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Molecular detection and phylogenetic characterization of Coxiella burnetii in dairy cattle from the Al Ain region, United Arab Emirates

Robert Barigye

rbarigye@uaeu.ac.ae

United Arab Emirates University

Nabeeha Abdelgaleel Hassan

United Arab Emirates University

Hassan Zackaria Ali Ishag

Abu Dhabi Agriculture Food Safety Authority

Asha Antony

United Arab Emirates University

Aboma Zewude

United Arab Emirates University

Ibrahim Mohammed Abdalla-Alfaki

United Arab Emirates University

Asma Mohammed Abdi

Abu Dhabi Agriculture Food Safety Authority

Mohammed Saleh Albreiki

Abu Dhabi Agriculture Food Safety Authority

Kaltham Ali Hussain Kayaf

Ministry of Climate Change and Environment

Mervat Mari Hassan Al Nuaimat

Ministry of Climate Change and Environment

Mohamud Sheek-Hussein

United Arab Emirates University

Gobena Ameni

United Arab Emirates University

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Abstract

Coxiellosis (Q fever), caused by *Coxiella burnetii*, is a globally important zoonosis with significant implications for livestock production and public health. Although serological evidence indicates endemic exposure in dairy cattle in the United Arab Emirates (UAE), molecular data on circulating strains and shedding dynamics remain limited. This study investigated *C. burnetii* infection and genetic characteristics in commercial dairy herds in the Al Ain region of the UAE using combined serological and molecular approaches. A cross-sectional study was conducted on two commercial dairy farms, where 400 cattle were sampled for blood, milk, and vaginal swabs. Serological testing by indirect ELISA revealed an overall seroprevalence of 30.0% (95% CI: 25.6–34.8%), with no significant difference between the study farms. PCR detection identified *C. burnetii* DNA exclusively in milk samples from seropositive animals (21.7%; 95% CI: 14.8–29.9%), indicating active mammary shedding, while no bacterial DNA was detected in blood or vaginal swabs. Sanger sequencing and phylogenetic analysis based on the *htpB* gene demonstrated complete sequence homogeneity among UAE isolates and clustering within globally distributed zoonotic lineages. These findings provide integrated molecular and epidemiological evidence of endemic *C. burnetii* infection and active milk shedding in UAE dairy cattle. The results suggest that mammary-associated shedding may play a key role in maintaining infection within herds and highlight the importance of combining serological and molecular approaches for accurate surveillance. This study contributes baseline molecular data from an underreported region and supports the need for One Health-oriented control strategies to mitigate both animal and public health risks.

1.0 Introduction

Coxiellosis (Q fever) is an emerging and re-emerging zoonotic disease of major significance to both livestock production and public health worldwide. The disease is caused by *C. burnetii*, an obligate intracellular, Gram-negative bacterium belonging to the family Coxiellaceae within the subdivision Proteobacteria[1]. In livestock, particularly cattle and small ruminants, infection is primarily associated with abortion and other forms of reproductive failure, resulting in substantial animal health and economic impacts[2].

In humans, *C. burnetii* infection is frequently subclinical; however, symptomatic cases may present as an acute febrile illness characterized by influenza-like symptoms, headache, and musculoskeletal pain[3]. A subset of infected individuals may develop chronic Q fever, which is associated with severe complications including atypical pneumonia, granulomatous hepatitis, and endocarditis[3]. Endocarditis, in particular, carries a high fatality risk, while infection during pregnancy has been linked to adverse outcomes such as abortion, stillbirth, and preterm delivery[4].

It has long been established that infected animals shed large quantities of *C. burnetii* into the environment through placental tissues, foetal fluids, milk, vaginal secretions, and faeces[5,6]. Shedding intensity usually peaks during abortion events and the periparturient period, facilitating environmental

contamination and long-term persistence of the pathogen within livestock production systems, particularly under intensive management conditions[6–8]. The economic consequences of reproductive losses due to *C. burnetii* can be substantial, as demonstrated by estimates of €161–336 million[9] to €250–600 million[10] during the Dutch Q fever epidemic.

