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# THE GEOGRAPHICAL DISTRIBUTION OF Q FEVER

MARTIN M. KAPLAN, V.M.D., M.P.H.
*Chief, Veterinary Public Health Section,*
*Division of Communicable Disease Services,*
*World Health Organization*

P. BERTAGNA, D.Sc.
*Division of Communicable Disease Services,*
*World Health Organization*

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SYNOPSIS

The results of a WHO-assisted survey of the distribution of Q fever in 32 countries and an analysis of reports published to date indicate that Q fever exists in 51 countries on five continents. Q-fever infection was most often reported in man and the domestic ruminants, such as cattle, sheep, and goats.

The disease was found to exist in most countries where investigations were carried out. Notable exceptions were Ireland, the Netherlands, New Zealand, Poland, and the Scandinavian countries. With the exception of Poland, where the results were inconclusive, all these countries import relatively few domestic ruminants—the most important animal reservoirs of human Q-fever infection. It seems, therefore, that the traffic of infected ruminants may be one of the most important, if not the most important, means for the geographical spread of Q fever. The importance, if any, of ticks associated with such traffic needs to be defined.

In 1950, the Third World Health Assembly, aware of the potential danger of Q fever to public health and of the large gaps in the existing knowledge of the disease, passed a resolution calling for a preliminary study of its prevalence throughout the world.[a] A programme of epidemiological research and surveys was accordingly encouraged by WHO in 33 countries.

In 1953, Berge & Lennette [16] published a report on Q fever in various countries, outlining the known prevalence at that time. Additional information has been acquired, particularly from areas not covered in their review, through the WHO-assisted surveys and from other published reports.

In this paper we shall attempt to summarize, rather than to review in detail, the available information on the geographical distribution of Q fever.

[a] *Off. Rec. Wld Hlth Org.* 1950, **28**, 3

Wherever it is justified by ample data, some indication is given of the reservoirs of infection in a country and of the areas affected. No attempt has been made to include data from all published reports concerning specific areas; preference has been given to those reports which have the most geographical information on proved infection, prevalence, and reservoirs of infection of the disease.

Nor are the epidemiology and natural history of Q fever discussed here, as these are excellently covered in the review by Stoker & Marmion on page 781 of this number of the *Bulletin*. As these authors point out, our areas of ignorance are large in respect of the natural history of this disease, and only tentative hypotheses can be advanced at this time. It is clear, however, that the principal reservoirs of Q-fever infection for man are to be found, singly or in combination, in cattle, sheep, and goats, and in ticks; the role of other domestic animals, rodents and other small mammals, and birds appears to be distinctly secondary. Ticks figure only very occasionally as a possible direct cause of human outbreaks, although they are important epidemiologically in certain areas in perpetuating *Rickettsia (Coxiella) burnetii* in nature. Thus, man appears to acquire his infection in the main from cattle, sheep, and goats.

## DIAGNOSTIC CRITERIA

Laboratory tests are the sole means of making a definitive diagnosis of Q fever since either the symptomatology in man and animals is non-specific or the infection is clinically inapparent. Babudieri [4] has considered in detail the laboratory procedures commonly used in Q fever, and only some aspects of these, and certain new procedures, are considered here. Particular attention is given to the reliability of these procedures for definitely establishing the presence of the disease in a locality.

### Isolation and Identification of " Rickettsia burnetii "

The isolation of *R. burnetii* is best done by inoculating laboratory animals, particularly guinea-pigs, with suitably prepared specimens.[4, 152] Pre- and post-inoculation samples of sera from the laboratory animals are tested for complement-fixing or agglutinating antibodies. Additional procedures may include passages of spleen tissue from animals having a febrile reaction, and cross-immunity tests, but the serological tests are usually accepted as sufficient proof of specificity (see below). Isolation of the organism is used to detect active infections, reservoirs, or shedders of *R. burnetii*, but if relied upon alone it will fail to detect many current as well as past infections.

## Complement-Fixation Test

This test has been the procedure most widely used in epidemiological surveys because complement-fixing antibodies persist for many months and even years after infection, although persistence in individual cases is unpredictable.[4, 100, 148, 207, a]

Recent studies by Stoker and his colleagues in the United Kingdom have shed important light on the application of the test to both human and animal sera.[a] These workers demonstrated marked differences in the sensitivity of the two antigens most widely used, prepared from the Nine Mile and Henzerling strains, respectively, of *R. burnetii*. For human sera, the Nine Mile antigen was more sensitive than the Henzerling antigen; [186] this was also observed by Taylor in Egypt.[204] However, with the sera of sheep from different localities, now one, now the other of the antigens proved the more sensitive. The authors point out that these differences of antigens might be significant, at least with respect to human sera, only during the first year after infection—a view which agrees with results obtained by Berge & Lennette.[17]

Stoker, Page & Marmion[a] also consider the question of non-specific reactions, and they attribute these to such varied causes as " primary atypical pneumonia " (of the type stimulating cold agglutinins) and conditions of storage and transit of blood specimens. Some doubts that brucellosis [115] and syphilis [162] might cause non-specific reactions for Q fever have been dispelled.[99, 138]

The difficulties and limitations of the complement-fixation test as pointed out by Stoker and his colleagues[a] are certainly significant when the test is used for clinical diagnoses of individual cases or for research purposes. It should be kept in mind, however, that these difficulties are encountered primarily in low dilutions of serum (usually below 1/10), and that otherwise the test retains a high degree of specificity and usefulness for detecting present and past infections.

The high specificity of the complement-fixation test is supported by the almost total absence of positive reactions in serum dilutions higher than 1/10 in tests involving many thousands of human and animal serum samples in " negative " countries, notably the Netherlands and the Scandinavian countries (see Table I). We shall discuss these points again as they relate to the WHO-assisted surveys and the interpretation of data reported from different countries.

## Agglutination Test

The agglutination test is at least equal in specificity to the complement-fixation test, and does not appear to have some of the limitations of the latter with respect to non-specific reactions.[a] However, much more antigen

[a] See paper by Stoker, Page & Marmion on page 807 of this number of the *Bulletin*.

—an expensive preparation—is required for the agglutination test [4, 100] than for the complement-fixation test. A micro-agglutination technique can be used to save antigen.[4] A capillary agglutination test, using stained antigen in a glass capillary tube, has been developed and appears to be a simple and useful procedure for screening tests on bovine and human sera [105, 195] as well as on mixed milk samples from cows.[106, a]

Many workers have found that blood-serum agglutinating antibodies usually disappear before the complement-fixing ones, or are less easily detectable, [12, 38, 100, 148, 193, 196] although others claim a slight superiority for the agglutination test.[4, 72, 105]

### Intradermal Test

This test in both man [4, 11, 75, 104] and animals [8, 117] appears to be useful for survey purposes from the standpoint of specificity and detection of past exposure to infection. The procedure has not been used very widely because of certain disadvantages, such as the necessity of second visits for reading purposes, high cost of antigen, difficulties in reading because of skin colour, and sometimes the production of undesirable local and systemic reactions.

### Anti-globulin Sensitization Test

An anti-globulin sensitization test, although a more involved procedure than the complement-fixation test, was found to have certain advantages in sensitivity over the latter and was much more sensitive than the agglutination test in examinations of human sera [38] and whey from cow's milk.[158] The test has not been widely used to date.

### Opsonin Test

The opsonin test was considered a valuable epidemiological tool in a large survey of human sera in the Los Angeles area. [176] The test, however, has not been applied on a large scale elsewhere.

