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A SURVEY TO DETERMINE THE INCIDENCE OF ANTIBODIES
AGAINST Q FEVER IN PERSONS IN UTAH

by
Carl H. Blank

A thesis submitted in partial fulfillment
of the requirements for the degree

of

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in

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INTRODUCTION

Q fever, as a recognized clinical entity, is often referred to as a new disease of man. Pneumonic in character but lacking clean-cut pathognomic characteristics, in all probability Q fever has been confused in the past with influenza, primary atypical pneumonia (1), or included as an undifferentiated disease under "virus pneumonia." Geddis Smith (2), writing in 1941, stated:

...influenza, pneumonia, "pleurisy", or bronchitis ... form a nosological jungle in which bacteria and viruses roam at will, despoiling the human race and defying both classification and control. Symptoms overlap and no one knows how many different diseases lurk behind them.

We can speculate that, before recognition of pneumococcic pneumonia, the "nosological jungle" of respiratory diseases must have spelled frustration for any epidemiologist who hacked his way into it attempting to establish order. The bacteriologist did make it possible to categorize the majority of the pneumonias as being caused by the pneumococcus. This forest of "influenzas" and pneumonias in which recognized causative organisms could not be found still remained to be cleared. Later, the pneumonias that did not respond to chemotherapy and antibiotics had to be differentiated from those that did.

When English workers in 1933 found they could produce an influenza-like disease in ferrets by subjecting them to bacterial free washings from a sick man's throat, they opened another clearing in the jungle. This made it possible to define true influenza, but left a wilderness containing diseases often called "virus pneumonia" or the "grippe" for lack of more specific differentiation. Again quoting Geddis Smith: "A disease without a known cause is like

Q fever
 Symptoms p²
 can get w⁷
 from tick
 bite or
 raw milk

kite without a tail: it bobs uncertainly in the wind." True, modern therapy has made it possible to reduce the dangers of the unknown on a hit or miss basis, but it is still necessary, before we can prevent a disease or hope to cope with it epidemiologically, to recognize the disease as a distinct clinical entity. An etiologic agent must be defined and its prevalence established.

The next step, after influenza, in clearing the jungle of the respiratory diseases, was recognition of the disease now called Q fever.

In 1937, Derrick (3) reported a new disease characterized by sudden onset, chilly sensations, headache, weakness, severe sweats, and a pneumonia attended by mild cough, scanty expectoration, and minimal physical findings. Actually, the only fact that set it apart as possibly a new disease was that eight of the nine cases he reported occurred among slaughterhouse workers in Brisbane, Australia. Perhaps for lack of a better name, or because he decided it was a distinct disease through questioning his patients, he called it Query fever, now abbreviated to Q fever. Derrick isolated the causative organism from the blood and urine of his patients. He named this organism Rickettsia burneti after Burnet (4), who recognized it as a member of the order Rickettsiales (5).

Almost simultaneously with Derrick's work in Australia, Davis and Cox (6) reported the discovery of a filterable infectious agent as the cause of the Nine Mile fever of the Bitter Root Valley of Montana. The organism, which they isolated from the wood tick, Dermacentor andersoni, was called Rickettsia diaporica by Cox (7) because of its filterability. It was later recognized that the

organism was not a true rickettsia because it was filterable (6,8), it did not stimulate the production of antibody against the X strain of Proteus vulgaris (1,9), and it did not produce the rash characteristic of other rickettsial infections; as a result the name Coxiella burneti was proposed by Philip (10) and it is so listed in Bergey's "Manual of Determinative Bacteriology" (11).

previous
line with
fever
new-1937

Dyer (12), in 1938, suggested the possibility of a relationship between American Nine Mile fever and Australian Q fever. He (13) and Bengtson (14) in the United States and Burnet and Freeman (15) in Australia have established this relationship conclusively. From a medical oddity peculiar to Australian slaughterhouse workers and to Nine Mile, Montana, Q fever has risen to recognition as a disease of world-wide importance from the standpoint of public health, agriculture, economics, and military operations.

Following their report on the previously mentioned nine cases, Derrick, Smith, and Brown (16) reported 152 naturally occurring cases, 141 of them among slaughterhouse and dairy workers, in Brisbane and surrounding rural areas. Until 1944, the study and recording of cases in Australia was maintained. During that time 217 cases of Q fever were confirmed in southeastern Queensland, 1 in northern Queensland (17), 10 in Adelaide, and 7 cases among laboratory workers (18). The majority of these cases were among slaughterhouse workers, farmers and farm-laborers, and forestry personnel.