Q fever also represents an important occupational health hazard. Human infection typically occurs via inhalation of aerosolized particles contaminated with animal excretions or reproductive materials[1,3], and disproportionately affects livestock farmers, veterinarians, abattoir workers, and laboratory personnel[11,13]. In the UAE, a recent One Health study reported seroprevalence rates of 24.4% among abattoir workers and 52.9% among slaughtered camels, highlighting ongoing occupational exposure risks[14].

Globally, *C. burnetii* infection has been reported across all continents, including the Middle East and Gulf region, where infection has been documented in domestic animals in Saudi Arabia, Oman, Iran, Lebanon, and Jordan[15–20]. In the Emirate of Abu Dhabi (EAD), UAE, recent serological investigations provide compelling evidence that *C. burnetii* infection is endemic in dairy cattle. A farm-based study in the Al Ain region reported a seroprevalence of 36.5% and a significant association between seropositivity and abortion history[21], while a later large-scale survey of 17 commercial dairy farms recently documented a seroprevalence of 49.0%[22]. Despite this growing serological evidence, however, molecular epidemiological data describing circulating *C. burnetii* strains in the UAE and the wider Gulf region have remained limited. Genetic characterization of circulating strains is essential for understanding transmission dynamics, zoonotic potential, and for informing evidence-based control strategies. Therefore, the present study was conducted to detect *C. burnetii* infection by PCR and to perform *htpB* gene-based phylogenetic analysis of *C. burnetii* strains circulating in commercial dairy herds in the EAD thereby generating baseline molecular data that will contribute to One Health–oriented surveillance and control efforts.

2.0 Materials and methods

2.1 Study settings, study design, sample size and sampling

A descriptive cross-sectional study was conducted at two large commercial dairy cattle farms located in the Al Ain Region of the EAD, both of which had been previously identified as seropositive for *C. burnetii*. The required sample size for each farm was calculated using the formula described by Thrusfield[23], assuming a 2% margin of error, a 98% confidence level, and an expected prevalence of 0.5. To account for potential non-compliance and design effects, the calculated sample sizes for each of the two study farms were increased to 200 animals. Within each farm, individual animals were selected by simple random sampling. For each selected animal, jugular blood, milk, and vaginal swab samples were collected under aseptic conditions.

2.2 Indirect enzyme-linked immunosorbent assays for anti-*Coxiella burnetii* antibodies

Clotted jugular blood samples were centrifuged, and sera were separated and stored at − 20°C until tested for antibodies against *C. burnetii* using a commercial indirect ELISA (Q fever *C. burnetii* antibody test kit; IDEXX Laboratories, Switzerland), according to the manufacturer's instructions. Test sera and kit-provided positive and negative controls were diluted 1:10 and added (100 µL) to microtiter plates pre-coated with inactivated *C. burnetii* antigen. Plates were incubated at 37°C for 60 min, washed, and incubated with a peroxidase-labelled anti-ruminant IgG conjugate for a further 60 min at 37°C. Following a second washing step, tetramethylbenzidine (TMB) substrate was added, and optical density was measured at 450 nm using a microplate reader (BioTek Instruments, USA). Results were expressed as sample-to-positive control ratios (S/P%), with samples showing S/P ≥ 40% considered positive and those with S/P < 30% considered negative for *C. burnetii* antibodies.

2.3 Real time PCR for the detection of Coxiella burnetii DNA from milk, vaginal swabs, and blood

2.3.1 Nucleic acid (DNA) extraction

Total nucleic acid was extracted from milk samples of seropositive cattle using the Animal Virus DNA and RNA Extraction Kit (PLUS) (Xi'an Tianlong Science and Technology Co., Ltd., Shaanxi, China), following the manufacturer's instructions. Briefly, 400 µL of lysate was processed, and nucleic acids were eluted in 80 µL of the supplied elution buffer.

