Q-Fever and One Health: Integrating Human, Animal, and Environmental Health

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ABSTRACT

In the intricate tapestry of infectious diseases, Q fever emerges as a formidable challenge, transcending species boundaries and demanding a holistic approach. This zoonotic ailment, fueled by the Coxiella burnetii bacterium, underscores the interconnectedness of animal, human, and environmental health—a narrative that unfolds across continents from Australia to the Netherlands. This abstract navigates the historical contours of Q fever, delving into its etiology, transmission dynamics, impact on animals, and the ominous specter it casts on human health. It highlights the global prevalence, with outbreaks resonating from the Australian abattoirs to the extensive Q fever epidemic that gripped the Netherlands from 2007 to 2010.The intricate pathogenesis of C. burnetii, its diverse manifestations in both humans and animals, and the challenges in diagnosis and prevention set the stage for a comprehensive One Health approach. This collaborative strategy, weaving together insights from human health, veterinary science, and environmental studies, emerges as a beacon in the fight against Q fever. Case studies from different corners of the globe, including South Africa, Europe, Australia, the USA, and the Netherlands, showcase the diverse efforts and challenges in implementing the One Health paradigm. The abstract also navigates through direct and indirect diagnostic approaches, underlining the complexity of detecting and managing this elusive pathogen. In conclusion, Q fever serves as a poignant exemplar of the intricate web connecting animals, humans, and the environment. As the world grapples with emerging infectious threats, the One Health approach stands as a crucial strategy, uniting experts across disciplines to safeguard the collective well-being of our planet's inhabitants.

Keywords: Q fever, one health approach, diagnosis, Epidemiology, human, animal

CITATION

Soomro H, Rajput ZI, Hassan MF, Khanzada M, Meghwar M, Soomro MA, Ishaque R, Rehman M and Junejo GA, 2023. Q-Fever and One Health: Integrating Human, Animal, and Environmental Health. In: Altaf S, Khan A and Abbas RZ (eds), Zoonosis, Unique Scientific Publishers, Faisalabad, Pakistan, Vol 4: 1-16. https://doi.org/10.47278/book.zoon/2023.134

CHAPTER HISTORY Received: 07-May-2023 Revised: 08-June-2023 Accepted: 20-July-2023

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1. INTRODUCTION

Coxiella burnetii is an intracellular bacteria that triggers Q fever, a widespread communicable disease. While it may be a significant public health concern in specific regions, enhancing global awareness of this illness is crucial. Current knowledge about *C. burnetii* remains somewhat limited, particularly concerning its resilience (both intracellular and environmental) and infectious characteristics. Ruminants are identified as the primary reservoir for this bacterium, releasing the pathogen through various means such as milk, feces, urine, vaginal mucus, and, notably, birth products. Inhalation emerges as the principal mode of contagion. Despite recurrently showing no symptoms in humans and animals, Q fever can lead to acute or chronic diseases. Vaccines containing inactive whole-cell bacteria have been evaluated for both human and animal use, although some shortcomings exist in this approach. Experimental recombinant vaccines hold significant promise (Porter et al. 2011).

2. HISTORICAL OVERVIEW

In Australia, the disease's initial emergence occurred amongst abattoir workers in 1935 in Queensland amidst an outburst of an unexplained feverish illness (Query fever) (Derrick, 1937). This zoonotic bacterial disease affects various hosts, including humans, ruminants, small rodents, dogs, cats, birds, fish, reptiles, and arthropods. Notably, ruminants like cattle, sheep, and goats are assumed to be the primary source of the pathogen (EFSA 2010).

3. UNDERSTANDING THE ETIOLOGY

Q fever, a globally significant acute (sometimes chronic) zoonotic ailment, arises from an obligate intracellular Gram-negative bacterium of the Legionellales order. It was assumed to be a rickettsia-like organism in mouse spleen and liver after exposure to abattoir workers' urine (Mitscherlich and Marth, 1984). Belonging to the *Coxiella* genus in the gamma division of Proteobacteria, *Coxiella burnetii*, much like other members of Proteobacteria, exhibits exceptional resistance to harsh environmental conditions and chemical agents, allowing it to persist for extended periods, occasionally even years. It predominantly targets circulating monocytes and macrophages in body tissues (Maurin and Raoult, 1999).