The disease is known to occur throughout Europe (18-23). Caminipetros (19) mentions serologic evidence of the disease in Turkey, and C. burneti has been isolated from animals in that country (24). Beatti (25) reported a case of Q fever contracted by

a British surgeon working in Iran. Blanc and Bruneau (26) mentioned two cases in Algeria in 1946. Although no human cases have been reported from Morocco, ticks of the Hyalomina species, infected with C. burneti, have been removed from camels, mules, and cattle (26,28).

During World War II, Q fever became recognized as a disease having military importance. In 1941, the German army became exposed to an influenza-like epidemic that the troops called "Balkan Grippe" (19,21,29). The German cases were the first recognized clinically in Greece. In 1943 the disease was shown to be endemic in that country (1,19). An outbreak of Q fever occurred among British paratroops evacuating Athens in 1945 (30), and 50 cases of Q fever were reported among British troops stationed at Salonika and Athens in 1947. In these latter infections, raw milk from sheep and goats was implicated as the transmitter of the contagium. United States Army personnel stationed in rural areas of Italy, in 1944 and 1945 (30), suffered six outbreaks involving 500 troops. In addition, 20 more persons in Italy were reported to have Q fever by the 15th Medical General Laboratory (31), and British troops stationed in Italy were subject to similar outbreaks. Eighty-five other cases among United States troops were reported from Corsica in 1944 (30).

It is interesting to note that in the above-mentioned outbreaks in Italy no natives were affected. Examination of 28 Italians, resident in the affected areas, showed 16 to have antibodies against Q fever. This and the Moroccan experience would suggest immunity of native populations in endemic areas.

In the Italian outbreaks, the etiologic agent was considered to be air-borne in dust contaminated by animal or insect feces (32).



Figure 1. World distribution of Q Fever in 1951. From Berge, T. O., and Lennette, E. W.,
World distribution of Q Fever: Human, animal, and arthropod infection. *Am. J.*
Hyg. 57:2, 125-43, March 1953.

Derrick was unsuccessful in attempting to connect the high prevalence of Q fever among meat and dairy workers with the presence of infected tick feces on the hides of cattle. The Australian workers implicated the bandicoot as the reservoir of C. burneti and four species of ticks as intermediate hosts (33,37). Cattle, the camel, the mule, and one species of tick have been implicated as hosts of C. burneti in Morocco. In Hamilton, Montana, C. burneti was first isolated from the wood tick by inoculation of guinea pigs. In addition, the Hamilton workers have infected a wide range of domestic and wild animals with C. burneti. These include bovines, sheep, goats, mules, horses, dogs, cats, chickens and parakeets, field mice, the white-footed mouse and house mouse, squirrels, the mountain rat, the porcupine, chipmunks, the cottontail rabbit, sparrows and pigeons (unpublished data from Rocky Mountain Spotted Fever Laboratory, Hamilton, Montana). In other studies, several species of ticks have been found to be intermediate hosts (38,39).

In the Western Hemisphere, except for one case of atypical broncho-pneumonia in Panama from which Cheney and Geib (40) recovered a strain of C. burneti and two cases described by Rodaniche and Rodaniche (41), Q fever seems to be important only in the United States. Prior to 1946, only one naturally occurring case of Q fever (42) was reported in the United States, but an outbreak among laboratory workers had occurred in 1940 (43). Since 1946, several American outbreaks have focussed attention on Q fever as a disease having public health importance. In 1946, an epidemic of Q fever in Amarillo, Texas, totalled 55 cases including two deaths (44,47). Another outbreak in Chicago, Illinois, a few months later, involved

33 cases (48). The exact mode of transmission in these outbreaks was not determined, but it was surmised to be the inhalation of dust particles or aerosols.

The endemic nature of the disease was realized in 1947 when Young (49) discovered several cases in Los Angeles County, California. Later studies showed the endemic area to extend into the neighboring counties of Orange and Ventura. In less than a year, 16 counties were involved. In Los Angeles County, 300 cases and three deaths were reported. A survey of 3,000 persons in this endemic area indicated that 1 percent of the population possessed serum antibodies for Q fever (1).