2.3.2. Amplification of *htpB* Gene, Sequencing and Phylogenetic Analysis

2.3.2.1. Amplification of *htpB* gene

A partial sequence of *htpB* gene of *C. burnetii* was PCR amplified by nested PCR following method from previous publication[24]. The PCR product for the first round was 521 bp while for the second round it was 325 bp. The AmpliTaq Gold™ 360 Master Mix kit (ThermoFisher) was used per the instructions of the kit manufacturer with the total volume of the master mix of 23.5 uL. The *htpB*F1: 5'-GCGGGTGATGGTACCACAACA-3' and *htpB*R1: 5'- GGCAATCACCAATAAGGGCCG-3' were used for first round, while *htpB*F2 5'- TGCTGGAATGAACCCCA-3' and *htp*BR2 5'- TCAAGCTCCGCACTCATG-3'[24,25] were used for the second round. About 1.5 µL of the extracted DNA was added to the master mix to complete the volume to 25 uL. The thermal cycling conditions performed as previously described To et al.,[24] and Selmi et al.,[25], and the amplicon was visualized in a 1.8% agarose gel.

2.3.3 Sanger Sequencing and BLAST analysis

The PCR product (325 bp) of the second PCR reaction used for sequencing using Sanger-sequencer at Abu Dhabi Agriculture and Food Safety Authority (ADAFSA) laboratory using the same primers used for

the second round of the nested PCR. The PCR products were purified with ExoSAP-IT™ PCR Product Cleanup Reagent (Thermo Fisher Scientific) and then cycling sequencing was performed using BigDye™ Terminator v3.1 Cycle Sequencing kit (Applied Biosystems). The reaction mixture totaling 20 µL that comprised of comprised 9 µL of water, 3.5 µL of 5× Sequencing Buffer, 1 µL of the BigDye™ Terminator v3.1, 1 µL of primers (3.2 pmol/µL), and 5.5 µL of DNA was used. The products were further purified with the BigDye™ XTerminator™ Purification kit (Applied Biosystems) and sequenced on a SeqStudio Genetic Analyzer (Applied Biosystems) using the 'MediumSeq BDX' run module. The obtained sequences were trimmed and assembled with CLC Genomic Workbench v.20 (Qiagen, Aarhus, Denmark) and subjected to BLAST analysis using the BLAST Tool at NCBI GenBank (Basic Local Alignment Search Tools) (http://www.ncbi.nlm.nih.gov/blast, accessed on 02-10-2025) to confirm the sequence identity.

2.3.4 Sequence Alignment and Phylogenetic Analysis

The *htpB* gene of the *C. burnetii* -UAE strains were aligned using the ClustalW program in MEGA 11[26] along with other corresponding *C. burnetii* reference sequences obtained from NCBI nucleotide database. In total 110 sequences were included in the phylogenetic tree which was constructed with the Maximum Likelihood technique and the Kimura 2-parameter model[27] within the MEGA 11 program with 1000 bootstrap replicates.

2.3.5 Statistical Analysis

Seroprevalence and PCR positivity proportions were calculated as the number of positive animals divided by the number tested, and expressed as percentages with corresponding 95% confidence intervals (CIs) based on the binomial distribution. Differences in seroprevalence and PCR detection rates between farms were evaluated using the chi-square test for independence. Statistical significance was assessed at a two-sided alpha level of 0.05. All analyses were conducted using standard statistical software.

2.4 Ethical Approval and Institutional Permissions:

Ethical approval for this study was obtained from the Animal Research Ethics Committee of the United Arab Emirates University (Ref. No. ERA_2024_4779; approved on 20 June 2024). In addition, permission to publish the research findings was also obtained from the Abu Dhabi Agriculture and Food Safety Authority and the Ministry of Climate Change and Environment, UAE. The authors confirm that all laboratory and field methods, used in the present study, were performed in accordance with the relevant guidelines and regulations. In addition, informed consent and permission were obtained from the farm owners for the use of their animals in this research.

