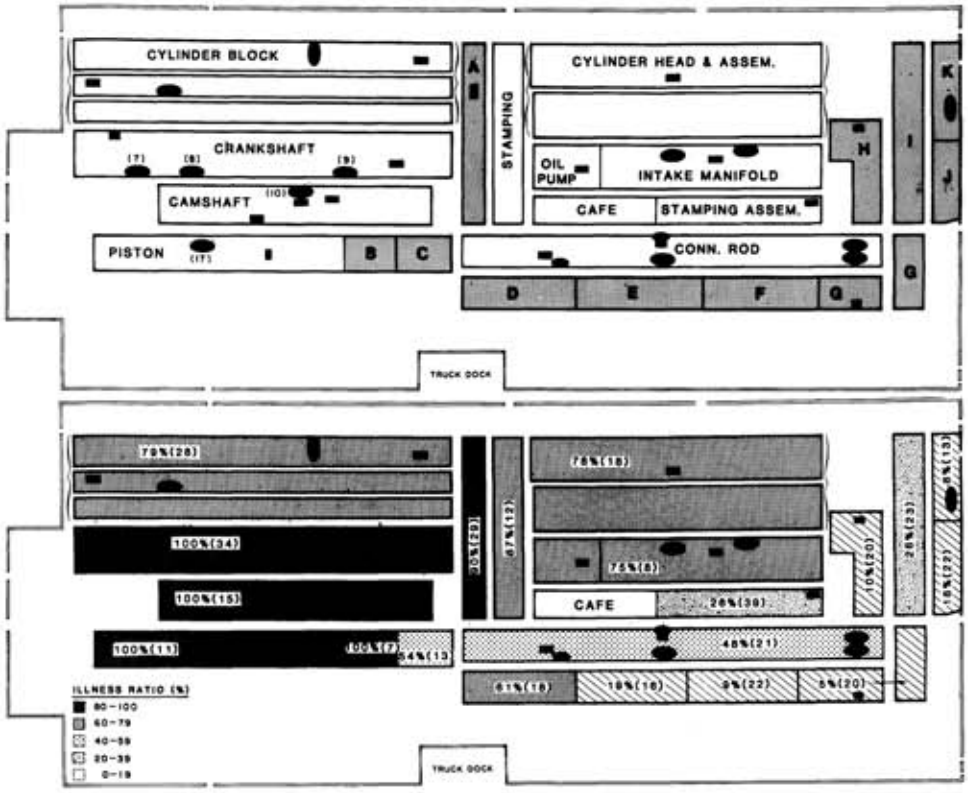

Figure 1 Top. Layout of plant 2 by department. The boxes (■) are washers and the ovals (⬬) are coolant systems. The parentheses beside the coolant systems give the unit's number. The shaded areas are the engine assembly line. **Bottom.** Attack rates of Pontiac fever by department. The percentages are the amount of ill workers in the total population of the department, excluding possibly ill workers. The total number of workers in the department who responded to the questionnaire is given in the parentheses.

Because of an oversupply of engines, plant 2 was shut down between 8 to 16 August (Figure 2), except for the crankshaft and stamping lines. At 0800 h on 17 August, plant 2 resumed production. By 0800 h on 19 August, most of the men on the crankshaft, camshaft, and piston lines had become ill and were complaining of headaches, severe body aches, high fever, and extreme fatigue. The three lines were shut down, and an investigation was begun. By 20 August, the investigation found that men who worked on other production lines and men who had worked on the engine assembly line in plant 2 on 17 and 18 August and who had shifted to plant 1 on 19 August were also becoming ill; however, men who had worked only in plant 1 were not ill.

As was the weekly routine, the coolant systems were tested for total bacteria count and pH by plant personnel on 19 August. Biocides Grotan BK and Grotan HDII (Gray Products, Division of Sterling Drugs Ltd., Toronto, Ontario, Canada) were added to all coolant systems on 21 August. Production on the crankshaft, camshaft, and piston lines resumed on 25 August with no recurrence of illness.

Methods

SURVEYS

Several questionnaire surveys were done as part of the investigation. Workers in plant 2 were questioned about their underlying medical problems, use of tobacco and alcohol, department and job descriptions, contact with aerosol sources (compressed air, parts washers, and coolant systems), symptoms they had, and areas in plant 2 other than their work stations that they had most likely visited on 17 to 19 August. Workers were also asked about the severity and duration of their illness and whether family members had become ill. A supplementary questionnaire was given to employees who worked in engine line sections G to K (Figure 1).