Raw milk samples taken from several dairies in Los Angeles contained C. burneti. In fact, the raw milk from 40 of 63 dairies in the Los Angeles area contained sufficient C. burneti to infect guinea pigs readily. In addition, 10 percent of 4,000 cows tested were infected. Proximity to dairies was a factor in more than 50 percent of the cases reported, but commercial pasteurization appeared to be almost 100 percent effective in eliminating infection from milk.

The Los Angeles studies brought about an interest in Q fever in the United States. The disease has been diagnosed, or infected ticks demonstrated, in Illinois (48), Texas (44,47,50,51), Massachusetts, Minnesota, Oregon (51), New York (52), Pennsylvania (53), Arizona (1), California (54,55), Montana (56), Idaho (57), and Wyoming (38).

Figure 2 shows Wyoming and Idaho to the north, Arizona to the south, and California only 500 miles removed from the western border of Utah to have reported human cases of Q fever or to have ticks

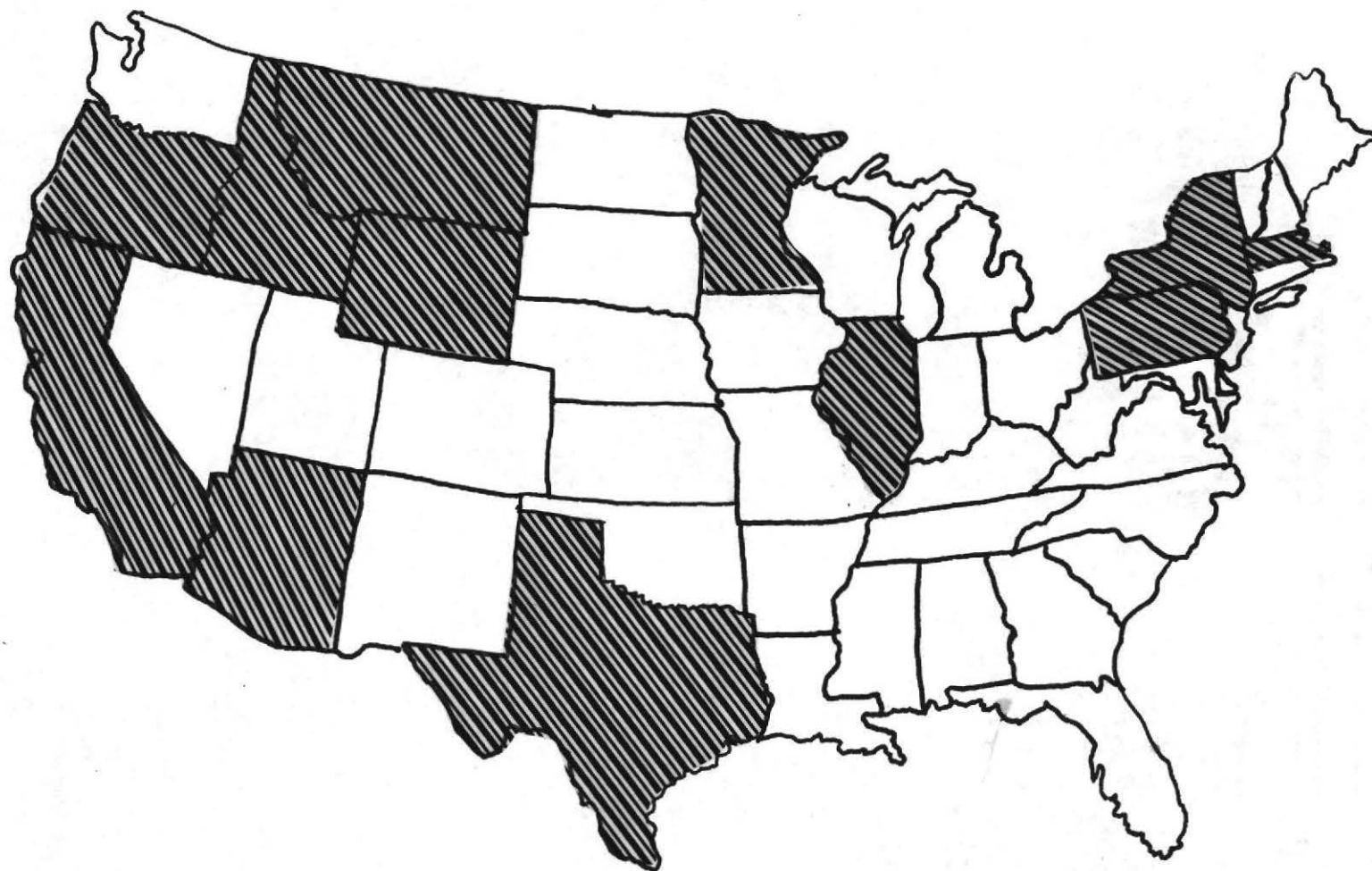


Figure 2. States in which Q Fever was reported or infected ticks isolated by 1954.

infected with the causative agent of the disease. Yet strangely enough, only one case of Q fever was reported in Utah before 1956.