3.0 Results

3.1 Serological and Molecular Detection of *Coxiella burnetii*

A total of 400 dairy cattle from two commercial farms in the EAD were tested for antibodies against *C. burnetii* using indirect ELISA. Overall, 120 animals were seropositive, corresponding to an apparent seroprevalence of 30.0% (95% CI: 25.6–34.8%). At Farm 7, 62 of 200 sampled cattle were seropositive (31.0%; 95% CI: 24.7–37.9%), while at Farm 11, 58 of 200 animals tested positive (29.0%; 95% CI: 22.9–35.8%) (Table 1). While evaluation of risk factors was beyond the scope of this study, a limited appraisal of the serology data demonstrated a higher *C. burnetii* seroprevalence among cattle aged 12–36 and 37–72 months, and comparatively lower seropositivity in animals older than 72 months. A chi-square test comparing farm-level seroprevalence showed no statistically significant difference between farms ($\chi^2 = 0.19$, df = 1, $p = 0.66$). In both herds, the number of animals sampled exceeded the calculated minimum sample size, increasing precision of the prevalence estimates. RT-PCR testing of ELISA-positive animals identified *C. burnetii* DNA in milk samples from 14 of 62 seropositive cattle at Farm 7 (22.6%; 95% CI: 13.2–34.7%) and 12 of 58 at Farm 11 (20.7%; 95% CI: 11.2–33.4%). Overall, 26 of 120 seropositive animals were milk PCR-positive (21.7%; 95% CI: 14.8–29.9%). Comparison of milk PCR positivity between farms revealed no statistically significant difference ($\chi^2 = 0.06$, df = 1, $p = 0.80$). No PCR-positive results were detected in whole blood nor vaginal swab samples from either farm (0%; 95% CI upper bound ≈ 3.0%). These findings suggest substantial prior exposure to *C. burnetii* within both dairy herds and confirm active bacterial shedding in approximately one-fifth of seropositive animals.

Table 1
Seroprevalence of *Coxiella burnetii* in dairy cattle from two commercial farms in the Emirate of Abu Dhabi, United Arab Emirates, determined by indirect ELISA, and corresponding RT-PCR detection of bacterial DNA in selected specimen types.

Study farm	Cattle population	Calculated sample size	Animals sampled	ELISA-positive n/%	PCR-positive Whole blood n/ %	PCR-positive milk n/ %	PCR-positive vaginal swabs n/ %
Farm 7	3,372	98	200	62 (31.0)	0 (0.0)	14 (23.0)	0 (0.0)
Farm 11	1,641	48	200	58 (29.0)	0 (0.0)	12 (21.0)	0 (0.0)
Total	5,013	146	400	120 (30.0)	0 (0.0)	26 (21.7)	0 (0.0)

3.2. Phylogenetic analysis of *Coxiella burnetii* strains

All the *C. burnetii* DNA detected in milk samples from seropositive animals were subjected to phylogenetic analysis; the *htpB* gene was sequenced and the BLAST analysis of the sequences (260 bp) confirmed the strains to be *C. burnetii* with a 100% identity to the references sequences of the *C. burnetii* available in the NCBI nucleotide database (Fig. 1). Moreover, the UAE *C. burnetii* sequences exhibited 100% nucleotide identity both within and between isolates obtained from the two farms (Fig. 2). Phylogenetic analysis based one the partial sequences of the *htpB* gene, further positioned the *C. burnetii* UAE strains within the *C. burnetii* cluster (Bootstrap value of > 90%), separate from the other