LABORATORY STUDIES

Environmental samples were taken on 19 August, 15 September, and 28 October from systems that could produce aerosols or exhaust humidified air in the plant or on the roof. Initially, environmental samples were given intraperitoneally to guinea pigs. However, because chemical components of the fluids were toxic to the guinea pigs, all environmental specimens were subsequently processed by acid decontamination and direct plating on charcoal-yeast extract agar (10), buffered charcoal-yeast extract agar (11), and on these agars supplemented with cephalothin (4 μg/mL), colistin (16 μg/mL), and vancomycin (0.5 μg/mL) (12).

Isolates needing L-cysteine for growth were tested with direct fluorescent antibody against the nine previously described *Legionella* species and several other *Legionella*-like organisms. Organisms that did not stain with any of these sera were further evaluated by biochemical testing, DNA hybridization, and determination of guanine-cytosine content (13, 14). The fatty acid composition was determined by gas liquid chromatography (15, 16).

Antimicrobial susceptibility testing was done with an agar dilution method (17) on a medium of yeast extract, oxoid agar, cysteine, and charcoal at pH 7.1. Buffered charcoal-yeast extract agar was not used because ferric pyrophosphate antagonizes tetracyclines and the pH of 6.9 inhibits erythromycin activity.

A survey of sera from employees of plant 2 was begun on 1 September. Three sets of serum samples were taken at approximately 2-week intervals. Sera from 330 employees were screened by the indirect fluorescent antibody test using 3 antigen pools that together included ten *Legionella* antigens: *L. pneumophila,* serogroups 1 to 6; *L. bozemanii; L. dumoffii; L. micdadei;* and *L. gormanii* (18). Sera from 25 ill persons and from 11 well persons were tested by complement fixation against influenzae A and B; parainfluenzae 1, 2, and 3; adenovirus; *Chlamydia;* and *Mycoplasma pneumoniae.* Sera from 20 ill persons and 11 well persons were also tested by complement fixation against respiratory syncytial virus and herpes simplex virus (19).

One serum sample was also taken from 104 controls from two groups of employees who had no exposure to plant 2. One group of 64 persons were exposed to coolant systems, but the other 40 persons were not.

All sera were tested by indirect fluorescent antibody for titers against WO-44C using heat-killed WO-44C as the antigen. Only one serum specimen was taken from all controls and from 72 of the 208 ill persons who had serum specimens drawn, whereas several serum specimens were taken from the remaining 136 ill persons. Therefore, the titer to WO-44C from the first serum sample drawn on or after 7 September (21 days after the peak of the outbreak) was arbitrarily used to calculate the geometric mean titers and the titer distributions. Because the geometric mean titers and the titer distributions for isolate WO-44C from the two control groups were not statistically different, the two groups were combined into one control group for the following analyses.

ADDITIONAL STUDIES

Smoke candle studies were done to determine the extent of the spread of production-generated aerosols from various points in the plant. Meterologic data for Windsor, Ontario, from 13 to 23 August 1981 were obtained from Environment Canada, Atmospheric Environment Service, Downsville, Ontario.

ANALYSIS

An ill person was defined as a worker who had at least three of the following four symptoms during August 1981: fever, chills, headache, or myalgia. A person was considered well if he claimed not to have been ill in August. Persons who were ill but did not meet the definition for an ill person were considered to be possibly ill.

The initial analysis of the questionnaire and serologic data was done with routine statistical tests. Workers who were possibly ill or who could not be assigned to a specific production or assembly line were excluded from the calculations for department-specific attack rates and from risk-factor analysis. Multivariate risk-factor analysis was done by chi-squared automatic interaction detection. This program first identifies by univariate analysis the factor most closely associated with illness and then divides the study population into risk groups on the basis of this factor. Within each risk group, the program identifies additional risk factors (20).

Results

SURVEY RESULTS

Six hundred ninety-five (80%) employees from plant 2 completed the questionnaire. Of these, 317 (46%) met the definition for an ill person; 270 (39%) were well; and 108 (16%) were ill but did not meet the definition for an ill person.