## THE WHO-ASSISTED SURVEY

In the WHO-assisted survey, started in 1951 in 33 countries, the basic procedures suggested were the complement-fixation test combined, where possible, with attempted isolation of *R. burnetii* in guinea-pigs (details are published elsewhere [90]). For survey purposes three approaches were advised to determine the presence of Q fever in a locality: (1) the examination of blood sera of employees in fat-rendering plants and abattoirs; (2) inocula-

[a] It is worth mentioning here the possibility of the conglutinating phenomenon operating in certain agglutination test reactions, particularly where bovine serum is involved.[12] Also, in tests with human sera, direct conglutination of rickettsia gave results comparable to the haemolytic complement-fixation test, both being superior to a straight agglutination test,[12] while a conglutinating complement-absorption test on human and bovine sera showed greater sensitivity than the complement-fixation test.[180] The use of these more complicated serological techniques (see also below) has been confined to a very few laboratories.

tion into guinea-pigs of milk samples pooled from 100 to 200 animals, with pre- and post-inoculation examination of the guinea-pig sera for complement-fixing antibodies; and (3) examination by complement-fixation test from individual animals selected at random in an area. These procedures, especially if used in combination, were selected as the most likely to detect the presence of Q fever in areas and were adaptable to the facilities of most of the laboratories participating in the survey.

A commercially-produced Henzerling strain antigen and positive control serum (guinea-pig) for antigen titration were supplied by WHO. Nine Mile strain antigen was substituted for the Henzerling antigen towards the end of the survey; thus, most of the results recorded pertain to the latter antigen. As indicated above, according to the findings of Stoker, Page & Marmion and Taylor [204] it is possible that with the Henzerling antigen a certain number of positive cases might have been missed. From the standpoint of the results of the survey, this would be important only as regards the countries shown to be negative, notably the Netherlands and the Scandinavian countries. In view of the large number of serum specimens examined in these countries (see Table I) the chances are very remote that the presence of infection would not have been detected.

The complement-fixation test used [90] followed primarily the Kolmer technique. Sera were screened at a 1/16 dilution and positive reactions were titrated out to 1/128. The usual controls (antigen, complement, red cells) included a known positive serum (homologous if possible), a known negative homologous serum, and anti-complementary activity of the serum under test. With these precautions it was felt that low-titred non-specific reactions and other limiting factors of the test pointed out by Stoker, Page & Marmion would not be important factors affecting the interpretation of the results, bearing in mind that the principal purpose of the survey was to detect the presence of Q fever in areas hitherto believed to be free from infection, and not to make individual diagnoses or careful comparative studies of prevalence.

## GEOGRAPHICAL DISTRIBUTION

The data in Table I are from published reports selected from a large number on the basis of their giving the most information on the definite presence of Q fever in a country, on the principal geographical areas involved, and on the natural sources of infection. The table also includes unpublished results from 32 countries participating in the WHO-assisted survey. We have no doubt overlooked some published reports in connexion with areas of countries involved, species affected within a country, and, perhaps, successful isolations of *R. burnetii*; but the data summarized in the table, showing both positive and negative results, are felt to give a reliable picture of the general situation.

**TABLE I. GEOGRAPHICAL DISTRIBUTION OF Q FEVER AND SPECIES AFFECTED**

| Country | Reference No. | Diagnostic test used | | Infected species [b] |
|---|---|---|---|---|
| | | complement-fixation test [a] | isolation of organism | |
| **Africa** | | | | |
| Algeria | 20 | + | + | Man, tick |
| Belgian Congo (Ruanda-Urundi) | 67, 68, 88 | + | + | Man, sheep, goat, cattle, horse, pig, dog, tick, body-louse |
| Cameroons (Douala and east and north-east) | 77, 133 | + | | Man, cattle |
| Egypt [c] (Cairo, Aswan, Nile valley) | 81, 166, 204 | + | + | Man, sheep, goat, cow, bull, water buffalo, tick |
| French Equatorial and West Africa (Ubangi-Shari, Upper Volta) | 73, 74, 77, 97, 129 | + | | Man, sheep, goat, cattle, donkey, dog |
| Kenya [c] (Muguga, Nairobi) | 82, 185 | + | | Man, cattle |
| Libya | 55 | + | | Man |
| Madagascar | 41 | + | | Man |
| Morocco (throughout) | 20, 21, 63 | + | + | Man, sheep, goat, cattle, gerbil, tick |
| Portuguese Guinea and São Tomé [d] | 132, 168 | + | + | Goat, cattle, birds, reptiles, tick |
| Southern Rhodesia | 65 | + | | Man |
| Sudan [d] | 166, 167 | + | + | Camel, bull, tick |
| Tunisia [c, d] | 39, 113 | + | | Man, sheep, goat, cattle |
| Union of South Africa [d] (Western Transvaal; Wellington, Cape Province) | 64, 136, 142, 153, 154, 155 | + | + | Man, cattle |
| **America** | | | | |
| Argentina | 7 | + | | Cattle |
| Brazil [c] | [e] | + | | Man |
| Canada (Quebec) | 126 | + | | Man |
| Martinique | 120 | + | | Man |
| Mexico | 163, 164 | + | | Man, sheep, goat, cattle |

[a] Taken as positive at titres over $^1/_8$ or $^1/_{10}$, depending on the dilution system used.

[b] For tick species which act as natural reservoirs of infection, see the article by Stoker & Marmion on page 781.

[c] One laboratory in each of these countries participated in the WHO-assisted survey.

[d] Agglutination test also found positive.

[e] Information obtained from a communication by J. Travassos, A. Ubatuba, N. da Paz Silva & M. T. de Mello to the sixth annual meeting of the Sociedade Brasileira para o Progresso da Ciencia, São Paulo, November 1954.

**TABLE I. GEOGRAPHICAL DISTRIBUTION OF Q FEVER AND SPECIES AFFECTED (continued)**

| Country | Reference No. | Diagnostic test used | | Infected species [b] |
|---|---|---|---|---|
| | | complement-fixation test [a] | isolation of organism | |
| **America** (continued) | | | | |
| Panama | 140 | + | + | Man |
| United States of America [c] (Arizona, California, Illinois, Massachusetts, Montana, New York, Ohio, Oregon, Texas, Virginia, Washington) | 15, 33, 51, 87, 98, 107, 108, 146, 147, 177, 182 | + | + | Man, sheep, cattle, tick |
| Venezuela [c] (Caracas) | 86 | + | | Man |
| **Asia** | | | | |
| Ceylon [c] | 143 | + | | Man, sheep, goat, cattle, buffalo |
| China (Peking) | 31 | + | | Man |
| India [c] (Bombay, Madras, Coonoor) | 173, 174 | + | | Man, sheep, goat, cow, bullock |
| Indonesia [c] | 205 | + | | Bovine |
| Iran (various provinces) | 42, 61, 71, 135, 183 | + | | Man, sheep, goat, cow, dromedary |
| Iraq (Habbaniya) | 208 | + | | Man |
| Israel [c] | 93, 190 | + | + | Man, cattle |
| Japan [c] (Kumamoto, Kanagawa, Okayama) | 92, 123, 165, 191 | + | | Man, sheep, goat, cattle |
| Jordan | 184 | + | | Sheep, goat, cattle, camel |
| Lebanon [c] | 192, 203 | — (600 cattle sera in 1954) + (capillary agglutination test on milk samples in 1955) | | |
| Malaya [c] | 23 | + | | Man, goat, cattle |
| Pakistan | 92 | + | | Camel |
| Turkey [c] (20 of 63 vilayets) | 78, 127, 128, 199 | + | + | Man, sheep, goat, cattle, buffalo, tick |
| Union of Soviet Socialist Republics (Central Asian oblasts) | 32, 94 | + | | Man, cattle |

[a] Taken as positive at titres over $^1/_8$ or $^1/_{10}$, depending on the dilution system used.

[b] For tick species which act as natural reservoirs of infection, see the article by Stoker & Marmion on page 781.

[c] One laboratory in each of these countries participated in the WHO-assisted survey.

