

13 facts to increase awareness to Q FEVER among general practitioners

Q Fever is a zoonotic infectious disease caused by *Coxiella burnetii*. All mammal species are permissive to the bacteria, but farmed ruminants (cattle, buffaloes, sheep and goats) are the main reservoirs for human infection, as detailed in the Terrestrial Animal Health Code of the World Organisation of Animal Health (<https://www.woah.org/en/disease/q-fever/>).

In animals, *C. burnetii* causes mostly subclinical infection, although it can also cause abortion series and other reproductive disorders. Infected females excrete a high bacterial load at birth/abortion. The bacteria are exceptionally resistant in the environment and can be aerosolized with farm dust particles. Inhalation of bacteria suspended in the air is the main route of infection. Consequently, people with direct animal exposure, like farmers, veterinarians or slaughterhouse personnel, are professionally at risk, but airborne transmission over several kilometers has also been described and linked to large scale epidemics.

Subclinical or asymptomatic infection is the most common presentation in humans. However, two clinical forms of Q Fever have been recognized in humans: acute

and chronic (the latter is also known as a latent or a persistent form). Originally, Q Fever has been named after an outbreak of the acute form of the infection, among Australian patients and was named Q as 'query' fever because the etiology remained unknown at the time. The chronic form has been recognized more recently. Pregnant women are supposedly at risk of miscarriage; immunocompromised people (including elderly) and persons with valvular defects may also be considered at risk of developing a clinical form of infection.

In most European countries, Q Fever in humans is a notifiable disease. The disease has been described all over the world, except for New Zealand, both in animals and humans.



ABOUT THE PATHOGEN

Coxiella burnetii is an intracellular Gram-negative bacterium, characterized by a survival mechanism (pseudospore formation) which enables it to resist durably under environmental conditions such as desiccation for several months, disinfection such as 0.5% sodium hypochlorite or UV radiation^[1]. It can survive several days in moist conditions (at least 7 days in water or milk at room temperature). It can withstand exposure to 60°C for 30 minutes, but can be inactivated by pasteurisation^[2]. All mammal species are considered receptive to *C. burnetii*, although ruminant species are considered its main reservoir. People professionally exposed to cattle or small ruminants (veterinarians, farmers, slaughterhouse personnel) are at risk of infection.



PURPOSE OF THIS FACTSHEET

To present in 13 points the relevant clinical and epidemiological data on human cases of Q Fever in order to raise awareness to *Coxiella burnetii* infection among GPs in Europe, with a focus on higher-risk groups.



ANNA PSAROULAKI

HEAD, ZOOSES AND GEOGRAPHICAL MEDICINE UNIT, HEAD, MICROBIOLOGY OF FOOD, WATER AND ENVIRONMENT UNIT, DEPARTMENT OF CLINICAL MICROBIOLOGY & MICROBIAL PATHOGENESIS, SCHOOL OF MEDICINE, UNIVERSITY OF CRETE AT HERAKLION.

“The diagnosis of Q Fever is difficult since the isolation of the pathogen is rarely performed due to the need of skilled personnel and the adapted facilities. The diagnosis largely relies on the detection of antibodies against the pathogen, which may sometimes be misleading due to cross-reactions. Q Fever can cause acute or chronic infection in humans. The absence, in most cases, of clear-cut symptoms and delayed diagnosis (associated to a limited awareness of the disease among medical staff) may lead to a large under-reporting. Due to the above, there is a gap in Q Fever reporting and the actual numbers of human cases, even though Q Fever in humans is a notifiable disease in Greece.”



DANIEL CIFO ARCOS

MEDICAL INTERN RESIDENT OF PUBLIC HEALTH AT THE SPANISH PUBLIC HEALTH SCHOOL AT INSTITUTO DE SALUD CARLOS III (MADRID) AND PhD CANDIDATE. HE IS PART OF THE SPANISH NATIONAL CENTRE OF EPIDEMIOLOGY IN HUMAN Q FEVER SURVEILLANCE.

“Diagnosing Q Fever in humans is challenging due to its often non-specific presentation. Understanding the disease’s epidemiology is essential to increase clinical suspicion – especially in high-risk groups such as pregnant women – and to help prevent underdiagnosis and underreporting.”