The illness had a mean maximum incubation period of 46 hours and was characterized by severe myalgia (in 93% of ill persons), chills (in 92%), fever with temperature ranging from 37.5 to 40° C (in 91%), and extreme fatigue (in 89%). Other symptoms included dizziness (in 68% of ill persons), nausea (in 51%), chest pain (in 36%), cough (in 32%), abdominal pain (in 30%), shortness of breath (in 28%), coryza (in 20%), vomiting (in 15%), and diarrhea (in 11%). The illness was short (median duration, 3 days) but severe enough to cause nearly 30% of the workers to miss work (median days of sick leave, 2). There were no fatalities, and only four workers reported similar illnesses in family members within 72 hours after the onset of the worker's illness.

A survey of chest roentgenograms from 237 ill workers, done on 3 to 4 September 1981, showed only 1 man to have bibasilar infiltrates, which may have been acute. Thirty-six workers submitted physicians' reports, but the physical examination and laboratory data were unremarkable.

Attack rates varied significantly by department (Figure 1 Bottom). In general, the attack rates by department decreased progressively from north to south. This trend was particularly striking in the stamping and engine assembly departments, which are divided into working sections (Figure 1). The departments with the highest attack rates were along a line extending from the piston, camshaft, and crankshaft departments in the northwest, to the cylinder head and assembly departments in the southeast.

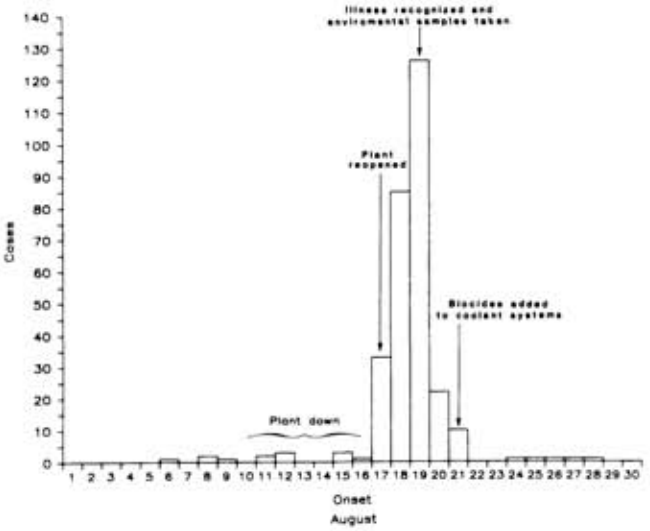

Figure 2. Time course of the Pontiac fever outbreak in plant 2.

The attack rate gradient suggested an association between the location in which an employee worked on 17 and 18 August and his risk of developing illness. The association was confirmed by findings of the risk-factor analysis using the chi-squared automatic interaction detection ($p = 6.4 \times 10^{-28}$). Several other factors, including visiting the piston ($p = 0.005$), crankshaft ($p = 0.032$), and cylinder block ($p = 0.030$) departments, were also closely associated with illness according to univariate analysis. However, when the individual risk areas (Figure 1 Bottom) were analyzed by multivariate analysis, risk factors other than where one worked were not identified for employees in the four highest risk areas. Only in the lowest risk area did factors other than work station seem to be associated with the development of illness. In this latter area, persons were at a higher risk of illness if they had visited the connecting rod department, which is located in a higher risk area ($p = 0.012$). Of those who did not visit the connecting rod department, persons who consumed more than ten alcoholic beverages in a week were at a higher risk of illness than those who drank less ($p = 0.026$). In addition, analysis of the data from the supplementary questionnaire given to engine line employees showed that ill persons working in the low-risk sections of the engine line had a slightly higher risk of developing illness if they visited engine line sections A, C, or D (Figure 1) ($p = 0.039$, 0.034, and 0.0023, respectively; 2-tailed Fisher's exact test). However, 24 workers became ill who worked in the lowest risk area and who did not visit any of the higher risk depart-

Table 1. Phenotypic Characteristics of WO-44C and Other *Legionella* Species

Characteristics	WO-44C	*L. pneumophila* (21)*	*L. bozemanii* (22)	*L. dumoffii* (22)	*L. gormanii* (23)	*L. jordanis* (13)	*L. longbeachae* (24)	*L. micdadei* (25)	*L. oakridgensis* (26)	*L. wadsworthii* (27)
Motility	+	+	+	+	+	+	+	+	−	+
Autofluorescence	None	Dull yellow	Blue-white	Blue-white	Blue-white	None	Dull yellow	Dull yellow	Dull yellow	Dull yellow
Oxidase	−	+	−	−	−	+	+	+	−	−
Catalase	+	+	+	+	+	+	+	+/−	+	+
Gelatinase	−	+	+	+	+	+	+	+	+	+
Beta-lactamase	−	+	+	+	+	+	+/−	−	+ (weak)	+
Hippurate hydrolysis	+ (weak)	+	−	−	−	−	−	−	−	−

* The numbers in parentheses are reference numbers.

ments.