Coxiella endosymbionts (Fig. 3). Notably, the *C. burnetii* clade includes strains from Europe, Africa, and Asia that infect multiple animal species, humans, and ticks, highlighting its zoonotic potential. The *htpB* gene sequences of the representative 16 *C. burnetii* strains from the UAE identified in the present study were submitted to GenBank databases under accession numbers: PZ332774 (SUB16145222 NDF6), PZ332775 (SUB16145222 NDF7), PZ332776 (SUB16145222 NDF8), PZ332777 (SUB16145222 NDF14), PZ332778 (SUB16145222 MSK1), PZ332779 (SUB16145222 MSK2), PZ332780 (SUB16145222 MSK3), PZ332781 (SUB16145222 MSK4), PZ332782 (SUB16145222 MSK5), PZ332783 (SUB16145222 MSK6), PZ332784 (SUB16145222 MSK7), PZ332785 (SUB16145222 MSK8), PZ332786 (SUB16145222 MSK9), PZ332787 (SUB16145222 MSK10), PZ332788 (SUB16145222 MSK11), and PZ332789 (SUB16145222 MSK12).

4.0 Discussion

Serological screening in this study demonstrated moderate to high exposure to *C. burnetii* in both farms, with seroprevalence estimates of 31.0% and 29.0% in Farms 7 and 11, respectively, and no significant difference between locations. These findings suggest relatively homogeneous exposure within the two intensive dairy systems and likely reflect similar patterns in comparable farms in the Al Ain region of the EAD. In the context of previous studies from the same area, the observed seroprevalence is consistent with endemic transmission. Earlier investigations reported seroprevalence estimates of 36.5%[21] and 49.0% across 17 commercial farms[22]. Differences between studies may be attributable to variation in study design, herd structure, sampling strategies, or temporal changes in infection dynamics rather than true reductions in exposure. Besides, comparable seroprevalence levels have been reported in endemic settings across Europe, the Middle East, and Africa, where herd-level exposure often exceeds 20–30%[7,18,19]. Collectively, these findings support the conclusion that *C. burnetii* infection is well established in dairy cattle populations in the study region. Moreover, the absence of farm-level differences further suggests shared risk factors, including similar management practices, biosecurity conditions, and potential environmental contamination.

As observed in this study, age-specific patterns of seropositivity provide additional epidemiological insight. Higher seroprevalence in cattle aged 12–72 months, coupled with low seropositivity in animals older than 72 months, suggests that infection is acquired early in life. This pattern is consistent with previous studies, where early exposure has been linked to environmental contamination, close animal contact, and immature immune responses[2,7,28]. Lower seropositivity in older animals may reflect waning antibody titres, selective culling of animals with reproductive disorders, or cohort effects related to historical management practices[8,9].

It is noteworthy that although ELISA is a sensitive tool for detecting prior exposure, it does not distinguish between past and active infection[8,10].The integration of serology with PCR in this study therefore provides a more comprehensive assessment of infection dynamics. Notably, *C. burnetii* DNA was detected exclusively in milk samples from seropositive animals, with no detection in blood or

vaginal swabs. Approximately 21–23% of seropositive cows were actively shedding bacterial DNA in milk, consistent with findings from other endemic dairy systems[6,7,29]. The absence of differences in PCR positivity between farms suggests that shedding is primarily influenced by host-level factors, including age, reproductive status, and immune function, rather than farm-specific conditions.

On the basis of the present study data as well as observations made elsewhere, milk represents a sensitive matrix for detecting *C. burnetii*, particularly during chronic or subclinical infection, due to the organism's tropism for mammary tissue and its capacity for persistent intracellular survival[5,11]. In contrast, detection in blood is typically transient and limited to acute bacteraemic phases, while vaginal shedding is largely associated with abortion or the periparturient period[6,10,29]. The absence of PCR positivity in vaginal swabs is therefore consistent with sampling outside peak shedding periods. The observed discordance between ELISA and PCR results may reflect the complex pathobiology of *C. burnetii*, where seropositive animals may no longer be shedding, while PCR-positive animals represent a more immediate transmission risk[7,8]. These findings underscore the value of combining serological and molecular approaches for accurate assessment of both exposure and active infection. The detection of active milk shedding in a substantial proportion of seropositive cattle has important implications. From an animal health perspective, persistent shedding contributes to environmental contamination and maintenance of infection within herds[6,29]. From a public health standpoint, it highlights occupational exposure risks associated with handling raw milk, despite the effectiveness of pasteurization in inactivating *C. burnetii*[5]. The presence of bacterial DNA in milk from asymptomatic animals reinforces the need for One Health–oriented surveillance in endemic settings[2,19].