4. TRANSMISSION DYNAMICS

Human infection can result from tick bites, the breathing of infection-inflicted airdrops, the utility of coarse dairy goods, direct contact with infected animals' milk, urine, excreta, semen, and other potential sources of contamination (Bernard et al., 2012). Clinical manifestations vary and can range from asymptomatic cases (around 60%) or self-restricting feverish infections characterized by exhaustion, nuisance, general discomfort, myalgia, and arthralgia to more severe pneumonia or hepatitis. While less common, complications such as endocarditis, osteomyelitis, and aseptic meningitis may arise. Roughly 1-2% of acute cases may progress to chronic disease (Schimmer et al., 2010). Chronic cases result in various additional symptoms, including hepatitis, pneumonia, heart involvement, neurologic signs, and even persistent fatigue (Morroy G, Keijmel SP, et al., 2016). It can also lead to long-lasting complications like endocarditis, hepatitis, or neurological symptoms (Tissot-Dupont H, Raoult D, 2007). Additionally, Q fever is associated with adverse outcomes in pregnancy, including abortion, neonatal death, preterm birth, and intrauterine growth retardation (Angelakis, 2010).

5. IMPACT ON ANIMALS

While Q fever is frequently asymptomatic in animals, cattle and camels are prone to infertility, metritis, and mastitis, while sheep and goats might experience abortion, stillbirth, and preterm birth (Angelakis et al. 2013).

6. UNDERSTANDING THE ZOONOTIC POTENTIAL

C. burnetii's zoonosis potency extends from the direct interaction between people and diseased animals, including wild and domesticated vertebrates and ticks capable of shedding the microorganism (Setiyono et al., 2005). Studies have demonstrated that improper effluent management and the shedding of vaginal mucus, feces, and urine are primary sources of environmental contamination (Beaudeau et al., 2006). Consequently, the environment becomes contaminated with traces of the pathogen found in dust, compost, fields, fleece, and windborne (Clark and Magalhaes 2018). Transmission amongst humans via close contact with small ruminants can lead to isolated infections or widespread outbreaks. Animal owners, families, employees, and veterinarians are at higher risk due to frequent exposure to small ruminants and contaminated materials (Plummer et al., 2018).

7. EFFORTS FOR PREVENTION

The World Organization for Animal Health (OIE) entitles Q fever as a disease affecting various animal species. To safeguard against Q fever, the OIE advocates for preventative measures, including standardized analytic testing and immunizations for small and large ruminants (OIE., 2019). Instances of localized and sporadic clusters are liable to be singled out in human and ruminant populace via a comprehensive analysis of conveyed cases (Bauer et al., 2020). However, accurately quantifying sporadic cases and minor outbreaks proves challenging, as these reporting systems rely on the vigilance of healthcare, veterinarians, and other pertinent stakeholders. It's reasonable to assume that under-reporting occurs because of these factors (Winter et al. 2021).

8. EPIDEMIOLOGY

The largest Q fever outbreak on record has emerged in a condensed populace where intensive farming sprouts exponentially. Reports of Q fever cases are also rising in countries such as France, Germany, and the USA (Dijkstra et al. 2012).

The case-control study unveiled that exposure to a slaughterhouse was the primary risk factor for *Coxiella burnetii* infection. A connection between slaughterhouse operations and the temporal distribution of cases was established. The initial peak of activity in 1996 occurred in week 9, with the first Q fever case identified four weeks later, aligning with the typical incubation period. The pandemic's zenith was in week 18, coinciding with heightened slaughtering activities in week 14. This suggests that the pathogen was harbored in a contaminated abattoir environment. The airborne transmission of the pathogen is also plausible (Carrieri et al., 2012).

Extensively, Q fever cases so far have been archived except in New Zealand. Perhaps upsurging figures of animals, including domestic mammals, marine creatures, reptiles, ticks, and birds, have been reported as bacterium shedders in recent years (Anderson et al. 2013). The recognition of Q fever as a reportable disease in the United States since 1999 led to a 250% upsurge in human cases between 2000 and 2004 due to enhanced case identification (McQuiston et al., 2006).

