Epidemiology of a Q Fever Outbreak in Los Angeles County, 1966

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Q FEVER, a febrile rickettsial infection of man caused by *Coxiella burnetii*, was first described in Los Angeles County by Young (*1*) in 1947. Subsequently, extensive surveillance demonstrated the infection to be enzootic among dairy herds and endemic among human beings working with dairy cows or their raw products and among persons living near dairies. In 1948–49, among Los Angeles County residents, the prevalence of Q fever complement fixing antibodies was 1.3 percent (*2*).

Since Q fever was made a reportable disease in Los Angeles County in 1955, not more than seven cases have been reported in a year (table 1). Until 1966 the cases were distributed throughout the county, and in any given year not more than one or two cases were clustered in a single geographic area. In 1966, five cases were reported from one geographic area, the San Fernando Valley, and because of the apparent clustering, the County of Los Angeles Health Department initiated an investigation. We discuss the clinical and epidemiologic data uncovered and the host-environment-parasitic relationships that may have contributed to the occurrence of this outbreak.

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Methods and Materials

Description of area. Los Angeles County covers 4,083 square miles and in 1966 had a population of 6,995,379. (These data, as of July 1, 1966, were obtained from the records and statistics division of the County of Los Angeles Health Department. The department has jurisdiction over all areas of the county except Long Beach, Pasadena, and Vernon.)

Climatologic considerations. The San Fernando Valley is a flat, arid region extending 25

Table 1. Reported cases of Q fever, by year, compared with peak month of influenza epidemic, Los Angeles County, Calif.

Year	Q fever cases[1]	Peak month of A_2 influenza epidemic
1955	5	.....
1956	7	.....
1957	7	October
1958	3	.....
1959	2	.....
1960	2	January
1961	3	December
1962	7	.....
1963	1	.....
1964	1	.....
1965	1	.....
1966	6	March
1967	0	.....

[1] Reportable since 1955.

miles east to west and 10 miles north to south. Mountain ranges enclose most of the valley floor. A narrow pass at the southeastern corner of the valley permits the entry of moist marine air during the spring and summer. Dry desert air enters from the north and northeast through mountain passes in the fall, winter, and early spring. The desert air often comes in strong gusts, known locally as the "Santa Ana" winds. Rainfall is heaviest between November and April (*3*).

Precipitation in the valley, shown in the following tabulation, was determined for the first half of each year—the period that encompassed the outbreak:

Year	*Precipitation, inches*
1955	12.17
1956	11.31
1957	11.73
1958	17.73
1959	8.95
1960	6.85
1961	2.63
1962	17.82
1963	8.79
1964	4.85
1965	8.92
1966	2.84
1967	10.63

SOURCE: U.S. Weather Bureau. The average precipitation over 11 years (1955–65) for the 6-month period was 10.15 inches (personal communication from J. Penfield, U.S. Weather Bureau, Van Nuys, Calif., March 1968).

Since Q fever was made a reportable disease, precipitation was exceedingly low in 2 years: 1961 and the year of the outbreak, 1966.

Data from other parts of Los Angeles County showed similar amounts of precipitation for 1955–67. The San Fernando Valley, however, differs in other climatic parameters from the rest of Los Angeles County (*4, 4a*). The valley is drier, and the relative humidity is lower than in the central or urban core of the county and much lower than in the coastal areas, which are regularly exposed to varying degrees of moisture-laden marine air.

The windiest part of the county that is inhabited is the San Fernando Valley. During the first 6 months of 1966, the dry, gusty Santa Ana winds were more prevalent than they had been in recent years (Penfield communication of March 1968). The fastest mean wind was 23.5 miles per hour for the valley and 18.3 miles per hour for the central or urban core.

Diagnostic criteria. The diagnosis of Q fever for a reported case was accepted when a fourfold rise in complement fixation (CF) titer or a single high CF titer (above 1:32) was obtained for serums that had been submitted to the viral and rickettsial disease laboratory, California State Department of Public Health (*5*). Surveillance forms were routinely completed on each reported case by the County of Los Angeles Health Department. The patients in this study were examined by their private physicians, not by the authors.

Results

During the first 6 months of 1966, six cases of Q fever were reported to the County of Los Angeles Health Department.

Clinical aspects. Each of the six patients experienced the abrupt onset of a severe upper respiratory illness associated with headache, retro-orbital pain, productive cough, high fever, and malaise. There was no history of rash. Chest roentgenograms were negative in all six. Respiratory symptoms lasted a minimum of 3 weeks in each instance. The diagnosis usually was made only after serologic test results became known.

Serologic studies. Four patients had a fourfold or greater rise in antibody titer by the CF test. Patients 1 and 2 (table 2) had high antibody titers in specimens obtained during convalescence and were considered presumptive positive cases; no specimens were available during the acute stage of the disease. CF studies of patients 4 and 5 for influenza A and B were negative despite the presence of an A_2 influenza epidemic in the community.

Epidemiologic considerations. Five of the six patients lived in the San Fernando Valley. The sixth, patient 3, lived in the Antelope Valley, where he worked as a ranch hand and was exposed to cattle infected with *C. burnetti.* The Antelope Valley is separated from the San Fernando Valley by the San Gabriel Mountains. Only one of the five patients living in the San Fernando Valley had occupational contact with dairy cattle. Patient 4, an agricultural student, had worked with the only herd in the valley that was subsequently shown to be negative for Q fever CF antibodies (personal communication from R. J. Schroeder and R. W. McIntyre, veterinarians for Los Angeles County, February 1968). None of the other five had contact with cattle, sheep, or goats within 1 month before the onset of illness. All patients denied that they had recently ingested raw milk.