**TABLE I. GEOGRAPHICAL DISTRIBUTION OF Q FEVER AND SPECIES AFFECTED** (continued)

| Country | Reference No. | Diagnostic test used | | Infected species [b] |
|---|---|---|---|---|
| | | complement-fixation test [a] | isolation of organism | |
| **Europe** | | | | |
| Austria [c] (Tyrol, Carinthia) | 85, 96, 200, 202 | + | + | Man, sheep, goat |
| Bulgaria (Sofia, Plovdiv) | 2 | + | + | Man, sheep, goat |
| Cyprus [c] | 187 | + | | Man, sheep, goat, cattle |
| Czechoslovakia (Prague) | 125, 137 | + | | Man, sheep |
| Denmark [c] | 198 | — (man and cattle; 1700 sera tested) | | |
| Finland [c] | 139, 201 | — (man and cattle; 3000 sera tested) | | |
| France [c] (Seine, Paris, Ardèche, Vaucluse, Allier, Montpellier) | 18, 30, 40, 47, 70, 76, 145 | + | + | Man, sheep, cattle, dog, pig |
| Germany, Federal Republic [c] (Hamburg, Eifel, Baden, Württemberg-Hohenzollern, Stuttgart) | 19, 26, 53, 60, 66, 83, 84, 95, 101, 102, 114, 122, 144, 169 | + | + | Man, sheep, goat, cattle |
| Greece [c] | 27 | + | + | Man, sheep, goat |
| Hungary (Budapest) | 54 | + | | Man |
| Iceland [c] | 149 | — (?) (few human and sheep sera tested) | | |
| Ireland [c] | 197 | — (man and cattle ; 800 sera tested) | | |
| Italy [c, d] (throughout) | 1, 3, 6, 9, 10, 24, 44, 45, 48, 62, 103, 116, 118, 119, 170, 172 | + | + | Man, sheep, goat, cattle, horse, dog, pig, goose, pigeon |
| Netherlands [c] | 89, 181, 209, 210 | — (man and cattle; 3200 sera tested, 1 doubtful reaction at 1/16) | | |

[a] Taken as positive at titres over $^1/_8$ or $^1/_{10}$, depending on the dilution system used.

[b] For tick species which act as natural reservoirs of infection, see the article by Stoker & Marmion on page 781.

[c] One laboratory in each of these countries participated in the WHO-assisted survey.

[d] Agglutination test also found positive.

**TABLE I. GEOGRAPHICAL DISTRIBUTION OF Q FEVER AND SPECIES AFFECTED** (concluded)

| Country | Reference No. | Diagnostic test used | | Infected species [b] |
|---|---|---|---|---|
| | | complement-fixation test [a] | isolation of organism | |
| **Europe** (continued) | | | | |
| Norway [c] | 188 | — (cattle) | | |
| Poland | 124 | — (?) (man; 1700 sera tested, 7 doubtful reactions below 1/16) | | |
| Portugal (throughout) | 56, 57, 59 | + | + | Man, sheep, goat, cattle, tick |
| Romania (Bucharest) | 35, 36, 37 | + | + | Man |
| San Marino | 172 | + | | Man, sheep, cattle, pigeon (?) |
| Spain [c] (Madrid and various provinces) | 34, 130, 131, 134 | + | + | Man, cattle, dormouse, wild rabbit, tick |
| Sweden [c] | 189 | — (man and cattle; 1100 sera tested) | | |
| Switzerland [c] (Aargau, St. Gallen, Zürich, Engadine) | 13, 22, 79, 80, 178, 179, 194 | + | + | Man, sheep(?), goat, cattle (?) |
| Union of Soviet Socialist Republics (Moscow oblast) | 32, 94 | + | | Man, cattle |
| United Kingdom of Great Britain and Northern Ireland [c, d] (Devon, Kent, London, Scotland, Wales) | 109, 110, 111, 112, 151, 156, 157, 159 | + | + | Man, sheep, goat, cattle, tick |
| Yugoslavia [c] (Belgrade ; Sokol, Bosnia; Ogulin, Croatia) | 43, 121, 150, 171, 206, 207 | + | + | Man, sheep, cattle |
| **Oceania** | | | | |
| Australia (New South Wales, Northern Territory, Queensland, South Australia, Victoria) | 14, 29, 49, 50, 52, 160, 161, 196 | + | + | Man, cattle, bandicoot, tick |
| New Zealand [c] | 141 | — (1400 sera tested) (Imported animals showed infection in quarantine) | | |

[a] Taken as positive at titres over $^1/_8$ or $^1/_{10}$, depending on the dilution system used.

[b] For tick species which act as natural reservoirs of infection, see the article by Stoker & Marmion on page 781.

[c] One laboratory in each of these countries participated in the WHO-assisted survey.

[d] Agglutination test also found positive.

It is apparent from Table I that the complement-fixation test was the principal means of establishing the presence of infection. In all instances where positives (+) are recorded in the table under that test, titres of over 1/8 (or over 1/10, depending on the dilution system used) were obtained either in a fairly large number of examinations, with adequate controls, or with sufficiently high titres to minimize the possibility that non-specific reactions could be solely responsible. The latter possibility becomes almost non-existent when one considers the lack of positive reactions encountered in the large number of tests, carefully performed with adequate controls, in the Netherlands and the Scandinavian countries.[a]

It can safely be concluded, therefore, that Q-fever infection exists in countries at least to the extent recorded in Table I, that is, in 51 countries of the world.

In the following pages are given the results of the survey country by country, under the broad grouping of the five continental masses.

## Africa

### *Algeria*

In 1951 Blanc [20] reported on two clinical cases, and *R. burnetii* was isolated from cattle ticks (*Hyalomma mauritanicum*).

### *Belgian Congo*

Giroud [67, 68] has described outbreaks of Q fever in Ruanda-Urundi on a number of occasions. The first isolation of *R. burnetii* was done in 1949 from three human subjects. The organism has also been isolated from cows' and goats' milk, dog and cattle ticks, and human body lice. Jadin [88] states that Q fever is exceedingly common in many places in Ruanda-Urundi. He describes an outbreak in which 450 cases with 40 deaths occurred among a population of 6000. In another outbreak, out of 32 patients selected at random, 25 were positive (titres: 1/160 and above). Ticks (*Ornithodoros moubata* and *Haemaphysalis leachi*) and human lice were found infected with *R. burnetii*. Serological tests on animals were clearly positive with cattle, goats, dogs, sheep, pigs, and horses (titres: 1/16 and above).

### *Cameroons*

Giroud et al.[77] state that in the slaughter-houses of Douala, workers have come into contact with cattle from all over the east and north-east of the Cameroons. Tests performed among 113 healthy workers resulted in

[a] In one instance (Lebanon), the presence of infection was indicated by an agglutination test where a survey with the complement-fixation test failed to uncover the disease. The highly endemic state of Q fever in countries surrounding Lebanon supports the probability of Q fever being present there also.

16 positive sera (titres: 1/16 to 1/40). A recent report [133] indicates that Q-fever infection was also diagnosed at Douala in a child with encephalitic symptoms.

*Egypt*

Halawani and collaborators,[81] conducting the WHO survey, reported that of 200 cows and buffalo cows, 22 were positive with titres ranging from 1/32 to 1/128. Of 15 goats and 217 sheep, 4 and 14 respectively gave positive reactions. Eleven positive reactions were obtained from 77 human sera. Research carried out by Taylor,[204] and by Taylor, Mount & Hoogstraal [166] indicates that ticks collected from camels (*Hyalomma dromedarii* Koch) and from bulls (*H. excavatum* Koch) imported from Sudan have been found infected with *R. burnetii*. An investigation in the Cairo abattoir showed that 9% of cows, 7% of sheep, 27% of goats, and 33% of gamooses (water buffaloes) were infected. Among 487 sera from Cairo, Aswan, and seven villages in five provinces along the Nile Valley, 20% were positive (titres: 1/8 and above). In one village of a sixth province 39% of 178 sera were positive, and of 52 sera from children under five years of age in the neighbouring villages, 54% were positive.