This pattern is notably prominent in countries like France, Spain, and the United States. Hyperendemic foci are also identifiable in some of these countries, such as Martigues in southeastern France, where the local mistral wind carries spores from sheep herds and raises Q fever incidence rates to 34.5/100,000 residents. Occasional outbreaks, often familial, may result from an acquaintance to a mutual basis, such as parturient pets like dogs or cats shedding *C. burnetii*(Eldin et al. 2017).

In Africa, the disease began to sprout in 1955 across nine countries, suggesting widespread infection all over the continent (Kaplan, 1955). Mali, Burkina Faso, Nigeria, and the Central African Republic, with high densities of native ruminants, have shown the highest Q fever seroprevalence rates (Dupont et al., 1995). Seroprevalence of Q fever varies greatly, i.e., it may be as low as 1%, as reported in Chad, and up to a prevalence of 16%, as Dupont reported in Egypt. Notably, because of limited analytical tools in many African countries, the factual potency of Q fever remains undervalued. In Tanzania, *C. burnetii* was suggested to be the causative agent in 5% of severe pneumonia cases (Prabhu et al., 2011). A survey in Tanzania revealed 26.2% of zoonotic infections among severely ill febrile patients, with 30% being attributed to Q fever (Crump et al. 2013).

Additionally, seroprevalence upsurges in domestic ruminants in most African countries. The catered cattle surveys range from 4% in Senegal to 55% in Nigeria. In Egypt, sheep herds exhibited a gigantic gain of 33% seropositivity. Goats and camels also showed significant seropositivity rates, with the latter suggested as a substantial reservoir. In rural regions, human homes are in close propinquity to domestic ruminants, facilitating zoonosis. Consequently, *C. burnetii*DNA was perceived in 2% to 22% of household samples in rural Senegal (Ratmanov et al., 2013). Specific *C. burnetii* genotypes have been identified in Africa, primarily in ticks, with only a few detected in humans (Sulyok et al., 2014 & Mediannikov et al., 2010).

9. THE OUTBURST IN NETHERLANDS

Amidst 2007 and 2010, the Netherlands grappled with its most substantial Q fever epidemic, tallying over 4,000 documented cases and a potentially even more significant estimated count exceeding 40,000 (Eldin et al. 2017). The outburst transpired in a populace with a historically low Q fever seroprevalence of 2.4% (Schimmer et al. 2012). Notably impacted were the Noord-Brabant province in the country's southern region, along with Gelderland and Limburg provinces (Roest et al. 2011).

The massive influx of animals, exemplified by a staggering 75% surge in the goat populace between 1985 and 2009, likely facilitated the entrance of *C. burnetii*-infected animals into the country (Delsing et al., 2010). Retrospective studies have revealed that the infection had already been initiated in 2005, marked by abortion cases exceeding 60% on some farms (Roest et al. 2011).

In response, Dutch authorities had to swiftly devise and execute a comprehensive public health strategy starting in 2008. Rampant abortion cases in goats and sheep prompted a nationwide vaccination program. Nonetheless, human cases persisted at alarming levels. Consequently, in 2009, a large-scale culling initiative was mandated to target over 50,000 goats and sheep, even those in gestation. The results were evident in 2010, as a decline in human cases became apparent (Eldin et al. 2017).

10. Q FEVER IN CAYENNE, FRENCH GUIANA

In Cayenne, the capital city of French Guiana, the bacterium is responsible for a staggering 24% of community-acquired pneumonia cases (CAP), marking the ever-peak incidence documented globally (Epelboin et al. 2012). The first case dates back to 1955, involving a slaughterhouse worker (Floch H. 1957). Subsequent sporadic cases surfaced over the next four decades. However, during the 1990s, a remarkable

Q fever incidence occurred. Within a cohort of febrile patients, seroprevalence rates surged from 2% in 1992 to 24% in 1996 (Eldin et al., 2014).