Because of these highly contrasting circumstances (prevalence of the disease in nearby states and only one report of the disease in Utah), a survey was conducted to determine whether Q fever was present in Utah, and if so to what extent.

PURPOSE AND SCOPE

The purpose of the survey was three-fold: 1. to determine whether Q fever is endemic in Utah; 2. to establish the incidence of the disease; and 3. to analyze the results for factors that might furnish further information useful to persons responsible for the public health.

On the basis that demonstrable antibody in the blood is indicative of past or chronic infection, specimens were chosen at random from various areas in Utah and were tested for Q fever antibody by complement fixation methods.

METHODS AND MATERIALS

Source of Specimens

Serums for this study were obtained primarily from specimens sent to the Utah State Department of Health, Bureau of Laboratories, for routine serologic tests for syphilis. A secondary source of specimens was the American Red Cross blood donor service.

Patient's names and addresses were taken from information included with each blood specimen. All specimens were identified by number, but no patient's home address was determined until all tests were completed so as not to prejudice the findings in any way.

Preparation of Serums

Serums to be tested were properly labeled for later identification and stored in the deep freeze (-28° C.) until they could be tested. After thawing, each serum was inactivated for 30 minutes at 56° C. before testing. Only clear serums, negative for syphilis serology were tested.

Test Technique

The Kolmer complement fixation (58) method was used in this study. The test was modified to use only half portions of all reagents in order to reduce the cost of the study.

Complement and hemolysin titrations were done each day that tests for Q fever were performed.

All serums were screened at a dilution of 1:8 in order to eliminate low titred, non-specific reactions. Serums showing any degree of fixation of complement were retested at serial dilutions of 1:8 to 1:64. Simultaneously, these serums were tested against Rocky Mountain Spotted Fever or South African Tick Bite Fever

antigens in order to determine the specificity of the reactions.

Whenever a serum gave a specific positive reaction for Q fever, an explanatory letter and a questionnaire (see next page) were sent to the physician who had submitted the specimen.

Results were recorded upon completion of the test as either negative (complete hemolysis) or positive for Q fever.

Antigen

Q fever antigen was supplied by the Rocky Mountain Spotted Fever Laboratory, Hamilton, Montana. Both Henzerling (59) and Montana Nine Mile (6) strains were used.

The antigen was used in the dilution recommended by the issuing laboratory. The sensitivity of each new lot was checked using known control serum. No significant variations in reactivity were noted.

Complement

Commercial dehydrated guinea pig complement was used throughout the survey. The complement was rehydrated according to the instructions supplied by the processor. When not in use, the dehydrated complement was kept under refrigeration (6-10° C.); the constituted complement was frozen.

Q fever Case Report

Patient's Name _____ Age _____ Sex Male _____
 Female _____
 Present Address _____ No. Years _____
 Former Address _____ No. Years _____
 Occupation _____
 Laboratory Number _____ Date Received _____
 Result of Complement Fixation Test for Q Fever _____

Has patient has a clinical history suggesting Q fever? Yes ___ No ___
 (Sudden onset, headache, myalgia, anorexia, chilly sensations
 and fever which may fluctuate one or more times daily, weakness
 and pleuritic pain, or general symptoms of primary atypical
 pneumonia).

If so, when: _____

Has patient had contact with livestock? Yes ___ No ___

Dairy Cattle _____
 Range Cattle _____
 Sheep _____
 Goats _____

Is patient's home near dairy (within one mile)? Yes ___ No ___

If so, please give name of dairy. _____

Regular milk supply is: Raw _____ Past. _____ Homog. _____

Is there a history of tick bite? _____ Yes _____ No

If so, when: _____

Remarks: _____

Physician _____

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RESULTS

The results of this survey are summarized in Tables 1 and 2. Table 1 shows that the percentage of positive specimens for the 29 counties ranged from 0.00 - 7.74 percent. The average for the entire state was 1.71 percent. One of the most interesting facts revealed by this survey is that a portion of the state representing only about 13 percent of the population produced 68 percent of the reactors. This is chiefly the southwestern part of the state.