Molecular analysis of the partial *htpB* gene confirmed that all PCR-positive samples were *C. burnetii*, with 100% nucleotide identity to reference sequences in the NCBI database. Identical sequences within and between farms indicate a high degree of genetic homogeneity among circulating strains. Similar patterns have been reported elsewhere and may reflect recent common ancestry, inter-farm transmission, or shared infection sources[10,15,25]. Furthermore, phylogenetic analysis placed UAE isolates firmly within the *C. burnetii* clade, clearly separated from *Coxiella*-like endosymbionts, consistent with the established discriminatory value of the *htpB* gene[10,15,25]. The clustering of UAE strains with globally distributed isolates from multiple hosts supports limited host or geographic structuring and reinforces the zoonotic nature of *C. burnetii*[15,30]. While *htpB* sequencing provides robust species-level resolution, higher-resolution approaches such as MLVA or whole-genome sequencing would enhance understanding of transmission dynamics.

A key strength of this study is the combined use of ELISA and PCR to distinguish exposure from active shedding. However, the cross-sectional design and reliance on single-time-point sampling may underestimate intermittent shedding. Longitudinal studies incorporating repeated sampling and quantitative PCR would provide greater insight into infection dynamics[8,29]. Nonetheless, these findings provide robust baseline data and support the use of milk-based PCR as a practical surveillance tool in endemic dairy systems. In conclusion, this study has provided integrated serological and molecular

evidence of endemic *C. burnetii* infection and active milk shedding in dairy cattle in the Al Ain region of the UAE. The findings highlight apparent early-life exposure, mammary-associated shedding, and genetic homogeneity of strains within globally distributed zoonotic lineages. Together, these results support the use of combined serological and molecular surveillance approaches and reinforce the need for coordinated One Health strategies to mitigate both animal and public health risks.

Declarations

Ethics approval and consent to participate

The research project was approved by the Animal Research Ethics Committee of the United Arab Emirates University (Ref. No. ERA_2024_4779) on the 20th June 2024. Publication of this manuscript has also been duly authorized by the UAE Ministry of Climate Change and Environment.

Consent for publication

Not applicable

Competing interests

The authors declare no conflicts of interest.

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Author Contribution

R.B served as the principal investigator and, together with G.A, A.A, I.M.A, K.H.K, M.M.A, and M.S.A contributed to study conceptualization, grant proposal development, data interpretation, and manuscript preparation. N.A.H and A.Z performed the indirect ELISA testing of blood samples while H.Z.A.I, A.M.A, and M.S.A performed the PCR, Sanger sequencing and phylogenetic analysis of the Coxiella burnetii strains. All authors reviewed and approved the final manuscript.

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Data Availability

The datasets generated and/or analysed during the current study are available in the GenBank of National Center for Biotechnology Information (NCBI) repository under accession numbers: PZ332774 (SUB16145222 NDF6), PZ332775 (SUB16145222 NDF7), PZ332776 (SUB16145222 NDF8), PZ332777 (SUB16145222 NDF14), PZ332778 (SUB16145222 MSK1), PZ332779 (SUB16145222 MSK2), PZ332780 (SUB16145222 MSK3), PZ332781 (SUB16145222 MSK4), PZ332782 (SUB16145222 MSK5), PZ332783 (SUB16145222 MSK6), PZ332784 (SUB16145222 MSK7), PZ332785 (SUB16145222 MSK8), PZ332786 (SUB16145222 MSK9), PZ332787 (SUB16145222 MSK10), PZ332788 (SUB16145222 MSK11), and PZ332789 (SUB16145222 MSK12). Any other datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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Figures