VITTORIO SAMBRI

DEPARTMENT OF MEDICAL AND SURGICAL SCIENCES, BOLOGNA UNIVERSITY, COORDINATOR OF THE COVID-19 LABORATORY NETWORK FOR EMILIA-ROMAGNA, HEAD OF THE DEPARTMENT OF LABORATORY AND TRANSFUSIONAL MEDICINE AND OF THE OPERATIVE MICROBIOLOGY UNIT AT AZIENDA UNITÀ SANITARIA LOCALE DELLA ROMAGNA.

“Q Fever remains one of the zoonotic infectious diseases with a very low detection rate in Italy, despite clear evidence of the persistent circulation of Coxiella burnetii in livestock. Given the potential prognostic implications of Q Fever when not properly diagnosed and treated, it is essential to raise awareness of this neglected disease among physicians and to promote the implementation of appropriate laboratory diagnostic methods. These include serological testing for phase I and phase II antibodies with adequate sensitivity and specificity, as well as the use of NGS sequencing systems for pathogen identification in biopsy samples, particularly in cases of organ-localized infection.”

ABOUT RISK AND EXPOSURE

There is a difference between risk and exposure, as well as there is a difference between hazard and risk. Coxiella burnetii is a biological hazard, but the risk of infection is the result of a combination between the level of exposure (bacterial load) and the person’s receptivity to the infection.

Regarding infection by C. burnetii:

- Laboratory tests, including a complete blood count, inflammatory parameters and liver function tests, to which may be added a circulating anticoagulant test and, if positive, a test for antiphospholipid markers.
- Antibodies directed against phase I (persistent infection) or phase II (acute infection) antigens of *C. burnetii* can be detected by the appropriate serological kits, with IgM and IgG titration for each antigenic phase. Ideally, seroconversion should occur within 2 to 4 weeks after the primary infection.
- Molecular biology can help in the diagnosis of Q Fever, especially in the case of focal persistent forms. The genome of *Coxiella burnetii* can be detected by PCR in plasma or other tissues (heart valves, vascular tissue, bone biopsies, etc.).

1. WHAT ARE THE MAIN CHARACTERISTICS OF *COXIELLA BURNETII*, THE CAUSATIVE AGENT OF Q FEVER?

Coxiella burnetii is a strictly intracellular (it grows in phagolysosomes) Gram-negative bacterium. It could be cultivated in cellular-free systems (agar plates) with a complicated procedure and only for research purpose. In the environment, it adopts an endospore-like form that shows high resistance to disinfectants and even to high temperature. *C. burnetii* is distantly related to *Legionella* spp.

Due to its environmental resistance and high infectivity – a minimal quantity of aerosol-associated bacteria may cause Q Fever by inhalation – *C. burnetii* is classified as a potential bioterrorism agent [3].

2. WHAT ARE THE ANIMAL ASPECTS OF Q FEVER?

Ruminants, wild and domestic, are the main reservoir of *C. burnetii* for human infection. Although a wide array of captive as well as non-captive wildlife species, as well as pets have been occasionally found seropositive [4] or positive by PCR (like ticks collected from animals), these species are not likely to play a relevant role in the epidemiology of the disease [5] (tickborne human infections have not been described to date) [6].

Domestic ruminants have been the focus of most animal studies, particularly small ruminants. *C. burnetii* seroprevalences ranging from 15% to 60% have been reported in goat herds from France, Italy, the Netherlands or Spain [7,8,9,10]. In sheep, even higher seroprevalences have been observed in various EU countries like Spain or Italy [11,12]. Data on seroprevalence in cattle are also available for countries like France, Spain, Germany, Denmark or Ireland [13,14,15,16]. A recent study conducted in France (Brittany) shows that cattle farms can be a significant source of human exposure linked to the professional activity: 89% of veterinarians and 56% of farmers were found to be seropositive [17] (keep in mind that the majority of infections remain subclinical, see point 3).

In domestic ruminants, similarly to humans, infection remains mostly subclinical. However, acute infection may cause abortion (in several countries such as France and Belgium, *C. burnetii* is included in the abortifacient pathogens screened for in case of abortion), and in some herds, a series of abortions. It also has an impact on other reproductive disorders (reviewed here [18]).

Although infected animals excrete the bacterium by the milk, fecal and vaginal routes, the bacterial loads associated with birth/abortion are the most important source of environmental contamination, and are usually associated with clustered human cases of Q Fever. Other contaminating events are the spray application of untreated manure (although a retrospective Dutch case-control study found no significant increase of human Q Fever prevalence in zones where manure from infected farms had been applied [19]) and the slaughtering process. In France, the geographic distribution of human cases and of small ruminant herds strongly overlap, a situation also observed in Spain or the Netherlands [20,21,22].