Of 119 questionnaires sent out as a follow-up on reactors, 63 were returned. Twenty of these contained insufficient information to evaluate. Of the 43 which could be evaluated, 93 percent implicated raw milk or farm animals. Furthermore, 55 percent showed the person involved was using raw milk at the time of the survey.

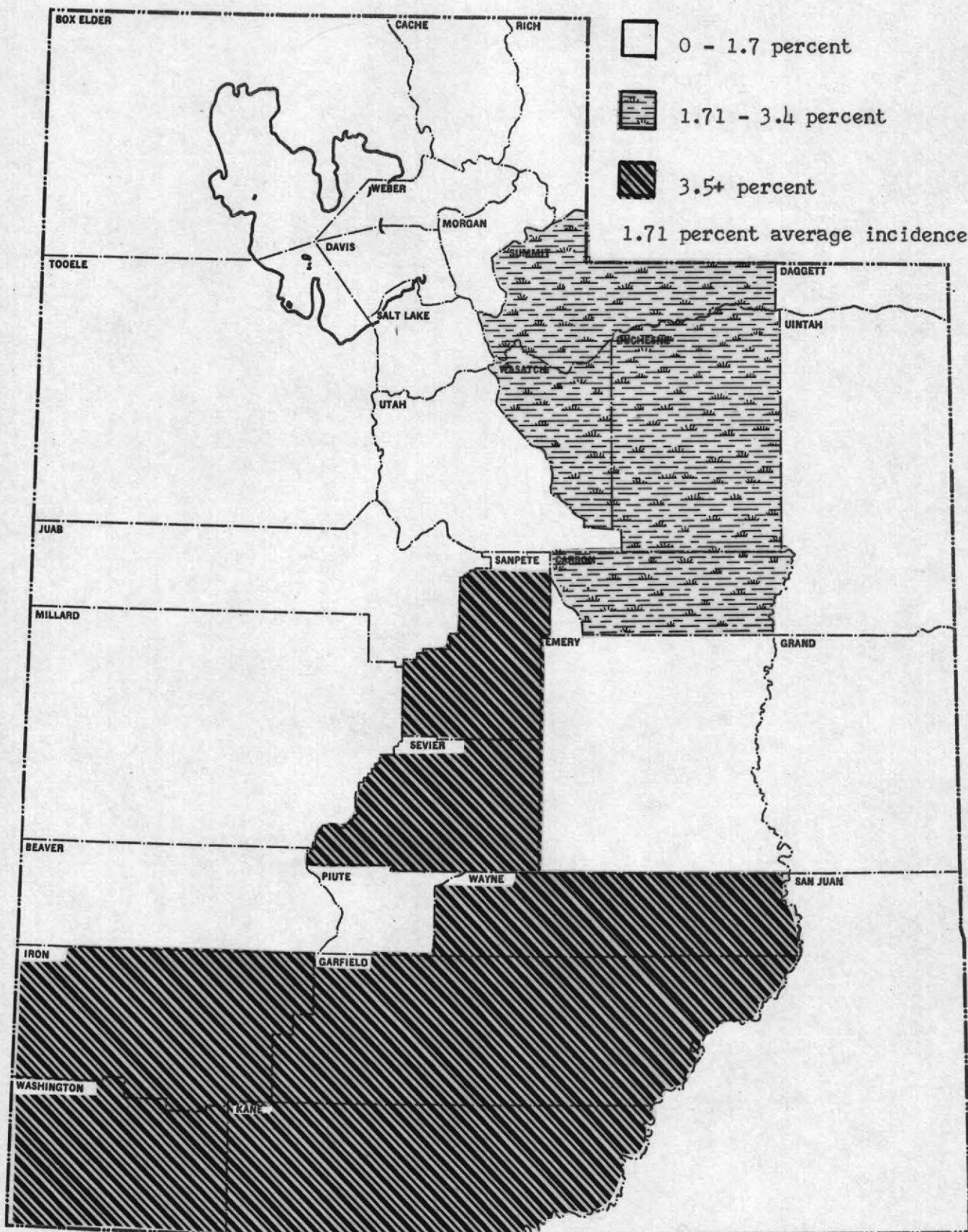
Table 1. Distribution of positive serologic reactors to Q fever in Utah by county, in decreasing order of incidence.