Because 99.7% of the respondents were men and 97% were white, consistent with the sexual and racial makeup of the plant, sex and race could not be evaluated as risk factors. Other factors not associated with illness were age; shift worked; underlying medical illness; regular use of medication; cigar and cigarette smoking; water consumption; showering at work; and working with or near compressed air, parts washers, and coolant systems.

CULTURE RESULTS

A *Legionella*-like organism, designated WO-44C, was isolated from a sample of coolant obtained 19 August from system 17 in the piston department (Figure 1 Top). No other legionellae or *Legionella*-like organisms were isolated from any of the other environmental samples. Unfortunately, coolant samples obtained on 19 August were available only from systems 7, 10, and 17.

Phenotypic characteristics of WO-44C are similar to those for previously reported legionellae (Table 1) in that the organism requires L-cysteine for growth, does not grow on blood agar, does not reduce nitrite to nitrate, or produce acid from carbohydrates. However, WO-44C is the only *Legionella* species that does not produce gelatinase and only the second to hydrolyze hippurate. The organism grows well at 25° C and 35° C, but not at 42° C. Like other legionellae, WO-44C is sensitive in vitro to rifampin (0.06 μg/mL) and erythromycin (0.05 μg/mL); in-vivo studies have not been done. The clinical relevance of these sensitivity measurements is unknown, because in-vitro measurements may not correlate well with in-vivo studies in *Legionella*.

WO-44C is antigenically distinct from other legionellae, because it does not stain with direct fluorescent antibody sera against the nine previously described species and other undescribed legionellae or *Legionella*-like organisms. On DNA hybridization, WO-44C is less than 10% related to all named *Legionella* species (Table 2). The guanine-cytosine content is 45.7%.

The cellular fatty acid composition is qualitatively and quantitatively similar to that of other legionellae, having the characteristic features of relatively large amounts of branched-chain acids and the absence of hydroxy acids (15, 16, 28, 29). The four major acids present in 48-hour cultures are a-17 (17%), i-16 (15%), 16:1 (18%), and 16:0 (26%); a-17:0 is present at 7%, and other fatty acids reported for legionellae are present at 1% to 4%. No cyclopropane acids were found at concentrations greater than 1%.

SEROLOGIC RESULTS

Testing sera from ill and well persons against influenzae A and B; parainfluenzae 1, 2, and 3; adenovirus; respiratory syncytial virus; herpes simplex; *Chlamydia;* and *Mycoplasma pneumoniae* was nondiagnostic. Indirect fluorescent antibody testing of sera from ill, well, and possibly ill persons against ten *Legionella* antigens, including *L. pneumophila,* serogroup 1, was also nondiagnostic.

Indirect fluorescent antibody testing of all available sera against WO-44C showed 28 ill persons (21% of ill persons with paired sera) to have had seroconversions to WO-44C ($\geq$ 4-fold rise in indirect fluorescent antibody titer). In addition, 1 possibly ill person and 4 well persons (5% and 33%, respectively, of those with paired sera) had seroconversion to WO-44C.

According to statistical testing, the indirect fluorescent antibody titers to WO-44C for ill persons (geometric mean titer, 296) were significantly different from those for well persons (geometric mean titer, 165; $p = 0.0006$), possibly ill persons (geometric mean titer,

Table 2. Comparison of DNA Relatedness of WO-44C and Other *Legionella* Species

Source of Unlabeled DNA	Source of Labeled DNA (WO-44C), RBR* (60 °C)
WO-44C	100†
L. pneumophila (Philadelphia 1)	2
L. bozemanii (WIGA)	3
L. micdadei (TATLOCK)	6
L. dumoffii (NY-23)	3
L. gormanii (LS-13)	5
L. longbeachae (LB-4)	5
L. wadsworthii (81-716A)	3
L. jordanis (BL-540)	3
L. oakridgensis (OR-10)	4

* RBR = relative binding ratio, which is (% heterologous DNA bound to hydroxyapatite)/(% homologous DNA bound to hydroxyapatite) × 100.