11. PATHOGENESIS

An intracellular pathogen solely responsible for unraveling acute and chronic phases, with a strict reliance on host cells. Inside eukaryotic host cells, it thrives within vacuoles that closely resemble phagolysosomes. This pathogen has a global presence maintains its stability in the environment through persistent infections in ruminant animals. In humans, aerosol-mediated infection leads to invading and controlling alveolar macrophages, a bacterial Type 4B secretion system and secreted effector proteins facilitate it (Shaw and Daniel, 2019).

From its initial discovery as a feverish illness among laborers in a meat processing plant in Brisbane, Australia, Q fever's association with animals has been established. However, the impact on domestic animals was initially considered minimal or absent. In humans, Q fever can manifest in various ways, including acute, chronic, asymptomatic, or mild forms (Maurin and Raoult 1999).

In animals, entry typically occurs through the oropharynx. This pathogen displays high infectivity and is capable of causing infection with exposure to just a single animal (McQuiston et al., 2002). As the chief replication in lymph nodes occurs, a subsequent phase of bacteremia persists for around 5 to 7 days. Following this, the microorganism localizes within the mammary glands and placenta of pregnant animals, a process observed in infected domestic animals (Woldehiwet 2004)

C. burnetii showcases distinct features that set it apart from other bacterial species. Notably, its capacity to bourgeon in lysosomal vacuoles within phagocytic cells and variations in the lipopolysaccharide (LPS) antigen during Phase I and II contribute to its distinctiveness. Moreover, *C. burnetii* can occur in two discrete physical forms: the metabolically latent SCV (minor cell variant), known for its resilience, and the metabolically vigorous LCV (large cell variant), residing within the host cell (Boden et al., 2014; Sireci et al.2021).

An intriguing aspect of this pathogen is its variable incubation period in humans, spanning from 2 to 4 weeks or even longer. This variability depends on factors such as the inoculation dose, infection route, and the antigenic phase of *C. burnetii*. Lipopolysaccharide (LPS) molecules in its cell wall, with unique structure and antigenicity, pose a significant feature (Abnave et al. 2017).

C. burnetii's antigenic diversity is paramount for serological diagnosis and vaccine development. Notably, acute Q fever is characterized by elevated concentrations of anti-phase II antibodies (IgG and IgM), whereas chronic infection exhibits high concentrations of anti-phase I antibodies (IgG and IgA) (Setiyono et al., 2005). The genetic diversity of *C. burnetii* is restricted, with roughly 30 diverse genotypes (M. Million, 2009).

Upon entering the body, the microorganism adheres to phagocytic cellular membranes, particularly monocytes/macrophages. avb3 integrin mediates adsorption of virulent bacterium, whereas avb3 and complement receptor CR3 mediate attachment of avirulent bacteria. Phase I bacteria continue within phagocytic cells, while Phase II bacteria are eradicated. Furthermore, bacteria belonging to Phase I are engulfed in much lower quantities than Phase II bacteria (Angelakis and Raoult, 2010).

Entering phagolysosomes, monocytes, and macrophages engulf small cell variants (SCVs). Within these compartments, SCVs fuse with lysosomal contents, transitioning into metabolically vigorous forms, undergoing progress, and eventually developing into large cell variants (LCVs). Both antigenic forms of *C. burnetii* typically coexist in the phagolysosomal niche. Nevertheless, Phase II bacteria are rapidly obliterated. The acidic habitat of phagolysosomes provides a favorable setting for *C. burnetii* growth. Notably, the bacteria can enormously replicate within this acidic environment, and its predisposition for

tenacious contamination stands out. The utterly evolving cycle of a metabolically active Phase I bacterium transpires inside this acidic niche (Ullah et al. 2022).

The average growth period for the acute phase is approximately 20 days. The severity of illness is determined by the bacterial strain's virulence and the infecting dose. For instance, the QPH1 plasmid-containing strain is more virulent than the QPRS plasmid strain (Patil and Raghunath 2022).