The largest recorded Q Fever outbreak in the world occurred in the Netherlands from 2007 onwards, with ultimately almost 3,500 human patients notified in three years. Dairy goats farming was considered a plausible cause.

3. WHAT ARE THE CLINICAL FORMS OF Q FEVER IN HUMANS?

In humans, Q Fever is most often asymptomatic or mild, most frequently a flu-like syndrome, but it can lead to complications and evolve into persistent focal forms (alternatively known as chronic Q Fever) in around 1-5% of cases [23]. In pregnant women, the impact of infection could cause miscarriage (see below). Several European countries consider Q Fever as an occupational disease, and human surveillance is highly heterogeneous (some countries consider it a notifiable disease, others don't).

After primary infection (airborne) and an incubation period of two to three weeks, around 60% of those infected remain asymptomatic. The other 40% develop acute, symptomatic Q Fever, which can present with several forms [26]:

- 1. Flu-like syndrome:** The most common manifestation, with high fever, muscle and joint pain, and often severe headache. **Due to the low specificity of these signs, many of these cases go undiagnosed.**

2. **Febrile hepatitis:** Fever and impairment of liver function, with elevated liver enzymes. However, classic clinical hepatitis manifestations such as hepatomegaly or jaundice are considered rare.
3. **Atypical pneumonia:** manifested by a febrile dry cough with or without dyspnea, or chest pain.
4. **Other rarer clinical forms:** acute pericarditis, myocarditis or endocarditis, pericarditis, lymphadenitis, meningitis or meningoencephalitis.

About 5% of symptomatic patients will be hospitalized, mainly for prolonged fever associated with hepatitis or pneumonia. Most infected people recover within a few days to a few weeks, as with the flu. However, a post-infectious fatigue syndrome may occur and last for several weeks – or even months [24]. The disease is estimated to cause around 300 hospitalizations a year in Spain or 200 in France, which, compared to surveillance data, may be a sign of underreporting and underdiagnosis [25].

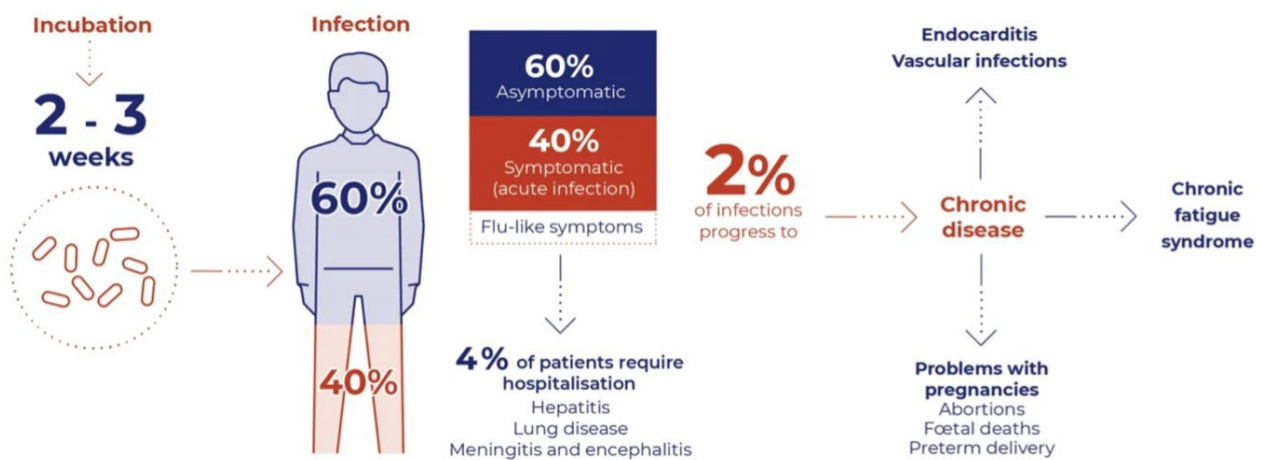


Fig 1. Main forms of Q Fever (*Coxiella burnetii* infection) in humans (from Dupuis et al., 1985).

Symptomatic Q Fever can lead to complications during pregnancy: miscarriage, in utero fetal death, prematurity [26].