*French Equatorial Africa and French West Africa*

Giroud et al.[74] have reported the presence of Q-fever infection since 1949. Positive complement-fixation reactions were obtained in humans, cattle, sheep, goats, donkeys, and dogs. In 1952, they also reported [73] that in one agricultural village almost 50% of the population gave positive reactions, whereas in another community comprising mainly hunters only 2 of 30 inhabitants were positive. Still in 1952, Le Gac, Giroud & Lemaigre [97] diagnosed serologically the presence of *R. burnetii* in two pygmies out of 20 examined in the Lobaye (Ubangi-Shari). In the control region of Ubangi-Shari, where domestic cattle are numerous, 35% of the people reacted with Q-fever antigen. These authors claim that the disease does not exist endemically. In 1953, Giroud et al.[77] at Bobo in Upper Volta carried out serological tests (*a*) on 31 shepherds, of whom 4 were positive (titre: 1/32); (*b*) among slaughter-house workers, of whom 9 reacted with titres of 1/16 or 1/32; and (*c*) on 19 cattle with one positive result (1/20). Recently, however, Pellissier [129] obtained only 0.7% positive complement-fixation reactions at titres of 1/4 or over among 814 sera tested.

*Kenya*

Harris,[82] in 1952, described one human clinical case with a positive complement-fixation reaction. In 1955, in the WHO survey, the screening of 169 sera of goats by the East African Veterinary Research Organization

in Muguga provided 77 (46%) positive at 1/16 or more.[185] Out of 170 cattle about 6% were positive (1/16). Among humans in Nairobi, 2 of 10 Africans were positive, one of 10 Asians, and 3 of 17 Europeans (1/16), or a frequency of approximately 16%.[185]

*Libya*

An explosive outbreak of 25 cases of Q fever occurred in February and March 1951 among men of the United States Air Force stationed in Tripoli, 22 of them being positive (titres: 1/20 to 1/320).[55]

*Madagascar*

The first case was diagnosed in 1951 by Courdurier, Buck & Quesnel [41] from 12 human sera (titre: 1/40) from cases with atypical pneumonia. A survey conducted by the same authors on 201 bovine sera and 30 human sera (farmers and abattoir workers) gave negative results.

*Morocco*

In 1947 Blanc, Martin & Maurice [21] notified the discovery of a focus of Q fever near Goulimine in southern Morocco. No cases of the disease in man were found there, but three strains of *R. burnetii* were isolated from ticks identified as *Hyalomma savignyi* Gerv. These ticks were widely distributed in the district on various domestic animals. In 1951, Blanc [20] described outbreaks of Q fever in six towns scattered throughout Morocco; *R. burnetii* was isolated from cattle ticks (*Hyalomma lusitanicum*, *H. savignyi*, *H. excavatum* Koch) and gerbils. In eight regions of the Ziz valley, Garin [63] examined 783 human and 187 animal sera; 38% infection was found in the humans, 55% in goats, 45% in cows, and 38% in sheep (titre: 1/40).

*Portuguese Guinea*

Strains of *R. burnetii* have been isolated in the Laboratory of Veterinary and Animal Industry from local species of ticks and from the milk of cows and goats, in various non-domestic mammalians, in birds, and even in reptiles.[168]

*São Tomé (Portugal)*

The complement-fixation test gave positive results in 4 of 49 human sera at a titre of 1/20.[132]

*Southern Rhodesia*

Gelfand & Berney [65] reported on two human cases with positive complement-fixation reactions in 1953 (titres: 1/50).

*Sudan*

It would appear that ticks collected from camels (*Hyalomma dromedarii* Koch) and from bulls (*H. excavatum* Koch) exported to Egypt were found infected with *R. burnetii*.[166, 167]

*Tunisia*

Cordier and co-workers [39] examined a number of animal sera with the following results: 193 goats (3% positive); 202 sheep (55% positive); 48 cattle (15% positive); 4 pigs (all negative); and 19 horses, of which two gave titres of 1/8 and 1/16 respectively. In 1952, the WHO survey revealed 27 positive sera from 230 goats, 9 positive sera from 929 goats and sheep, and 2 positive sera from 103 human beings; 58 sera from abattoirs were also examined, and those from 2 cattle, 2 sheep, and 2 goats were found positive. In 1953 a serological survey among goats, sheep, cattle, and human beings gave the following results:

| | | | | | | |
|---|---|---|---|---|---|---|
| Goats: | 35 | significant | reactions * | out | of | 417 |
| Sheep: | 36 | ,, | ,, | ,, | ,, | 173 |
| Cattle: | 1 | ,, | ,, | ,, | ,, | 51 |
| Man: | 9 | ,, | ,, | ,, | ,, | 187 |

* Titres 1/16 - 1/32

Of the significant results in man, none was strongly positive and it was not possible in any case to recover rickettsiae from human blood. The results indicate that Q fever occurs only in enzootic form.[113]

*Union of South Africa*

Gear and co-authors [64] reported a clinical case of Q fever with a positive complement-fixation reaction in the Western Transvaal in 1950; this was in a young boy living on a farm. A second case was reported in a farm manager living in the Witwatersrand by Saner & Fehler,[142] and Ranking[136] also described a case in Ladysmith of an infected electrician working in a dairy, the consumption of raw milk being considered the possible source of infection. As a result of a prolonged investigation through 1951 and 1952 carried out by the South African Institute for Medical Research,[153, 154] it appears that Q fever shares a place with tick-bite fever as the commonest rickettsial disease in South Africa. Almost all adult South Africans appear to be immune. Most cases of infection occur in immigrants recently arrived from overseas and in South-African-born children.[155] During 1952, diagnosis was confirmed in 53 cases and suggested in another 157 cases by serological tests. One of the cases, born in South Africa, contracted the infection in the Wellington district of Cape Province. This is the first case reported from that area. In addition the infection was transferred from the patient's blood to guinea-pigs in three cases.

## America

*Argentina*

Babudieri & Parodi [7] examined the sera of 15 cases of atypical pneumonia in humans, 22 sheep sera, and 28 bovine sera by the complement-fixation test: only two cattle sera were positive at a titre of 1/32 or more.

*Brazil*

According to Travassos and co-workers, from June 1953 to March 1954, complement-fixation tests of bovine sera performed in Caixas (in the vicinity of Rio de Janeiro) gave positive results in 114 of 358 sera examined at titres of from 1/20 to 1/640.

*Canada*

Pavilanis, Lépine & Morisset [126] examined 218 human sera in Quebec Province and obtained 10 positive results (5%) at a titre of 1/32 or more.

*Martinique*

Q fever was diagnosed in 1952 in two human subjects (titres: 1/80) who had visited a cave containing bats.[120]

*Mexico*

In 1950, Sylva Goytia,[163] using the Kolmer technique, examined the sera of 889 cows; 13 proved positive (titres: 1/16 and 1/32). In the course of another survey in 1952 he observed 67 positive human sera out of 4298 examined (2%), 23 positive bovine sera out of 1217 (2%), and 19 positive sera from goats and sheep out of 386 (6%).[164] The titres were from 1/8 to 1/64, with the majority at 1/8.

*Panama*

In 1947 two indigenous cases of Q fever were notified by de Rodaniche & de Rodaniche; since then sporadic cases have been serologically confirmed.[140]

*United States of America*

The first recognized outbreak of Q fever among abattoir workers and livestock handlers occurred in 1946 in Texas.[87] In the same year the disease was reported by Shepard [146] among workmen in the Chicago meat-packing houses. In 1947 Young [182] found that Q fever occurred to a considerable extent in the neighbourhood of Los Angeles (Southern California), and

Lennette [98] later reported that 176 human cases had occurred in Central and Northern California in 1948. It was found that 3% of the bovine sera, 38% of the sheep sera, and 44% of the goat sera were positive. In 1948 Shepard [147] made a general serological survey of bovine sera in the cities of 37 States of the USA. Among 1700 cattle sera, 27 positive sera were found from dairy cows in 10 States and from beef cattle in 5 States (most titres were at 1/8, with a few instances at 1/128). The positive sera, with two exceptions, came from cows in the western two-thirds of the country. There seemed to be a higher rate in some of the West North Central States and States nearby. Altogether from 1948 to 1949 a total of 350 individuals had Q fever in Northern California.[33] In 1950, Bell, Beck & Huebner [15] examined 9804 human sera in Southern California and found that 488 (5%) were positive. The majority of positive cases were from hide, fat-rendering, and dairy plant workers. In three dairy herds in Los Angeles County, with a total of 2300 cows, Luoto & Huebner [107] found infection rates varying between 16% and 30%. For Southern California, Luoto, Winn & Huebner [108] give the imposing figure of over 15% infection of approximately 120 000 dairy cattle. Vose [177] has drawn attention to the occurrence of Q fever in Massachusetts in the Eastern United States and Doddananjayya [51] found 6 positive out of 289 human sera and 9 positive bovine sera out of 327 in eastern Washington.