The immune regulation of *C. burnetii* involves T-cells, but its control doesn't lead to complete eradication. The presence of *C. burnetii* is perceived in individuals who sound to have recovered and in the dental pulp of guinea pigs that were experimentally infected and seemingly cured (Honstettre et al., 2004 & Aboudharam et al., 2004). Even months or years after infection, *C. burnetii* DNA may yet remain detectable in bloodstream monocytes or bone marrow (Capo et al., 2003). Within vertebrate hosts, the infection prompts the development of granulomas in affected organs. The formation of these granulomas is enabled by the movement of monocytes via the vascular endothelium. A central lipid vacuole is a defining feature of a distinctive Q fever granuloma encircled by a ring of fibrinoid material (Maurin and Raoult, 2004) .

During the acute phase, only a small number, if any, of individual bacteria can be identified within granulomas. The role of TLR4 becomes evident in the creation of granulomas; mice lacking this receptor show a reduction in granuloma numbers. In response to infection, specific immunoglobulins are produced. Phase II antigen mainly stimulates the production of IgG, while IgM targets both phase I and II cells. Convalescent patients' monocytes exhibit the ability to eliminate *C. burnetii*. TLR4 also affects the cytokine response (interferon and tumor necrosis factor) after acute infections (Honstettre et al., 2004 & Maurin and Raoult, 2004).

12. ONE HEALTH

Throughout history, the concept of One Health has united experts of all diversified categories, such as animal, human, and environmental Health, on a local and global scale, all working together to ensure the well-being of both people and organisms (CDC, 2018). Collaborative efforts include enhancing communication, equipping clinicians with better knowledge and attitudes towards Q fever management, reinforcing laboratory capabilities, improving veterinary parameters, environmental monitoring, human and animal sero-surveillance, and facilitating access to screening and vaccination. An essential aspect of this collaboration is establishing animal surveillance systems and promoting data sharing and intelligence exchange between public Health and veterinary agencies (Dorko et al., 2012).

To fortify individuals from the infection, it becomes essential for both human and veterinary health experts to possess comprehensive knowledge about Q fever's diagnosis, control, prevention and its potential as a zoonotic disease (Winter and Campe 2022).

The yearly rate of reported cases in the US varies from 0.28 to 2.40 per million people. Comparable rates are observed in England and Wales. On the other hand, Australia reports a higher annual incidence, ranging from 15 to 49 cases per million individuals (Mahumud et al. 2019). The urgency of disease control becomes evident due to its significant impact on human Health, the potential for transmission through animal movements, extensive involvement of both animals and humans, insufficient national readiness for outbreak management, and diagnostic challenges (Burke et al. 2012).

The economic repercussions of Q fever are substantial, involving diminished livestock production alongside costs incurred for medical consultations, laboratory tests, hospitalization, and reduced productivity. This collective impact necessitates international assistance and response to address Q fever effectively (Palmer et al., 2007). Recognizing the interconnectedness of communicable diseases between *Homosapiens* and animals, the One Health approach presents robust strategies for managing the financial burdens associated with Q fever (Dantas-Torres et al., 2012).

13. BRIDGING OF Q-FEVER AND ONE HEALTH

The recent outbreak of infections occurring beyond the traditional high-risk workplaces within the community has broadened the perspective of Q fever. It's no longer viewed solely as an industry-related ailment but recognized as a broader public health concern (Tan et al. 2022). The concept of One Health serves as a worldwide strategy, fostering collaborative efforts across human Health, animal health, and environmental sectors. This approach is essential in the realm of infectious diseases, where 75% of infectious diseases are zoonotic (Pearsall 2019).

Effective management of zoonotic diseases, including Q fever, might necessitate a deeper grasp of the interconnected factors that pave the way for emerging illnesses. Achieving this involves cross-sector communication, engagement of stakeholders, and data sharing via a unified platform (Rahaman et al., 2019 & Kahn LH, 2019).

In this context, the concept of One Health is a timely and pertinent reminder of the practical realities that demand multi-sector collaboration, mainly when catering to zoonotic ailments. In Australia and internationally, implementing the One Health approach has yielded success in managing Q fever eruptions (Bond et al., 2016; Biggs HM et al., 2014 & Vellema P et al., 2014). Reporting of Q fever instances may be inflicted by factors like the nearness of wildlife to human housings, extensive ecological contamination from livestock and wildlife, geographic remoteness of susceptible populations, and restricted access to medical care (Karki S et al., 2015). Furthermore, there's limited information about altogether dissemination among livestock and human (Alvarez J et al., 2018).