† All reactions were run in duplicate. Reassociation of WO-44C DNA in homologous reactions was between 51% and 71% (average, 59%). However, these values are arbitrarily designated as 100%, and heterologous reactions are normalized to them. Control reactions containing only labeled DNA showed 0% to 1.5% of labeled DNA was bound to hydroxyapatite. This background binding was subtracted from heterologous reaction results before normalization.

128; $p = 0.007$), and controls (geometric mean titer, 71; $p < 0.0001$). The distribution of the indirect fluorescent antibody titers is shown in Figure 3. Geometric mean titers for well persons and possibly ill persons were not statistically different from each other but were significantly higher than the geometric mean titer for controls ($p < 0.001$ and 0.0152, respectively). The well and possibly ill persons had been in plant 2 at the time of the outbreak and may have been exposed to the etiologic agent, whereas the controls had no recent contact with plant 2. Because the illness definition was closely correlated with both location of work and indirect fluorescent antibody titer, the serologic data were stratified by the illness definition to ascertain whether indirect fluorescent antibody titer was a function of the definition or whether it varied independently with location of work and, therefore, dose of bacteria received. Ill persons who had worked in the three highest risk areas (departments closest to system 17) had a higher geometric mean titer (345) than did ill persons who worked in the two lowest risk areas (geometric mean titer, 154) ($p = 0.0339$, Wilcoxon rank sums). Geometric mean titers for well persons who worked in the higher and lower risk areas were not statistically different.

Smoke candle studies showed that regardless of the point of release, the smoke particles spread horizontally, giving a visually detectable level of particles over a large portion of three to four production lines. The particles also left the plant through roof windows and re-entered the plant several production lines away from the source.

Discussion

The outbreak in engine plant 2 was very similar to the two previously reported outbreaks of Pontiac fever. The outbreak was characterized by a self-limited, severe, flulike illness with a short incubation period, a high attack rate among workers nearest the presumed source, no evidence of secondary spread, and no fatalities. The epidemic curve is consistent with an explosive common-source outbreak (Figure 2). Food- and water-borne spread were ruled out by the findings of the epidemiologic survey, which showed no common source of food or water for the ill employees. Airborne spread from one or more of the coolant systems, including system 17, was the most likely mode of transmission in this outbreak. There was probably a continuing exposure for at least 1 full working day, because workers from all three shifts became ill.

The attack rate gradient was also consistent with airborne spread of the etiologic agent. The departments with the highest attack rates were along a line from the northwest to the southeast, the same axis as the wind direction from 0700 h to 1200 h on 17 August. Wind direction is a major determinant of air flow in plant 2, which in the summer uses natural ventilation through windows and large doors, some of which will accommodate trains and trucks.

The results of the smoke candle study show that smoke released from a point source could spread over a significant portion of four production lines at visually detectable concentrations. Further from the source, the smoke

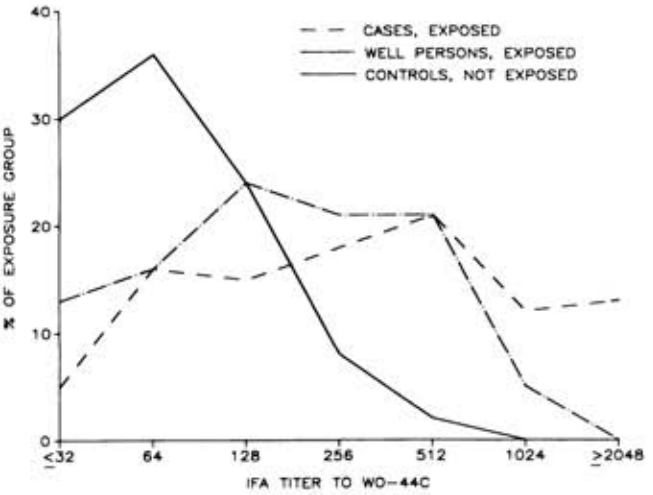

Figure 3. Distribution of peak indirect fluorescent antibody (*IFA*) titers in ill workers (cases), well employees, and controls.

particles are diluted by air, but the particles remain suspended until they strike solid objects, flocculate, are filtered, or become condensate nuclei. Because smoke particles (0.01 to 2 μm diameter) and aerosols generated by machining and grinding operations (99% are 3 μm or less in diameter and 50% are 1 μm or less) are similar in size, their behavior in air currents may also be similar (WABEKE R. Personal communication.), suggesting that coolant aerosols could be spread over a large area in relatively high concentrations.