After primary infection, independently of its clinical expression (including subclinical cases), a chronic infection may occur (1-5% of the cases). Its main clinical manifestation is chronic endocarditis, but also vascular infection, rare osteoarticular infection and, even more rarely, lymphadenitis are also described. Valvular prosthesis and vascular grafts are important risk factors. Pregnancy is also considered a risk factor for chronic infection [26]. In the Netherlands, over 200 chronic cases have been identified further to the 2007-2009 outbreak. Although the majority of these cases were identified within 2 years of the initial infection, that interval was longer than 2 years for 22% of the patients (with 9.2 years in one case). Chronic Q Fever-related mortality occurred in 30% of these patients and most of them had underlying conditions [27].

Confirmed human cases of Q Fever are compiled by the E-CDC and are freely available online in the Surveillance Atlas of Infectious Diseases (<https://atlas.ecdc.europa.eu/public/index.aspx>). A total of 805 confirmed human cases were reported in 2023 in the UE27, with Spain, Romania, France, Germany and Hungary being the most affected countries [28]. A recent systematic review reported a total of 81 human Q Fever outbreaks published in the literature globally, with 10 involving more than 160 cases. Most outbreaks occurred in settings not directly related to livestock (e.g. residential areas or facilities), in relation with the possible airborne transport of the pathogen over some distance (time spent outside has also been found as a risk factor). However, 17 were associated with traditional risk practices (ruminant farms, slaughterhouse and veterinary schools) [29]. A recent survey performed in eight European countries, interrogating general practitioners whose practice is exclusively located in rural areas showed there is a wide diversity of professional experience with Q Fever [30]. In Spain and France, 60 to 70% of GPs mention encountering such cases within the last 5 years, while in Poland and Italy, 70% of respondents mention they have no previous experience with the disease. Take together, these elements point at a significant level of under-detection of the infection in humans.

4. ARE PREGNANT WOMEN AT RISK?

Based on observational data ^[29] (including Q Fever seroprevalence studies in pregnant women), there is no convincing evidence that asymptomatic Q Fever-positive pregnant women are at increased risk of adverse pregnancy outcomes. So far, no prospective study has shown the usefulness of treating *C. burnetii* seropositive, asymptomatic pregnant women. For this reason, screening for Q Fever is not recommended during pregnancy.

The potential impact of *C. burnetii* on pregnancy is a source of concern for female veterinarians and farmers. Indeed, miscarriages have been described following acute Q Fever occurring during pregnancy and treated for the usual duration (14 days). In these cases, placentitis linked to *C. burnetii* has been demonstrated ^[31,32]. In the literature, it is recommended to extend the duration of antibiotic therapy with co-trimoxazole (see below) to prevent adverse pregnancy outcomes. However, since co-trimoxazole is a folic acid antagonist, folate supplementation is required if the treatment occurs over the first trimester of pregnancy ^[26]. Finally, retrospective data from the large outbreak in the Netherlands ^[33] found no association between residing in a Q Fever-affected area and both preterm delivery and perinatal mortality. There was a weak association with the pregnancy outcome: small for gestational age.

It is recommended that chronic forms of Q Fever are systematically investigated in women who have had acute symptomatic Q Fever during pregnancy. Treatment consists of the administration of co-trimoxazole during pregnancy and a change after delivery to doxycycline and hydroxychloroquine. As a precautionary measure, breastfeeding is not recommended in such cases, even though the lactogenic transmission has never been documented ^[26].

5. ARE THERE GUIDELINES FOR WOMEN WITH PROFESSIONAL EXPOSURE TO RESERVOIR ANIMALS?

A woman who is seropositive before starting pregnancy may be considered as protected, since natural (post-infectious) immunity against Q Fever is well described in humans, but a complete level of certainty about sterilising immunity provided by *anti-C. burnetii* antibodies is still missing. For women of childbearing age who have been treated for acute Q Fever, some experts recommend delaying the onset of pregnancy until at least one month after recovery from the infection.

6. SHOULD A WOMAN SEROPOSITIVE TO Q FEVER BEFORE PREGNANCY BE CONSIDERED AS PROTECTED?

This may relate to farming and veterinary practice, and also to wildlife exposure (e.g. hunting and conservation projects).

Whenever possible, pregnant women that are professionally exposed to domestic ruminants are advised to stay away from herds. This may limit the risk without completely eliminating it since airborne transmission is plausible even at a distance. It is also difficult to be complied with by livestock farmers or veterinarians in rural areas. The French High Council for Public Health recommends that pregnant women avoid high-risk events such as farrowing taking place on farms ^[34]. During pregnancy, women can wear a well-fitted FFP2 mask or visor if exposed to abortion products; this proves however nearly impossible to comply with in daily professional life.