*Venezuela*

A survey carried out in the region of Caracas in 1951 on 524 persons, the great majority of whom dealt with animals, revealed 21 positive sera (titres: 1/16 and over) and 36 doubtful sera (1/8).[86] Iragorry [86] examined 524 human sera in 1952 for the WHO survey and found 15 positive with titres of 1/32 or over.

## Asia

*Ceylon*

Schmid and co-authors [143] reported in 1952 that Q fever was present in human clinical cases and domestic animals. In the WHO survey in 1951 3 positive sera were detected in 586 human sera examined. In 1952, sera from 151 abattoir workers showed 4% infection and 480 other human sera were negative. Other results were: cattle, 3% positive of 100 sera; sheep, 2% positive of 102 sera; goats, 8% positive of 104 sera; buffaloes, 2% positive of 100 sera; pigs, 100 sera, all negative.

*China*

In 1951, of 37 sera from patients with atypical pneumonia in the Peking district, 5 were positive with the complement-fixation test with a titre of 1/16 or more.[31]

*India*

The WHO survey was conducted in the Bombay area and around Coonoor, Southern India. During 1949, 901 human sera were examined, of which 3 were positive at a titre of 1/40 to 1/80. The sera of 43 bandicoots were negative. In the Bombay area during 1952, 3% of 367 human sera were positive, 10% of 376 sheep sera were positive, and 142 cattle sera were negative. At Coonoor, during 1952, 7 of 46 human sera were positive at a titre of 1/16 or more, 14 of 80 cattle sera and 2 of 44 sheep sera were positive, and 12 bullock and 6 buffalo sera were negative. The survey of the prevalence of Q fever in the Nilgiris and Coimbatore districts of Madras State was continued in 1953. The sera of 99 persons, 149 cows, 75 bullocks, 6 buffaloes, 52 sheep, 2 goats, and 2 horses were tested—a total of 385. The screening was done with a 1/16 dilution of the sera. Positive results were found in 7 human beings, 19 cows, 4 bullocks, and 2 sheep—a total of 32—of which 23 with a titre of 1/16, 7 with 1/32, and 2 with 1/64.[173, 174]

*Indonesia*

To date the WHO survey has revealed 188 bovine sera positive at a titre of 1/16 and one serum positive at 1/32. Of 161 human sera, all were negative.[205]

*Iran*

Courdurier, Buck & Quesnel [42] in 1952 found 2 human subjects positive at titres of 1/80. Giroud & Yassemi,[71] also in 1952, reported that in Kermanshah in western Iran 2 bovine sera were positive with titres of 1/20 and 1/40 respectively, and 2 goat sera and one sheep serum gave titres of 1/320. Rafyi & Maghani [135] examined 267 animal sera with the complement-fixation test and the capillary agglutination test; 115 of these sera (from 2 dromedaries, 8 cows, 95 sheep, and 8 goats) were positive with titres of 1/16. In Nardine, half-way between Teheran and Meshed, out of a group of 120 human sera examined with the complement-fixation test, the sera of 2 children, aged 5 and 6 years respectively, were positive with a titre of 1/20. At Taiabad on the Afghan frontier, 4 of 22 human sera gave titres of 1/5.[61] Ansari has stated that evidence of the existence of the disease in human subjects has also been given for seven villages in the region of Sabzewar.[183]

*Iraq*

An outbreak of Q fever was notified and serologically confirmed by the complement-fixation test in three blood samples at Habbaniya, the titres being 1/16, 1/64, and 1/128 respectively.[208]

*Israel*

The first outbreak of Q fever was described in 1949 by Klopstock and co-authors.[93] Altogether 40 positive sera were detected at titres of from 1/32 to 1/2054. Investigations in Kibbutz Naan showed that 3 of 20 cattle sera were positive at titres of 1/16, 1/64, and 1/128 respectively.[190]

*Japan*

In 1951 the WHO survey detected 21 positive bovine sera (titre:1/32) out of 1106 tested.[191] Two camels out of 5 from Pakistan reacted with titres of 1/32 and another at 1/16.[92] In 1952 in Kumamoto Prefecture, Kyushu Island, the sera of 45 veterinarians and others associated with animals gave 13 positive results, and 4 positive results were obtained in 107 other sera. A total of 441 other sera were examined with negative results. Serum samples from 21 of 1550 cattle, 4 goats of 91 sheep and goats, and 3 of 5 camels gave positive reactions to the complement-fixation test at a dilution of 1/16 or over.[123]

From March 1952 to March 1953 serum samples were taken from persons who had come into contact with many sorts of livestock throughout Japan, and complement-fixation tests were carried out. Among 954 serum samples sent from 17 prefectures, 24 of 39 suspect sera showed titres of 1/8 or higher, 17 of them being from Kumamoto Prefecture, 5 from Kanagawa, and 2 from Okayama; altogether 5 persons showed titres higher than 1/32, 4 in Kumamoto and 1 in Kanagawa.[165]

*Jordan*

No cases of Q fever have so far been diagnosed in Jordan, but antibodies to *R. burnetii* have been found in sera of sheep, goats, cattle, and camels.[184]

*Lebanon*

Guinea-pig inoculations of 600 pooled milks and 600 complement-fixation tests on cattle sera were all negative.[192] But the capillary agglutination test of 55 serum samples from different dairy herds gave 6 positive with titres of 1/16 or over.[203]

*Malaya*

Bush [23] reported the first probable case of human clinical infection in Malaya in 1952; the infection was thought to be caused by the ingestion of infected milk. Five hundred human and animal sera were examined, and 2 positive complement-fixation reactions were found in human sera

and 6 in cattle and goats. In the WHO survey in 1951 2 of 247 human sera were positive, 4 cattle sera showed titres of 1/16, 2 goat sera showed titres of 1/16 and 1/32 respectively, and 3 cattle from Indonesia reacted at 1/16. In 1952, 407 human sera were examined with negative results.

*Pakistan*

Serological evidence of infection in 2 camels out of 5 at titres 1/16 and 1/32 respectively has been found.[92]

*Turkey*

The first outbreak occurred in Anatolia in 1948, and the disease was soon recognized in widely separated parts of the country, with an area of heavy infection around Ankara.[127] In 1951, during the WHO survey, Minett carried out a survey at Etlik and showed that 10 of 119 bovine sera were positive and 3 of 72 sheep sera. Golem [78] in 1951 stated that no naturally infected ticks had so far been found in the country. However, in 1952, 2 out of 50 samples of *Ornithodoros lahorensis*, a common tick, were found naturally infected with *R. burnetii*.[128] In 1952 at Ankara, the WHO survey revealed 15 positive reactions in 66 human sera; in 1953, 127 human sera were examined with 7 positive results at titres of 1/20 or more. Of 1590 pneumonia cases, 286 showed titres of 1/20 or more. Among animals, 356 sheep sera, 278 goat sera, and 362 cattle sera each gave 13% positive results; 3 out of 4 dogs were positive, and 2 out of 49 buffaloes.[199] In 1951, Golem [78] stated that Q fever had been encountered in human cases in 20 out of the 63 vilayets of Turkey.