The coordination and collaboration process entails a spectrum of measures, ranging from enhancing human surveillance to instituting animal surveillance. It includes fostering data exchange and intelligence sharing between veterinary and public health entities, improving communication and equipping clinicians with enhanced Q fever management knowledge. Additionally, consolidating laboratory capabilities, refining veterinary control protocols, monitoring the environment, conducting human-animal sero-surveillance, and facilitating access to screening and immunization all form integral components of this collaborative approach (Dantas-Torres et al., 2012 & Dorko et al., 2012).

Table 1 enlists various studies related to One health approach of different locations in preventing and controlling Q Fever.

14. DIRECT DIAGNOSTIC APPROACHES

Such methods involve bacterial or its parts detection.

14.1. DIRECT VISUALIZATION AND STAINING

An alternative method is Stamp-Macchiavello staining, also known as Macc or conventional Giemsa stain. However, direct visualization through bacterioscopic examination offers limited sensitivity and specificity, as it might be mistaken for other infections like Brucella spp., Chlamydophila spp., or Chlamydia spp (Guatteo R et al., 2006).

14.2. IMMUNOHISTOCHEMISTRY (IHC)

In chronic cases, IHC is employed to diagnose Q fever. This technique helps locate *C. burnetii* in tissues preserved in acetone/paraffin (Angelakis and Raoult 2010). The avidin-biotin-peroxidase complex IHC staining method was given by
Dilbeck and McElwain (Dilbeck and McElwain 1994).

Table 1: Studies relating to One health approach of different locations in preventing and control of Q Fever

Location	Study Type	One Health Practiced	Observed and Expected Outcomes	Comments
South Africa (Simpson et al. 2018)	Cross-sectional	• Risk factor inspection amongst farmers, herders, and veterinary staff. • Human Serology Recommended • Arranging sessions to educate and train human and veterinary experts alike on zoonosis.	• Q fever included in the differential diagnosis of febrile illnesses • Positive Q fever serology demonstrated • Educated clients for better disease prevention	• Diagnostic challenges related to febrile illnesses identified. • The small sample size and non-random selection of participants limit the generalizability of the results.
Europe (Mori et al. 2018)	Systematic review	Practiced • Risk factors reviewed: • Occupational factors e.g., farmers, abattoir workers Husbandry factors e.g., goat farming Environmental factors e.g. infected livestock transportation. Recommended • Q fever observation in high occurrence countries Collaboration across disciplines	• One Health emerges as a paragon for Q fever control, addressing complex interactions between the reviewed factor. • Promote optimum Health of humans, animals, and the environment	• One Health focus was drawn from the Netherlands experience, which may fail to appreciate the subtleties of Q fever epidemiology that govern possible control options in other countries.
Australia (Bond et al. 2016)	Outbreak Investigation	Practiced • Multidisciplinary epidemiological investigation and animal serology • Skin and serological testing for workers, subsequent vaccination • PCR testing of aborted materials, vaginal swabs, environmental samples • General measures, e.g. biohazard sign erection • Site surveillance launched • Health education • Management of farm environment e.g. management Recommended • Mandatory vaccination for all occupational contacts • Further research to identify possible interstate introduction of Q fever • Livestock vaccination	• Comprehensive risk assessment techniques and consensus control measures developed • Workers protected by HEPA* filters • Goats identified as likely source of the outbreak • Controlled human cases without source control • Could not prevent infections in workers' family members • Ongoing farm environmental contamination due to intensive breeding and milking of goats demonstrated • Presumably, these public health measures controlled the outbreak • Prevent acute Q fever cases. • Traditionally held views that interstate importation of *C. burnetii* to Victoria may be established. • Livestock and wildlife prevalence of *C. burnetii* could be established. • Reduced environmental shedding	• Key similarities with the Dutch outbreak include outbreak source, both occurring at goat farms; use of human vaccination, and application of a One Health approach. Differences include magnitude of the outbreaks, livestock vaccination was not used in the Australian outbreak because of manufacture