The patients' onsets of symptoms were not clustered in time but were randomly distributed over the first 6 months of 1966. The one significant factor common to all five patients from the valley was living or working near, but not on, a dairy farm (table 2). Three of the five patients lived within 1 mile of two dairy farms. Patient 2 did not live 1

Table 2. Serologically confirmed cases of Q fever in Los Angeles County, 1966

Patient's occupation	Age	Sex	Date of onset	CF titer (acute serum)	CF titer (convalescence serum)	Farm employment	Contact with—			Drank raw milk	Distance of residence or work from dairy farm, miles
							Cattle	Sheep	Goats		
1. Teacher	45	Female	Feb. 1	(1)	[2] 1,024	No	No	No	No	No	[3] 0.9
2. Engraver	46	Male	Feb. 12	(1)	[2] 128	No	No	No	No	No	.5
3. Ranch hand	48	Male	Mar. 7	<8	32	Yes	Yes	Yes	No	No	0
4. Agricultural student	23	Male	Apr. 1	<8	256	Yes	Yes	No	No	No	[3].1
5. Administrator	52	Male	May 24	8	512	No	No	No	No	No	[3].2
6. Animal trainer	46	Male	June 11	<8	128	No	No	No	No	No	1.0

1 Serum not available during the acute stage.
2 Presumptive positive.
3 Lives near two dairy farms.

mile or less from a dairy, but worked in a small building located less than 0.5 mile from an infected dairy.

Discussion and Conclusion

The factor most frequently responsible for transmission of Q fever to man is thought to be the airborne spread of *C. burnetii* (*6*). Milk, urine, feces, and placentas from domestic cattle, sheep, and goats can be infected with the organism, and contamination of the soil with this hardy organism permits the development of an infected aerosol (*7*). Rainfall, relative humidity, and wind direction and velocity are all important variables that promote or deter the aerosolization of soil (*8, 9*). Man usually becomes infected by inhaling the contaminated aerosol. Living within 1 mile of an infected dairy herd greatly increases the risk of acquiring an infection (*2*). Only a small number of organisms are required to initiate the process (*10*).

Other less common modes of spread responsible for human infection are ingestion of raw milk (*11*) and, rarely, person to person contact (*12*). Transmission of *C. burnetti* organisms by ticks, the vectors in most rickettsioses, is not often responsible for Q fever in man. Nevertheless, ticks are important in maintaining the disease in nature (*13*).

In the 1966 outbreak, the mode of spread in all cases probably was airborne. All the patients lived within 1 mile of an infected dairy herd (*14*). Only one of these patients worked with dairy cattle, and that herd was not infected with *C. burnetii* when examined 1 year later (Schroeder-McIntyre communication of February 1968). A search was initiated for any unusual conditions that appeared during the first half of 1966. None of the patients had recently moved to his current residence. None had changed occupations, and no direct contact with infected cattle, goats, or sheep was noted. The ingestion of milk could not be implicated. Since the major mode of spread appeared to be airborne, we looked for the climatic conditions favoring aerosolization of the soil. An outbreak of Q fever related to arid, windy conditions in northern California in 1960 (*9*) is a relatively recent example of this mechanism.

Climatic conditions in Los Angeles between January and June 1966 were found to be different from most comparable 6-month periods. Precipitation was low—2.84 inches, as shown in the text table. Lack of the usual amount of precipitation in the San Fernando Valley, a traditionally dry area, coupled with the presence of more than the usual dry, high-velocity winds, probably increased aerosolization by *C. burnetii* contaminated soil from dairy farms. The dust created probably spread to adjacent residential areas or places of employment and resulted in the study cases. Wind direction was not an important factor in the occurrence of these cases.

Other factors may have contributed to the increased number of cases reported in 1966. During the first half of the year, A_2 influenza was epidemic in the county. Clinically severe cases of respiratory disease were evaluated with a serologic screen for the following respiratory agents: influenza A and B, adenoviruses, Q fever, and *Mycoplasma pneumoniae*. This screening uncovered the study cases of Q fever. In 1960–61, no influenza epidemic occurred. The resultant decrease in the number of serologic screening tests ordered may account for the low reported incidence of Q fever in 1961, despite the presence of favorable condi-

tions for aerosolization by *C. burnetii*: that is, the low level of precipitation. In addition, it should be noted that outbreaks of A_2 influenza in Los Angeles County had not been associated with a geographic cluster of cases except in 1966.

The few cases reported in 1966 in the San Fernando Valley probably represented only a small proportion of the actual infections that occurred, since many infections are either asymptomatic (*15*) or attributed to the prevalent respiratory disease. In addition, clinically apparent infections may have nonrespiratory manifestations and be present with hepatitis, endocarditis, aseptic meningitis, or an infectious mononucleosis-like syndrome (*16, 17*). Q fever is usually recognized only when it has a protracted course that necessitates extensive diagnostic tests. These reasons may account for the usual low reported incidence of Q fever.

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Six cases of Q fever—an unusual number—occurred in one area of Los Angeles County during the first 6 months of 1966. The area of increased incidence was a known endemic and enzootic area for Q fever. A unique ecological setting existed in which dairy farms were situated in the midst of residential areas. Most of the farms contained cows infected with *Coxiella burnetti.*

Direct contact with the infected animals or their fresh products was not the only mode of spread. Infection by aerosol occurred in persons whose homes were within 1 mile of the dairies. Various climatologic factors affected the spread of rickettsia by this route. A marked decrease in precipitation and an increase in dry, gusty winds were thought to have been major factors responsible for the increased incidence of reported cases. An epidemic of A_2 influenza occurred during early 1966. The serologic screening for respiratory infectious agents done on persons with severe cases of acute respiratory infections in this period uncovered most of the Q fever cases.