*Union of Soviet Socialist Republics*

Q fever is reported to exist in all Central Asian oblasts and has been confirmed serologically.[32, 94]

## Europe

*Austria*

Five cases of Q fever were reported by Hintermann [85] in Carinthia in 1950-51. The disease was also reported by Lass [96] in 1952. Among 360 cases observed by him since 1948, tests were made on 100 sera, of which only 24 reacted at titres of 1/16 to 1/40 and 16 at 1/8 to 1/10. The WHO survey conducted in the Tyrol in 1951 revealed 4 out of 9 positive human sera ; one sheep serum and one goat serum were also positive.[202] In 1952, 293 human sera were examined, of which 2 were positive, and in 1953 one positive (titre : 1/32) was detected among 114 human sera.[200]

*Bulgaria*

The first two cases of human Q fever were diagnosed in 1948 in Plovdiv. Angeloff [2] indicates that Q fever exists in Bulgaria both in human beings and in animals. From 1950 to 1953, 426 human sera were examined in the Institute of Microbiology of the Academy of Science at Sofia, of which 110 were positive at titres of from 1/20 to 1/1600. Up to May 1954, out of 621 human sera examined, 202 had been found positive and the examination of 207 sheep and goat sera revealed 31 to be positive. It is noteworthy that out of 112 samples of human blood examined in Sofia during the influenza epidemics in 1952, 22 were found positive.[2]

*Cyprus*

In 1952, the WHO survey showed negative results for the sera of 12 abattoir workers and 9 laboratory employees ; 2 positive cases were found in 7 sera obtained from farm workers. Results from animal sera were positive in 13 out of 19 cattle (68%), 24 of 60 sheep (40%), and 11 of 31 goats (35%).[187]

*Czechoslovakia*

In 1952, positive reactions were obtained with 19 human sera out of 139, including 4 of 34 laboratory workers (titres : 1/20 and 1/80). Antibodies were also present in the sera of 3 out of 10 sheep kept near the laboratory.[125] The disease has occurred in epidemic outbreaks since 1953. In May of that year it broke out among 6 persons on a State farm in an isolated community; the outbreak continued until the middle of June and finally involved 15 cases in all. Two other cases occurred also in June 1953 in a village 10 km away from the first farm ; and it was shown that 35 cases had also occurred in farm workers in January and February 1953 at a time when flocks of sheep were being affected by the disease. In December 1953, another outbreak involved 13 people in a community situated over 30 km away from the other two localities. Yet another outbreak of 6 cases occurred in another area in April 1954. In both these last two outbreaks the infection was derived from cows. Again, in the spring and summer of 1954, 30 cases occurred among farm workers in another locality. All these cases were confirmed by serological evidence.[137]

*Denmark*

A total of 1700 bovine sera have so far been examined in the WHO survey with negative results.[198]

*Finland*

Two thousand bovine sera and milk samples have been examined to date in the WHO survey with negative results.[201] This is confirmed by the

investigations of Rislakki & Aminoff,[139] who have examined 1000 blood samples of cattle from 137 communes in different parts of Finland and the milk from 24 farms in southern Finland (Uusimaa) with negative results.

*France*

Schuch [145] reported on 30 cases of Q fever in the Strasbourg area in 1948, and Bertrand-Fontaine and co-authors [18] described the first case in Paris in 1949. The first case occurred in the Seine Department in 1951 [47] and Coudert & Gaté [40] have described the infection in the Lyon area where 10 human cases were diagnosed. In 1952 two outbreaks of Q fever took place in the Department of Allier affecting 31 people out of 53 in the post office of Moulins and 12 out of 55 in the post office of Montluçon. All were due to *R. burnetii* present in air-dust of postal bags. Serological tests on sheep and pigs were clearly positive (titres : 1/20 to 1/80) and doubtful with cattle and dogs (titre : 1/10).[70] In the Departments of Ardèche and Vaucluse and in the Paris region, 26 of 137 sera tested gave titres of from 1/10 to 1/160.[76] During the period 1949-52, 1838 human sera from all parts of France were tested ; 48 of these showed positive reactions for Q fever with titres of from 1/40 to 1/60.[70] The WHO survey was conducted around the Montpellier region in southern France with the following results in 1952 : 130 sheep sera, 7 positive ; 10 goat sera, all negative ; 24 human sera, one positive (titre : 1/16). In 1953, 31 suspected cases were examined, of which 4 were found positive. Since the beginning of the survey, the Centre de Recherches sur la Fièvre ondulante at Montpellier has examined a total of 388 sheep sera originating from 27 different herds, of which 22 sera from 3 herds were positive.[30]

*Germany, Federal Republic*

The available information seems to indicate that Q fever is widespread throughout western Germany, but particularly in the region from Hamburg down to the Austrian border and following the western border. Epidemics of Q fever in man occurred in the Tübingen district of south-western Germany [84] in 1948, in the neighbourhood of Stuttgart from November 1948 until May 1949,[60] again in Stuttgart from October 1949 to January 1950, involving 100 people,[101] and in the Württemberg area in the spring of 1950.[26, 114] However, Germer & Glockner [66] state that since 1951 no outbreak of Q fever has taken place in Württemberg-Hohenzollern but that sporadic cases have been observed. They consider that the disease is endemic. Serological tests of 77 men who had previously suffered from the infection (from 1947 to 1951) were positive in 53 cases (69%) with titres of from 1/16 to 1/512. In formerly epidemic places, 37 of 160 sera (19%) from human beings, cattle, and sheep gave positive reactions ; and in non-epidemic places, 139 of 1321 sera (11%) were positive (titres : 1/16 to 1/64). Altogether, 176 of

1558 sera (12%) were positive. At the beginning of 1950 an important epidemic broke out in the Eifel region (Ahrtal) involving 400 people, of which 90% were men between the ages of 20 and 60 years. The infection originated in flocks of migranting sheep.[169] In southern Baden 200 human cases occurred in February 1950 among 1116 inhabitants of adjoining villages near Freiburg. The outbreak was apparently associated with large flocks of sheep.[144] Another outbreak was notified in two villages and in a country estate in northern Baden in 1951 : 10% of 147 people and 3% of 3677 people, respectively, were involved, while half the estate population (30 cases) was affected. Moreover, a few cows and goats were found infected.[19, 83] In northern Hessen in 1952, 11 people were confirmed positive by serological test.[122] In February 1950, 90 cases were reported near Heidelberg.[95] Eymer [53] described an outbreak in Munich in 1951 among six persons living in the same house. In 1951 at the Tropeninstitut of Hamburg, Lippelt [102] examined 168 human cases of Q fever, 56 of which were infections occurring in his own laboratory (titres : 1/8 to 1/2000).

*Greece*

Caminopetros [27] showed that Q fever occurred in Greece in 1946. He later succeeded in isolating *R. burnetii* from the milk of sheep and goats.

*Hungary*

Farkas, Gerö & Takatsy [54] examined the sera of 307 abattoir workers in Budapest in 1950 and found 10% of the samples positive.

*Iceland*

In 1951 a brief survey was carried out by Sigurdsson [149] on the sera of human beings and sheep, with negative results. The number of tests was too small to permit of any definite conclusions.

*Ireland*

Investigations were carried out by Meenan [197] during the WHO survey and under the auspices of the Medical Research Council of Ireland. During the period 1950-51, 444 human sera from different sources and 280 cattle sera were examined, but none was positive. During 1953, 137 sera received from the National Blood Transfusion Association, consisting of blood collected at different meat-processing plants throughout the country, proved negative.