7. WHEN TO SUSPECT Q FEVER IN A PATIENT?

Given the often-non-specific clinical presentation of Q Fever, a thorough assessment of potential exposure based on known risk factors is essential. Outbreaks in Europe have been associated with slaughterhouses, infected sheep or goat farms, untreated manure application from infected farms and transhumance of livestock ^[25,35]. Additional at-risk environments include areas surrounding farms, even where no direct livestock contact occurs, such as non-agricultural industries, and outdoor activities close to potential sources of infection, including grazing areas. The local epidemiological context and the patient's occupational history should also be considered. In this regard, questioning the patient about visiting or owning an open farm is relevant.

Q Fever should be considered in cases of prolonged fever with altered general condition (severe fatigue, severe headache, etc.) and hepatic cytolysis, or in case of fever accompanied by a dry cough – with or without dyspnea, or even chest pain.

Individuals with heart valve disease, aneurysms, vascular prosthesis, severe immunodepression (ongoing chemotherapy) and pregnant women are at risk of developing a severe chronic form of Q Fever ^[26]. However, such cases are too rare in these at-risk populations to justify implementing systematic screening for Q Fever.

8. IS THERE A CONSENSUAL DIAGNOSTIC FRAMEWORK FOR Q FEVER IN HUMANS?

The general practitioner or specialist can confirm a suspicion of Q Fever based on ^[26]:

1. Laboratory tests, including a complete blood count, inflammatory parameters and liver function tests.
2. Antibodies directed against phase I (chronic infection) or phase II (acute infection) antigens of *C. burnetii* can be detected by the appropriate serological kits, with IgM and IgG titration for each antigenic phase. Ideally, seroconversion should occur within 2 to 4 weeks after the primary infection.
3. Molecular biology can help in the diagnosis of Q Fever, especially in chronic forms. The genome of *C. burnetii* can be detected by PCR in plasma or other tissues (heart valves, vascular tissue, bone biopsies, etc.). It must be underlined that under the current regulation for in vitro diagnostics (IVDR), a limited number of tests are certified for this purpose.
4. To diagnose chronic forms, additional tests are required: cardiac echography (transthoracic and transoesophageal), PET scan, etc.

A diagnosis of Q Fever in an at-risk individual should be followed up with specialized care.

9. ARE THERE GUIDELINES FOR PATIENT TREATMENT?

Recommended antibiotic therapy in cases of symptomatic, probable or confirmed Q Fever is doxycycline (200 mg per day) for 2 to 3 weeks. In case of doxycycline intolerance, alternatives are minocycline, clarithromycin, fluoroquinolones or co-trimoxazole.

In some cases, particularly when high risk of chronic Q Fever is suspected, prolonged antibiotic prophylaxis with doxycycline and hydroxychloroquine has been recommended (up to 24 months) ^[26]. Hydroxychloroquine is recommended based on its effect elevating lysosomes pH and facilitating doxycycline activity. However, this recommendation is only based on in vitro studies and is, therefore, debated (low grade of evidence).

In pregnant women, treatment of acute Q Fever is based on antibiotic therapy with co-trimoxazole, generally for several months, to be determined on a case-by-case basis by the physician (folate supplementation is needed over the first trimester of pregnancy).

10. WHAT ARE THE PREVENTIVE MEASURES FOR PROFESSIONALLY EXPOSED PERSONS?

It is advisable to wear a well-fitting FFP2 mask and gloves in situations where there is a risk of direct exposure (contact/inhalation) to the bacteria: farrowing, handling of abortion products. This however proves difficult (to nearly impossible) to comply with in daily professional life. The knowledge of the infection status of the herd is desirable, but no national certification plan has been approved so far for ruminant farms in any of the EU27 member states.

In the event of massive exposure to *C. burnetii*, chemoprophylaxis has been proposed with antibiotic therapy for 5 days (except during pregnancy). This could be considered in the case of an exposed, non-immune professional potentially at risk of developing a severe form of Q Fever. In Europe, vaccination is not an option (see point 11).

FOCUS ON FARM VISITORS

It is advisable to limit the presence of non-professional visits on farms during the at-risk period (calving/lambing/kidding), particularly of vulnerable people (pregnant women, immunocompromised people etc.). If visits must be made during this period, it is advisable to isolate whelping females in a closed space, not accessible to visitors, to limit airborne exposure.