USA (Dunne et al.,2009)	Review	Practiced • Multidisciplinary diagnostic facilities • Quick result production • Fewer communication pitfalls among stakeholders • Public-private partnerships Joint investigation of Q fever cases • Human and animal serology Recommended • Vector-borne disease control requires human, animal and' vector surveillance	• Sample testing from a range of source • Stewardship and collaborations • Coordinated local responses against diseases and threats • Positive Q fever serology demonstrated • Shared resources and expertise • Animals and humans are protected	• Local, state, and federal levels involving public and private partnerships that combine human, animal, and ecological sectors help minimize resource exhaustion in control of zoonotic diseases.
Netherlan d (Enserink 2010)	Review	Recommended • Resolution of conflict between human and animal health experts. • Improved analytic approach • Immunization	• Through improved parameters, controlling Q-fever. • Minimized human interaction with animals	• Whilst outburst, human-animal communicative interaction was found to be minimal, contrary to what was believed to have a strong network. In this scenario, One Health needs a practical approach to minimize the gap rather than a theoretical way of thinking.

14.3. BACTERIAL CULTURING

Cultivating *Coxiella burnetii* remains a complex task, and the diagnostic sensitivity of this method is low. However, advancements now permit the cultivation of *Coxiella burnetii* in a cell-free laboratory medium without a host cell (Kuley et al. 2015). As Coxiella is intracellular in vivo, this new medium accurately replicates the organism's metabolic requirements within the phagolysosome. This discovery significantly enhances the potential for *Coxiella burnetii* research. Bacterial isolation is rarely pursued because of its high infectivity, particularly in veterinary medicine (Müller et al. 2014).

14.4. PCR

DNA from *Coxiella burnetii* has been successfully identified in several materials, including cell cultures, biopsies, blood, arthropods, and serum samples (Bennett and Banazis 2014). While conventional PCR cannot enumerate the microorganisms present, the introduction of real-time quantitative PCR (RTq PCR) transforms this method into a rapid diagnostic tool that provides measurable data. RTq PCR can be automated, making it suitable for extensive research. Many primers are accessible for diagnosis, with a commonly used primer derived from the often-repeated DNA sequence IS1111 (present in 7 to 120 copies per genome) known for its high sensitivity (Mori et al. 2017).

15. INDIRECT DIAGNOSTIC APPROACHES

15.1. CFT (COMPLEMENT FIXATION TEST)

CFT was the traditional serological diagnostic method in veterinary medicine, as recognized by the OIE. Usually employing phase 2 antigens, CFT can detect around 65% of infections during the second week post the inception of clinical manifestations and up to 90% by the fourth week (Porter et al. 2011).

15.2. ELISA (ENZYME-LINKED IMMUNOSORBENT ASSAY)

ELISA serves as an alternative technique for diagnosing animals and humans alike. This method offers improved accuracy, simplicity, and standardization compared to CFT (van der Hoek et al. 2012). Furthermore, a significant correlation between highly positive ELISA results and the prevalence of goat abortions has been observed (Rousset et al. 2007).

16. CONCLUSION

Q-fever stands as an emerging zoonotic disease that prevails like wildfire. In the realm of infectious diseases, Q fever is a prominent example that caters to animal, environmental, and human health relationships, underscoring the significance of the One Health approach. As this zoonotic disease traverses the boundaries between species, it demonstrates the imperative need for collaborative efforts that bridge the expertise of human health professionals, veterinarians, and environmental experts. Whether they unfold within heavily populated regions or remote corners of the globe, Q fever outbreaks lay bare the intricate web that links animals, humans, and their shared environment.

A shining example emerges from the Netherlands, where a massive Q fever outbreak spurred the nation into action. The dynamic interplay of increased livestock populations and human interaction with the environment led to a pivotal realization – that the Health of animals, humans, and the atmosphere is inextricably linked. As the Dutch authorities grappled with the complexities of controlling the outbreak, they recognized the need for a comprehensive strategy that integrates human and animal health concerns while acknowledging the environmental factors at play.

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