*Italy*

Q fever was first detected among the civilian population by Terzani & Rossi [170] in Florence early in 1948. Further cases were afterwards reported

in various parts of the country, and in 1951 Babudieri,[3] in a comprehensive review, showed that the disease had become widespread throughout Italy. Epidemics were recorded in the Brescia area in the north, at Sestri Levante in the north-west, in the Florence area, extensively along the east coast from Montecchio to Teramo, in the south-west at Terracine and Caserta, in the extreme south-west at Grottaglie, and in Palermo in Sicily. Isolated outbreaks were also reported in Sardinia and in the Aeolian Islands at Lipari and Leni.[103] During the winter of 1950-51, an outbreak occurred in the small town of San Lorenzo, in the province of Pesaro; 242 cases were reported, of which 44 were confirmed serologically.[6] Monaci & Scaglioni,[119] examining the sera of 482 individuals working in 8 tanneries in northern Italy, noted that 38 (8%) were positive; and Angela and co-workers,[1] in 1952, found that 26% of the workers in the Turin abattoir gave positive reactions to the complement-fixation test.

From a conference on the epidemiology of Q fever in Italy held in 1952,[44] it appears that the annual aggregate figure for human cases of the disease in the country is about 10 000. The extent of human infection made it necessary for serious investigations to be carried out among animals. Davoli & Signorini [45] examined 1052 animals in the Florence abattoir in 1950 and found positive reactions in 6% of cattle, 33% of sheep, 20% of goats, 1% of pigs, and 10% of horses. Monaci & Nuvolone [118] tested 1078 blood samples from animals at the Milan abattoir in 1953; 4% of cattle, 42% of sheep, 9% of goats, and 1% of pigs gave positive complement-fixation reactions. In the city of Bologna, 4 of 19 dogs tested were positive, with titres of from 1/4 to 1/32. In Imola, 4 of 16 dogs tested were positive.[9] Badiali & Zoli [10] first reported the presence of Q fever in the Ravenna area in 1953. The sera of 113 abattoir workers and street cleaners were examined and 14% were found to contain antibodies. In 1953, a survey was made by Davoli & Signorini [45] of a large number of animal sera in the Florentine region in Tuscany; these sera were obtained from farms where cases of Q fever in human subjects had occurred, while control sera were obtained from farms where no infection was known to exist. From the infected premises it was found that 14% of 56 bovine sera were positive and 41% of sera from 226 sheep and goats; while from the presumably non-infected premises, 6% of 403 bovine sera were positive and 20% of sera from 547 sheep and goats. In addition, 1% of 224 pigs, 11% of 37 dogs, and 10% of 32 horses were found to react positively to the complement-fixation test as well as 2 of 16 rabbits.

In Sicily, Mirri [116] showed that the infection was widely distributed among animals, and Buttista [24] and Gallo [62] both demonstrated the presence of antibodies in about 30%-40% of cow, sheep, goat, and dog sera examined. However, serum tests in pigeons, rabbits, mice, chickens, sparrows, and ravens gave negative results, although 2 of 40 rats had titres of 1/16.

In May 1954 an epidemic outbreak involving 112 people occurred in Dommegge di Cadore in the province of Belluno, where no cases had previously been notified. Serological tests showed that a herd of sheep was responsible.[48]

The information available today indicates that Q fever has spread practically all over Italy.

*Netherlands*

In 1951, the WHO survey covered 2152 human sera with negative results in all cases. In 1951, one positive bovine serum (titre: 1/16) was detected out of 486 examined.[a] Jansen[89] noted that in 62 cases of abortion and sterility in cattle free from brucellosis the complement-fixation test for Q fever was negative.

Up to 31 December 1952, 2677 complement-fixation tests had been carried out on 1902 human sera, 488 bovine sera, and 287 guinea-pig sera. According to Wolff[209] all tests gave negative results. At the end of 1953 the same author examined a total of 3229 sera (2411 from humans, 524 from cattle, and the rest from guinea-pigs and rabbits). None was positive.[210] Examination of more than 3000 blood samples in 1954 also gave negative results.[181]

*Norway*

In 1952 the WHO survey on bovine sera yielded negative results.[188]

*Poland*

Up to the end of 1952, 1022 human sera had been tested with negative results in the WHO survey. In 1953 Parnas and co-workers[124] examined 421 workers in the abattoirs of Lublin, 79 in the abattoirs of Warsaw, and 158 in those of Katowice. All of them, with the exception of one (positive at 1/16) were found negative. Six others seemed to give a positive reaction, but the reaction was not clear enough.

*Portugal*

The disease has been shown by Fonseca et al.[57] to exist throughout Portugal. The tick *Hyalomma rufipes* is considered to be essential for the maintenance of infection among the animal population. Positive reactions were found in 23% of the cows, 20% of the goats, and 27% of the sheep which were tested. From 1947 to 1951, 48 human cases were diagnosed in the country.[59] Out of the 14 employees of a cattle breeder, two had positive complement-fixation reactions (titres: 1/20 and 1/40).[56]

[a] Re-examination of the herd of origin of this cow gave negative results; in view of subsequent negative results on large numbers of serum specimens this positive reaction can be discounted.

*Romania*

The first 5 human cases of Q fever in Romania were identified in 1947 by Combiesco and co-workers.[35] In 1948 Combiesco et al.[36] reported upon 3 laboratory infections among workers investigating a new rickettsial infection which eventually proved to be Q fever. The same authors [37] in 1949 notified the discovery of 9 other cases among the personnel of the Dr I. Cantacuzino Institute of Sera and Vaccines in Bucharest (titres: 1/64 to 1/128).

*San Marino*

In 1950, Valli [172] reported on his investigation of an outbreak of Q fever among 22 persons; sheep and cattle were considered the main sources of infection, but it was thought that pigeons might also be responsible.

*Spain*

In 1949, Perez Gallardo and co-workers [130] reported on the isolation of *R. burnetii* from three species of ticks, two species being collected from calves in Seville and the third from a dormouse in Madrid. The first human case of Q fever in Spain was described by de Prada, Gay & Llorente [134] in 1950. Clavero and co-authors [34] mentioned 4 cases of Q fever contracted in 1950 by laboratory workers and serologically confirmed. Perez Gallardo and co-authors [131] showed by complement-fixation tests that Q fever occurs in cattle and wild rabbits in many provinces of Spain.

*Sweden*

During 1951 and 1952, 1107 bovine sera were examined in the course of the WHO survey with negative results.[189]

*Switzerland*

The first cases of Q fever in Switzerland were identified by Gsell [79] in 1947. In 1948 an outbreak took place in Bremgarten (Canton Aargau) in a furniture-manufacturing house, involving 59 of the 107 men employed.[80] Q fever is stated by Wiesmann [179] to be endemic throughout Switzerland and the adjacent territories. Between 1947 and 1951, 1080 human infections were diagnosed at St. Gallen and a large number were also found in Zürich ; cattle, sheep, and goats and their products were considered responsible. In the Engadine valley in 1948 an outbreak involved 19 people in a factory in Chur,[178] and in 1950 another outbreak was accompanied by infectious abortion in sheep.[179] Baumgartner [13] also reported the detection of Q-fever antibodies in the sera of 30 persons clinically affected in Dielsdorf. Burgdorfer et al.[22] reported 3 human cases in eastern Switzerland in 1951. In the WHO survey in 1952, 20 human sera were found to be negative and in

1953, 50 cases of atypical pneumonia were examined and 4 were positive in the complement-fixation test.[194]

*Union of Soviet Socialist Republics*

Q fever is reported to exist in various parts of the Moscow oblast and has been serologically confirmed.[32, 94]