Would events likely to reveal a Q Fever infection occur (abortion series, presence of placenta on the farm/in pastures), visits to the farm should be suspended. If public visits are regular, it is essential to know the status of the herd. Another preventive gesture is to collect and destroy placentas and aborted fetuses.

The particular case of open farms (receiving visitors all-year round) is addressed in a dedicated factsheet, available on the European Q Fever committee's website.

11. VACCINATION A PUBLIC HEALTH BENEFICIAL APPROACH?

Q Fever is probably an immunizing disease in humans. Seroprevalence rates are very high among veterinarians and farmers, particularly in areas where *C. burnetii* circulates in livestock (to be noted that circulation may take place while undetected/unsuspected). The duration of seropositivity is highly variable, but considered of long duration. A seropositive person can therefore a priori be considered protected and not at risk for working with infected livestock.

As of 2025, the only approved human vaccine is an inactivated formulation available exclusively in Australia. It is not recommended for use during pregnancy. Also, candidate vaccinates have to be checked in serology beforehand, because seropositivity has been associated with severe side effects.

12. IS *C. BURNETII* A FOODBORNE PATHOGEN?

Q Fever is transmitted mainly by air, and therefore by inhalation. Although *C. burnetii* excretion in milk is commonly observed, it has been debated that there might be a risk of human contamination through food, even by raw milk from infected herds ^[36]. The French Food Safety Agency recently published an opinion concluding that the risk of illness linked to the ingestion of raw milk and products derived from ruminants infected with *C. burnetii* to be nil to virtually nil for the general population ^[37].

13. WHY IS THERE AN INTEREST IN CONTROLLING Q FEVER IN FARMED RUMINANTS WHEN MOST HUMAN CASES ARE SUBCLINICAL?

Although Q Fever is usually asymptomatic or mild, in certain individuals it can lead to severe, acute or persistent forms of the disease, justifying its control. Moreover, the objective of prevention and control is as important for professionals (farmers, veterinarians, slaughterhouse staff) as for non-professionals living close to animals, due to airborne transmission over long distances. In this regard, awareness should be raised in people involved in disease control, such as sanitarians, including veterinarians and human health professionals, and in the public, particularly in those individuals at-risk. Also, domestic ruminant vaccination provides a repeatable strong decrease in bacterial excretion load ^[38], and this contributes to the prevention of exposure among both professionals and neighboring residents.