The results of the epidemiologic survey strongly suggest that the etiologic agent was airborne and spread through the plant from a source in the northwest production lines. Aerosols from system 17, from which WO-44C was isolated, were probably responsible for the outbreak. This system was not being circulated during the shutdown period from 8 to 16 August 1981, because the piston department was not operating. Although the data needed to prove this hypothesis are not available, one can speculate that during the period of disuse, bacterial overgrowth, which is known to occur more readily when coolants are not circulated, decreased the pH of the coolant and led to separation of the oil-water emulsion (TRUDELL C. Personal communication.). This separation could have been favorable for the growth of WO-44C. With the resumption of production on 17 August, the machines serviced by system 17 may have generated a contaminated aerosol that was spread through the plant.

Risk factors other than location of work could not be identified in the four highest risk areas, but workers in the lowest risk area who visited the connecting rod department and who drank more than ten alcoholic drinks per week were at an increased risk of illness. Thus, persons exposed to a large dose of bacteria developed illness regardless of their underlying state of health, whereas persons exposed to fewer bacteria developed clinical illness more readily if they had an additional risk factor, such as a history of heavy alcohol consumption.

The significant difference in geometric mean titers to WO-44C between the ill persons, well persons exposed to

plant 2, and controls; the seroconversions to WO-44C; and the absence of diagnostic titers to other possible etiologic agents indicates that WO-44C was the etiologic agent of the outbreak. Also, the geometric mean titers and the titer distribution... the well persons and the possibly ill persons who wer ...posed to plant 2 were significantly higher than those for the controls who had no exposure to plant 2. Most of these employees from plant 2 worked in lower risk areas, suggesting that a low dose of bacteria may have been sufficient to cause an antibody response but was not adequate to cause clinical illness. An alternative explanation for the elevated titers in well persons is chronic exposure within the plant.

The epidemiologic and serologic findings of this investigation strongly suggest that WO-44C was the etiologic agent of a large outbreak of Pontiac fever. Results of the laboratory studies have shown that the organism has the phenotypic characteristics typical of legionellae, but that it is distinct from all previously described *Legionella* species. We believe that WO-44C should be recognized as a new *Legionella* species. We propose the name *Legionella feeleii* species nova (fé lei i) (*feeleii*, modern Latin genitive noun), because James C. Feeley, Ph.D., has participated in the laboratory investigations of all reported outbreaks of Pontiac fever and has developed artificial media capable of supporting the growth of legionellae.

The type strain of *L. feeleii* is WO-44C (ATCC 35072). A description of *L. feeleii* is found in the text, Tables 1 and 2, and Figure 3. *Legionella feeleii* is one of the few *Legionella* species that can be identified phenotypically; only *L. feeleii* and *L. pneumophila* hydrolyze hippurate, and *L. feeleii* is the only described *Legionella* species that does not produce gelatinase.

ACKNOWLEDGMENTS: The authors thank Mary Ann Schott, Harold McEachern, Barry Fields, Dr. Herta Wulff, Dr. Roger McKinney, Dr. Robert Weaver, and Linda K. McDougal for laboratory support; Dr. Michael Silverstein of the International United Auto Workers and the members of UAW Local 200; Thomas O. MacAdoo of Virginia Polytechnic Institute and State University; Dr. Harold W. Rossmoore of Wayne State University; Drs. G. Martin, R. Andreychuck, and J. Carlson of the Ontario Ministry of Health; Dr. Richard A. Condon; Dr. Donald Krogstad for his critical review; Frances Wilson for graphics; and Burnese Harper and Iris Lansing for secretarial support.

The Field Investigation Team consisted of Frank Crosswell, UAW health and safety representative for plants 1 and 2; William Simmonds, David Rattray, William Sproat, M.D., Frank Viola III, M.D., David Sugano, D.P.H., and Roger L. Wabeke of Corporation A; Shiela Barker, R.N., P.H.N., Martin J. Strong, C.P.H.I., of the Windsor-Essex County Health Unit; Evelyn Wallace, M.D., of the Ontario Ministry of Health; and Nicholas R. Paul, M.Sc., of the Windsor Public Health Laboratory.

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► Requests should be addressed to Loreen A. Herwaldt, M.D.; Division of Bacterial Diseases, Center for Infectious Diseases, Centers for Disease Control; Atlanta, GA 30333.

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