COUNTY	EST. POPULATION	NO. TESTED	% POP.	NO. POS.	% POS.
Sevier	12,885	155	1.20	12	7.74
Iron	10,481	217	2.07	16	7.37
San Pete	14,451	121	.84	8	6.61
Kane	2,465	73	2.96	4	5.48
Washington	10,620	259	2.44	13	5.02
Wayne	2,358	22	.93	1	4.55
Garfield	4,424	45	1.02	2	4.44
Carbon	26,703	711	2.66	24	3.37
Wasatch	5,973	73	1.22	2	2.60
Summit	7,165	97	1.35	2	2.06
Duchesne	8,833	147	1.66	3	2.04
Millard	10,030	124	1.24	2	1.61
Juab	6,305	68	1.08	1	1.47
Beaver	5,234	144	2.75	2	1.38
San Juan	5,759	76	1.32	1	1.32
Davis	34,237	234	.68	3	1.28
Weber	91,384	553	.61	5	0.90
Utah	88,926	489	.55	4	0.82
Uintah	11,179	251	2.25	2	0.80
Cache	35,990	341	.95	2	0.58
Box Elder	21,202	213	1.01	1	0.47
Salt Lake	297,038	2,080	.70	8	0.38
Tooele	16,041	314	1.96	1	0.32
Daggett	378	10	2.65	0	0.00
Emery	6,642	57	.86	0	0.00
Grand	2,001	23	1.15	0	0.00
Morgan	2,706	32	1.18	0	0.00
Piute	2,010	21	1.04	0	0.00
Rich	1,760	2	.11	0	0.00
Totals	745,180	6,952	.93	119	1.71

Table 2. Distribution of positive reactors according to sex.

COUNTY	FEMALES	NO. POS.	% POS.	MALES	NO. POS.	% POS.
Sevier	67	6	8.96	52	5	9.62
Iron	142	8	5.63	72	8	10.67
San Pete	75	5	6.67	37	3	8.11
Kane	26	4	15.38	9	0	0.00
Washington	121	10	8.26	38	2	5.26
Wayne	4	0	0.00	8	1	12.50
Garfield	26	1	3.85	19	1	5.26
Carbon	88	1	1.14	46	1	2.17
Wasatch	44	1	2.27	9	1	11.11
Summit	44	2	4.55	15	0	0.00
Duchesne	64	3	4.69	27	0	0.00
Millard	36	1	2.78	18	1	5.56
Juab	13	0	0.00	5	0	0.00
Beaver	84	2	2.38	60	0	0.00
San Juan	22	0	0.00	32	1	3.12
Davis	96	2	2.08	34	1	2.94
Weber	173	2	1.16	85	1	1.18
Utah	174	2	1.15	96	1	1.74
Uintah	65	2	3.08	39	0	0.00
Cache	120	1	0.83	37	1	2.70
Box Elder	113	0	0.00	36	1	2.78
Salt Lake	903	2	0.22	758	4	0.53
Tooele	189	0	0.00	125	1	0.80
Daggett	0	0	0.00	10	0	0.00
Emery	12	0	0.00	10	0	0.00
Grand	15	0	0.00	8	0	0.00
Morgan	6	0	0.00	3	0	0.00
Piute	12	0	0.00	9	0	0.00
Rich	1	0	0.00	1	0	0.00
Totals	2,735	55		1,698	34	
Average			2.00			2.00

Figure 4. Distribution of Q fever in Utah, 1956



DISCUSSION

Although a 1 percent sample of each county's population was originally planned, the actual percentages varied for one of three reasons: 1. The source of specimen was not finally determined until the tests on the specimen had been completed. 2. When a significant number of specimens had been examined and a definite trend established from the more populous counties, the tests were discontinued. 3. Some counties had no physicians submitting blood to the State Health Department. Although a few tests were run on patients from these counties, not enough were obtained to have value in a survey of this type.

The majority of the serums were from a portion of the population that would be considered "healthy," because many specimens came from potential blood donors, applicants for marriage licenses, pre-employment physicals, and pregnant women.

Two reasons prompted selection of specimens from these groups of persons. The more important was that in a survey of this nature an attempt was being made to locate possible past infections. If Q fever had proved to be endemic to the entire state, and if specimens had been taken from diagnostic problem cases, the percentage figures of incidence might have been disproportionate to the total number of specimens tested. The percentage of reactors recorded might actually be too low when we note that these "diagnostic" bloods were not included in the survey.