REFERENCES

- Maurin M, Raoult D. Q Fever. *Clin Microbiol Rev*. 1999 Oct;12(4):518-53.
- EFSA, 2024. Story map on Q Fever, available online: <https://storymaps.arcgis.com/stories/7f9d9bceee-4b838eaaa0d2576ee0c0>.
- NIAID Biodefense Pathogens | NIAID: National Institute of Allergy and Infectious Diseases [Internet]. 2024 [cited 2025 Apr 19]. Available from: <https://www.niaid.nih.gov/research/niaid-biodefense-pathogens>.
- Gonzalez-Barrio D, Ruiz-Fons F, 2019. *Transbound. Emerg. Dis.*, 66, 2, 662-71. doi: 10.1111/tbed.13085.
- Yessinou R.E. et al., 2022. *Ticks Tick-Borne Dis.*, 13, 3, 101926. doi: 10.1016/j.ttbdis.2022.101926.
- González-Barrio D. et al., 2016. *Environ. Microbiol. Rep.*, 8, 5, 708-14. doi: 10.1111/1758-2229.12431.
- Gache K. et al., 2017. *Epidemiol. Infect.*, 145, 15, 3131-3142. doi: 10.1017/S0950268817002308.
- Bontje D. M. et al., 2016. *Prev. Vet. Med.*, 123, 71-89. doi: 10.1016/j.prevetmed.2015.11.004.
- Rodríguez N. F. et al., 2010. *Transbound. Emerg. Dis.*, 57, 1-2, 66-67. doi: 10.1111/j.11865-1682.2010.01116.x.
- Rizzo F. et al., 2016. *Prev. Vet. Med.*, 130, 10-17. doi: 10.1016/j.prevetmed.2016.05.014.
- Ruiz-Fons F. et al., 2010. *BMC Vet. Res.*, 6, 3. doi: 10.1186/1746-6148-6-3.
- Barlozzari G. et al., 2020. *Epidemiol. Infect.*, 148, e9. doi: 10.1017/S0950268819002115.
- Paul S. et al., 2012. *Prev. Vet. Med.*, 107, 1-2, 57-64. doi: 10.1016/j.prevetmed.2012.05.015.
- Fanelli A. et al. 2020. *Vet. Ital.*, 56, 3, 193-197. doi: 10.12834/VetIt.2321.132371.
- McCaughy C. et al., 2010. *Epidemiol. Infect.*, 138, 1, 21-27. doi: 10.1017/S0950268809002854.
- Konputtar A. et al., 2024. *Vet. World*, 17, 12, 2811-2828. doi: 10.14202/vetworld.2024.2811-2828.
- Beaudeau F. et al., 2021. *Zoonoses Public Health*, 68, 2, 144-152. doi: 10.1111/zph.12803.
- Gisbert P. et al., 2024. *Animals (Basel)*, 14, 9, 1313. doi: 10.3390/ani14091313.
- van den Brom R. et al., 2015. *PLoS One*, 10, 3, e0121355. doi: 10.1371/journal.pone.0121355.
- Cifo D. et al., 2023. *Bol. Epidemiológico Sem*, 31, 1, 56-64.
- Commandeur M. et al., 2014. *Int. J. Environ. Health Res.*, 24, 2, 137-157. doi: 10.1080/09603123.2013.800963.
- Tissot-Dupont H. et al., 2004. *Emerg. Infect. Dis.*, 10, 7, 1264-1269. doi: 10.3201/eid1007.030724.
- Eldin C. et al., 2017. *Clin. Microbiol. Rev.*, 30, 1, 115-190. doi: 10.1128/CMR.00045-16.
- Spronk I. et al., 2023. *Epidemiol. Infect.*, 151, e179. doi: 10.1017/S0950268823001401.
- Miyar I. et al., 2025. *Bol. Epidemiológico Sem.*, 33, 1, 58-70.
- Ghanem-Zoubi N. Paul M., 2020. *Clin. Microbiol. Infect.*, 26, 7, 864-870. doi: 10.1016/j.cmi.2019.10.024.
- Buijs S. B. et al., 2021. *Clin. Infect. Dis.*, 73, 8, 1476-1483. doi: 10.1093/cid/ciab476.
- European Food Safety Authority (EFSA), European Centre for Disease Prevention and Control (ECDC), 2024. *EFSA J.*, 22, 12, e9106. doi: 10.2903/j.efsa.2024.9106.
- Tan T. et al., 2024. *One Health*, 18, 100667. doi: 10.1016/j.onehit.2023.100667.
- FMR, 2024. Q Fever Awareness & Prevention - Human doctors' perspective. In preparation.
- Ben Amara A. et al., 2010. *PLoS One*, 5, 12, e15315. doi: 10.1371/journal.pone.0015315.
- Munster J. M. et al. *Placenta*, 33, 2, 128-131. doi: 10.1016/j.placenta.2011.11.012.
- de Lange M. M. et al., 2015. *BMJ Open*, 5, 4, e006821. doi: 10.1136/bmjopen-2014-006821.
- HCSF, 2013. *Fièvre Q. Recommandations de prise en charge* [Internet]. Rapport de l'HCSP. Paris: Haut Conseil de la Santé Publique. Available from: <https://www.hcsp.fr/Explore.cgi/avisrapportsdomaine?clefr=401>
- Georgiev M. et al., 2013. *Eurosurveillance*, 18, 8, 13-25. doi: 10.2807/ese.18.08.20407-en.
- Gale P. et al., 2015. *J. Appl. Microbiol.*, 118, 5, 1083-1095. doi: 10.1111/jam.12778.
- Opinion of the Anses of the 10 July 2010 (8 p.) [in French]. Access at <https://www.anses.fr/fr/system/files/MIC-2010sa0043.pdf>
- Gisbert P. et al., 2024. *Animals (Basel)*, 14, 10, 1484. doi: 10.3390/ani14101484.



EUROPEAN Q FEVER COMMITTEE

The Q FEVER COMMITTEE, co-chaired by Professors Raphaël Guatteo and George Valiakos, was created in July 2024 with the support of Ceva Santé Animale.

Discover who we are, our mission, and the latest expertise on Q Fever - Simply scan the QR code or visit the link below.

WWW.EUQFEVERCOMMITTEE.COM

