Coxiella burnetii infection (Q-fever) Fact Sheet

1. Disease overview

Q fever is an acute zoonotic febrile **infectious** disease caused by the bacterium ***Coxiella burnetii***. which can infect mammals, birds, reptiles, and arthropods. It causes a mild disease in ruminants, but can cause abortions and stillbirths in cattle, sheep, and goats.

Q-fever disease is a **WOAH-notifiable disease**.

Q-fever is listed in the **European Animal Health Law** under category **E**.

2. Agent

Q fever is caused by the bacterial pathogen *Coxiella burnetii,* a **gram-negative** pleomorphic **intracellular** coccobacillus, phylogenetically related to Legionella. A key feature of its cell envelope is its lipopolysaccharide (LPS), which undergoes **phase variation**. The virulent **Phase I** form expresses a complete, smooth LPS typical of isolates from infected hosts. In contrast, repeated passages in cell culture or embryonated eggs induces **Phase II**, characterized by a truncated LPS and markedly reduced virulence. This LPS variation is central to the bacterium's antigenic profile and pathogenicity.

Coxiella burnetii undergoes a dimorphic developmental cycle, alternating between two morphological forms: the **Small Cell Variant** (SCV) and the **Large Cell Variant** (LCV). The SCV is a metabolically quiescent, environmentally resistant form resembling a spore, which is highly stable and infectious. Upon entry into a host cell, the SCV differentiates into the LCV, a larger, metabolically active, and replicative form. This ability to persist as a highly resistant SCV contributes significantly to its environmental stability and low infectious dose.

As an obligate intracellular pathogen, *C. burnetii* replicates within a specialized, acidified compartment termed the **Coxiella-containing vacuole**. This vacuole fuses with lysosomes, creating a unique environment beneficial to bacterial growth.

3. Geographical Distribution

According to WAHIS data, the agent was reported in the EU in the last 2 years.

For more detailed information and dynamic maps, visit the *Geographical Distribution* section of the **disease profile** (accessible via the button in the top right corner).

4. Animal hosts

4.1. Primary animal species affected

Common reservoirs are domestic cattle, sheep, and goats, followed by horses, dogs, swine, camels, pigeons, ducks, geese, and turkeys. Wild birds, squirrels, mice, rats, cats, and rabbits can also serve

as reservoirs. Although Q fever can occur at any time of the year, most cases occur in the spring and early summer (April and May), the birthing season for cattle, sheep, and goats.

4.2. Clinical Signs

Q fever is typically a mild disease in animals, primarily affecting cattle, sheep, and goats. While infections are most often **subclinical**, when signs are present, they are predominantly linked to **reproductive dysfunction**, especially in ruminants during the final stages of pregnancy. The most serious consequence is reproductive failure, particularly **late-term abortion**, stillbirths, mummification of the fetus, and fever in the dam.

In **sheep and goats**, the most significant clinical sign is late-term abortion, often accompanied by the delivery of stillborn or weak offspring. Although abortions may occur sporadically, *C. burnetii* is capable of triggering sudden outbreaks of abortion in previously unexposed flocks or herds (known as *abortion storms*) which can result in substantial economic losses. Severe placentitis is a frequent pathological finding in aborting animals. Non-specific signs such as transient malaise or anorexia may precede abortion but are generally mild and transient.

In **cattle**, *C. burnetii* infection is commonly asymptomatic. When clinical signs are observed, they primarily involve late-term abortions, stillbirths, birth of weak calves, retained placenta, metritis, and infertility. Subclinical mastitis has also been associated with *C. burnetii* in dairy cows.

Other domestic animals, such as **cats and dogs**, are generally asymptomatic carriers. However, experimental infections in cats have shown transient fever, lethargy, and anorexia. Wild animals similarly tend to be asymptomatic.

4.2.1. Incubation Period

The incubation period of Q-fever disease, defined as the interval between infection and the onset of clinical signs, is generally variable and often difficult to precisely determine due to the predominantly subclinical nature of the infection. However, in ruminants that develop clinical signs, particularly reproductive issues like abortion, signs typically manifest weeks to months after exposure.

In experimental infections in goats, the incubation period has been shown to be as short as 3 days.

4.2.2. Morbidity and mortality

Direct mortality in animals due to *C. burnetii* is low. Animals, even those experiencing abortions, generally recover without severe systemic illness or fatal outcomes. Results obtained from the EFSA's systematic literature review resulted in one publication on experimental infection with *C. burnetii* meeting the inclusion criteria, which reported a fatality rate of 14% in goats.

4.2.3. Zoonotic Potential

Q-fever is a **zoonosis**.

5. Transmission

In animals, *Coxiella burnetii* transmission is primarily through the **inhalation of aerosols** containing contaminated material. High concentrations of the bacterium, up to a billion per cubic centimeter, are shed, particularly during parturition, in birth products (placenta, amniotic fluid), vaginal discharges, feces, and milk from infected ruminants. The long-lasting spore-like form can resist heat and drying, contaminating dust, bedding, and pastures and spreading long distances.

Q-fever can be also transmitted via **ticks** such as: Dermatocentor, Haemaphysalis, Hyalomma, Ixodes, Rhipicephalus, which pass the bacteria from an infected to a susceptible animal, and whose

faeces contain the bacteria, thus also contaminating the environment. While ticks can serve as vectors and play a role in maintaining *C. burnetii* in wildlife populations, their epidemiologic importance in direct animal-to-animal transmission within domestic livestock is generally considered secondary to aerosol transmission, especially during periods of birthing. The ingestion of contaminated materials, such as unpasteurized milk, can also lead to infection.

6. Diagnostic tests

WOAH-recommended tests for **agent detection** are PCR, culture and genotyping.

For **immune response detection**, the recommended tests are ELISA, indirect immunofluorescence assay, and complement fixation test.

The following table presents data on the sensitivity and specificity of several diagnostic tests from studies deemed eligible according to EFSA's systematic literature review protocol:

Target	Test	Specificity	N of studies	Sensitivity	N of studies
Antibody	Complement fixation test	99.83%	2	17.76%	2
Antibody	Indirect ELISA	100%	1	100%	1

7. Prevention and control

7.1. Vaccination

Currently, there is an inactivated Phase 1 approved vaccine in Europe, Coxevac.

7.2 Treatment

The treatment of infected animals is controversial. Some veterinarians advocate for the use of oxytetracyclines. And others claim that antimicrobial therapy has little efficacy. There were no papers evaluating treatment efficacy that met the inclusion criteria for the systematic literature review.