The second reason was that past experience has shown that it is not desirable to give physicians or patients any advance information about a survey of this type. When physicians and their patients

become aware of surveys, there is usually a sudden rise in the number of reported cases of the disease under study. In order to avoid such a "scare" situation, the aforementioned "healthy" specimens were selected. By so doing, no great inconvenience either to physician or patient would be caused by any delay in reporting positive results.

During the course of this study, Uintah County was surveyed on a large scale by the State Health Department. Q fever data from this survey were maintained independently of the main Q fever survey. A total of 3,407 blood samples from Uintah County were tested. They represented an additional 30.48 percent of the population of Uintah County. Twenty-five positive tests were found. The percentage incidence in this special study was 0.73, which compares favorably with the 0.80 percent incidence recorded in Table 1 for Uintah County.

If the percentage reactivity for the entire survey is projected into the entire population of the state, the real public health importance of Q fever in Utah may be seen.

To illustrate, if eight of 2,080 persons in Salt Lake County showed antibodies against Q fever, one might speculate that almost 1,100 persons in the county have been infected. The dispersion of persons infected can be seen even in those counties which actually had a low percentage incidence of reactors.

When a county such as Sevier with a reactivity rate of 7.74 percent is encountered, one wonders why actual cases from this area were not reported, especially after noting that in 1948 a survey of 1,238 slaughterhouse workers in Fort Worth, Texas, showed 8.0 percent, or almost 100, to have serum antibodies for Q fever (50). And This was in a specialized occupational group!

One reason that Q fever is not being reported in Utah may be that the disease is not being recognized. Since the symptomology of Q fever is so general and so similar to that of influenza and the common cold, Q fever is not easily differentiated from them. Consequently, patients having these symptoms are given the standard treatment of an antibiotic along with an order to rest.

In fairness to the physician, we must note that laboratory facilities for the detection of viral and rickettsial diseases have been limited. In fact, because of these limitations, a survey of this type could be conducted with a minimum of outside pressure, since no requests for Q fever testing were made on any of the bloods chosen for this survey.

Piute County presents a special problem. No human serum tested from Piute County showed complement fixing antibody for Q fever.

No full time physician lives in Piute County. Samples from that area had to be obtained when physicians from Sevier County held clinics in Piute and submitted blood specimens to the Richfield Branch Laboratory. However, about 1 percent of the population was tested, and not a single reactor was found. This seems remarkable since neighboring counties showed high rates of reactivity among their populations.

A complete epidemiological study by the proper public health officials might be warranted in those counties that have shown a high incidence of reactors, and in any county, such as Piute, that seems to be a "misfit" epidemiologically speaking. Such a study should include a survey of possible animal reservoirs. An attempt should also be made to isolate the rickettsia from milk. Elaborate

laboratory and animal housing should be acquired before attempting such an isolation, however.

Although a great deal of personal interviewing would be necessary, a vigorous attempt should be made to determine whether those persons having blood tests positive for Q fever have ever been in areas endemic for Q fever outside of Utah. If so, where, when, and for how long were they exposed? From such information an epidemiologist could determine the steps to take in the control of Q fever in Utah.

CONCLUSIONS

The accumulated data suggest that Q fever either has been widespread in certain portions of Utah, or that it is in the process of spreading. The high incidence in some counties of persons having complement fixing antibodies in their blood points to a problem of public health significance.

With the exception of Piute County, an almost unbroken "belt" can be drawn from southwestern Utah into the central portion of the state. This belt includes Washington, Kane, Iron, Garfield, Wayne, Sevier, and San Pete counties.

Even in those counties showing a relatively low percentage incidence of Q fever, a projected estimate of persons infected shows that large numbers of persons have been exposed to Q fever.

Since Q fever has not been recognized by Utah's physicians, the public health importance of Q fever has not been fully realized.

SUMMARY

An attempt was made to show the incidence and distribution of Q fever in Utah. A complement fixation test for Q fever was performed on serums sent to the Utah State Department of Health, Bureau of Laboratories, for routine syphilis serology. Of the serums tested, 1.71 percent showed antibody for Q fever.

In some counties, the reactive serums exceeded 7.0 percent of the specimens tested from those counties (based on an approximately 1 percent sample of the population).

Q fever was shown to be a disease having public health importance in Utah.

Suggestions are made for further investigation.

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