*United Kingdom of Great Britain and Northern Ireland*

Stoker [156] first reported on Q fever in Great Britain in 1949, and *R. burnetii* was isolated from a patient in a London hospital during the same year.[109] Sporadic outbreaks have also been reported in various areas, particularly in Kent and Devon.[112] In 1952, Slavin [151] reported on the results of a country-wide survey among cattle in Great Britain. It was shown that 2% of cattle had antibodies to *R. burnetii.* Blood samples from guinea-pigs inoculated with milk for the detection of tubercle bacilli showed Q-fever infection in 7% of farms in England, 2% of those in Wales, and 1% of those in Scotland. In 1953 Marmion et al.[111] analysed the prevalence of infection in 69 sporadic cases in humans and cows, including those already mentioned.[109, 110, 156] In addition to 32 cases in Kent and 8 in Devon, 10 occurred in the London area, and a few were located over the Midlands and North and South Wales. The frequency of infection in cattle in 1031 herds in the Eastern counties, Devon, and Kent, was determined by the examination of milk samples, and it was found that 3% of herds in Devon and 8% of those in Kent were infected, whereas only 1% of herds were infected in the Eastern counties. In an explosive outbreak in southern Kent, Stoker & Thompson [159] found that 13 of 15 people living on a dairy farm had contracted the disease ; there were 3 other cases among 9 visitors to the farm. Epidemiological findings and laboratory tests, including pooled milk tested for *R. burnetii* by guinea-pig inoculation and the complement-fixation test on sera (titre : 1/8) did not suggest that the cows were the reservoir of infection ; but serological tests of sheep have shown evidence of infection with *R. burnetii* (titres: 1/40 to 1/80) and indicate that sheep are responsible for the infection of man in this area.[112] Although there was some evidence pointing to pigs as a source of infection, it was not possible to confirm this in the laboratory.

With regard to south-east England, Stoker [157] states that the rate of infection in man is 2.96% and that " after allowance is made for the selection of blood donors by age, it is calculated that this represents some 30 000 infections in Kent alone ".

*Yugoslavia*

The first outbreak of Q fever was reported by Simovic and co-authors [150] in Bosnia in 1950. In August 1950 an acute outbreak involved 50 persons

out of 400 in the village of Sokol in Bosnia.[121] Cvjetanovic et al.[43] reported on an epidemic in Ogulin, Croatia, in April 1952 ; 26 cases proved serologically positive. At the Institute of Hygiene of Zagreb 28 sheep out of 521 examined were positive (titres: 1/16 to 1/64), but of 53 human cases examined from 1950 to 1952 all were positive with titres above 1/20.[207] During the WHO survey the sera of 130 sheep were examined in 1951, of which 5 were positive. Up to the end of 1952, all attempts, in connexion with clinical cases of Q fever, to isolate *R. burnettii* from the milk of cows and sheep proved unsuccessful.[206] During 1953, 184 human sera were examined, of which 1.65 % were positive at titres above 1/10 ; 28 positive sera were detected in 521 sheep. The results of serological tests carried out by the Virology Department of the Institute of Hygiene in Belgrade from January 1952 to March 1953 indicate that 74 patients were confirmed as cases of Q fever with titres of from 1/8 to 1/1024.[171] Serological tests on various animals showed 54 sheep to be positive out of 323 examined and only 1 cow out of 165.

## Oceania

### *Australia*

Since Derrick [49] published his report in 1937 on the discovery of a new disease entity—subsequently known as Q fever—among meat factory workers in Queensland, other authors have reported on the condition in other parts of Australia. Infection among abattoir workers in South Australia was first detected in 1947,[160] this being the first State other than Queensland in which the disease was observed. Stokes [161] isolated *R. burnetii* from 4 clinical cases, and 5 others were positive in the complement-fixation test. In August and September 1952, Carley & Pope [29] in Queensland isolated for the first time two strains of *R. burnetii* from naturally infected ticks, *Ixodes holocyclicus*. According to Derrick et al.,[50] 3 serologically demonstrated cases of Q fever occurred in 1952 in the Mackay district of Queensland. Beech, Howes, & Miles [14] state that observations made on the sera of aborigines in the Northern Territory strongly suggest that there is an endemic form of Q fever in the Hermannsburg area, where 3 of 23 sera tested were positive at titres of 1/20 to 1/40. Six other positive sera came from two other areas, one to the east of Katherine and the other to the west of Alice Springs. Stokes [161] indicates that since Q fever was recorded in South Australia for the first time, 42 sera giving positive findings have been discovered, as well as 6 laboratory infections. Cattle have been shown to be the chief source of infection. All patients whose sera were tested 12-16 months after their illness had complement-fixing antibodies varying in titre from 1/8 to 1/2048 ; 29 months after infection, one man still had a titre of 1/512. Negative results were obtained in tests on 793 bovine sera from the northern areas of South Australia and 641 sera of dairy cattle from central and southern districts.

The WHO survey in New South Wales began in 1951, and by the end of 1953, 700 cattle sera had been examined with negative results, except for one serum with a titre of only 1/16. Similarly, 700 cattle sera have been tested in the Northern Territory, again with negative results. More recent information, however, indicates that the presence of Q fever has been serologically demonstrated in Victoria and New South Wales.[196] A survey of abattoir workers in Perth and Wyndham in Western Australia conducted by Drummond & Thorburn [52] did not reveal evidence of past infection with *R. burnetii*. An outbreak of Q fever recently took place in the abattoirs of Adelaide, South Australia.[196]

*New Zealand*

There is no evidence of the existence of Q fever in New Zealand ; the import of livestock is subject to the presentation of a certificate of freedom from serological evidence of infection.[141]

## DISCUSSION

It appears conclusively established from the foregoing that Q fever is totally absent from certain countries, particularly Denmark, Finland, Ireland, the Netherlands, Norway, and Sweden. In all these countries large numbers of human and animal serum specimens were screened with negative results. There is somewhat less certainty as to the absence of the disease in Iceland and Poland. Only relatively few examinations were performed in Iceland, and no conclusions can therefore be drawn. The negative results in Poland were obtained with serum specimens taken from abattoir workers in Warsaw, Lublin, and Katowice and a few doubtful reactions were obtained, but these do not appear to have been significant. It should be noted, however, that the surrounding countries (Germany, Czechoslovakia, and the USSR) harbour the infection.

In seeking an explanation for the apparent absence of Q fever from Ireland, the Netherlands, and the Scandinavian countries, one common factor emerges : the import of cattle, sheep, and goats into all these countries is relatively slight. The role of ticks carried by these ruminants has yet to be clarified. We plan to investigate these points further, and it is expected that a report, including the results of further serological surveys in these countries, will be made at a later date.

## RÉSUMÉ

A la suite d'une résolution de la Troisième Assemblée Mondiale de la Santé, en 1950, l'OMS a encouragé l'exécution d'enquêtes épidémiologiques visant à établir la répartition géographique de la fièvre Q dans le monde. Les résultats de l'enquête effectuée dans 33 pays, complétée par des études individuelles parues au cours des dernières années, sont résumés dans cet article.

La prévalence de la maladie a été déterminée au moyen de tests de laboratoire, qui seuls permettent de déceler l'infection, les symptômes chez l'homme et les animaux n'étant pas spécifiques ou l'infection clinique étant inapparente. Les tests recommandés étaient la fixation du complément ainsi qu'un essai d'isolement de *Rickettsia burneti* sur le cobaye. Il avait été conseillé de procéder aux trois épreuves suivantes pour dépister la présence éventuelle de la fièvre Q dans une localité: 1) examen du sérum du personnel des fondoirs et des abattoirs; 2) inoculation au cobaye d'échantillons de lait provenant du mélange des laits de 100-200 animaux, avec épreuve de fixation du complément avant et après inoculation; 3) test de fixation du complément appliqué à des animaux choisis au hasard. L'OMS a fourni les antigènes — au début de l'enquête une souche commerciale Henzerling, à la fin une souche Nine Mile — et un sérum positif de cobaye pour titrage de l'antigène.

Après avoir donné des résultats détaillés sur les résultats de l'enquête dans 63 pays et territoires, les auteurs concluent que la fièvre Q est certainement présente dans 51 d'entre eux. Elle n'existe pas dans un certain nombre de pays, en particulier le Danemark, la Finlande, l'Irlande, les Pays-Bas, la Norvège et la Suède, qui tous n'importent que très peu de bétail. Le rôle des tiques dont les animaux domestiques sont porteurs est encore à élucider. Les recherches continuent.

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