	Description	Scientific Name	Query Cover	Per. Ident	Accession
☑	Coxiella burnetii isolate DRM95 60 kDa heat shock protein (groEL) gene, partial cds	Coxiella burnetii	92%	100.00%	MG860513.1
☑	Coxiella burnetii isolate CB1 GroEL (groEL) gene, partial cds	Coxiella burnetii	92%	100.00%	OQ883856.1
☑	Coxiella burnetii strain 701CbB1 chromosome	Coxiella burnetii	92%	100.00%	CP014553.1
☑	Coxiella burnetii strain 2574 chromosome	Coxiella burnetii	92%	100.00%	CP014555.1
☑	Coxiella burnetii Z3055 complete genome	Coxiella burnetii Z3055	92%	100.00%	LK937696.1
☑	Coxiella burnetii strain Scurry_Q217 chromosome	Coxiella burnetii	92%	100.00%	CP014565.1
☑	Coxiella burnetii strain CbB1 heat shock protein B (htpB) gene, partial cds	Coxiella burnetii	92%	100.00%	EU888861.1
☑	Coxiella burnetii strain nine mile phase II chromosome, complete genome	Coxiella burnetii	92%	100.00%	CP035112.1
☑	Coxiella burnetii strain CB13 chromosome	Coxiella burnetii	92%	100.00%	CP103433.1
☑	Coxiella burnetii isolate CB1 GroEL (groEL) gene, partial cds	Coxiella burnetii	92%	100.00%	OQ627011.1

Figure 1

Percentage similarity of the partial *htpB* gene sequence (260 bp) of the study strains to reference sequences following NCBI BLAST analysis. The top 10 most similar sequences are shown.

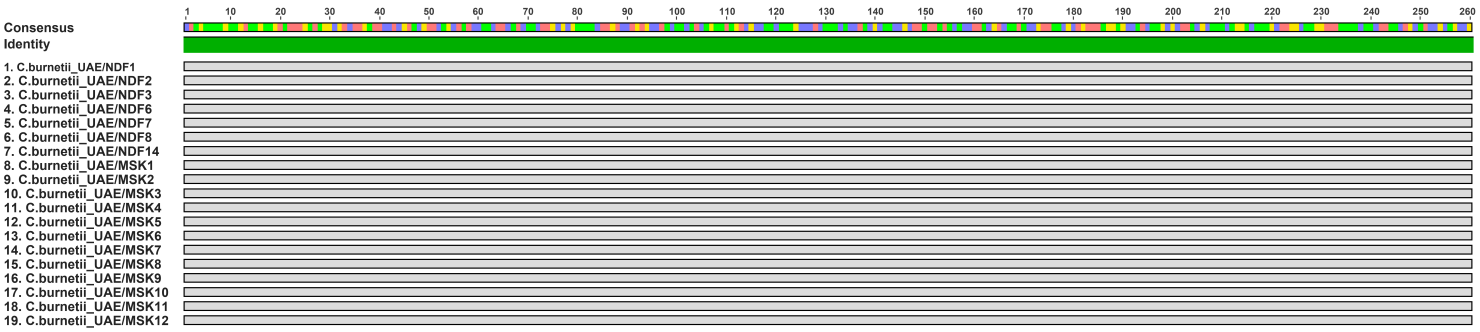


Figure 2

Alignment view of the *htpB* sequence of *Coxiella burnetti*obtained from the two study farms in the EAD, UAE (Strains 1-7 from Farm 7 and strains 8-19 from Farm 11). The alignment showed 100% identity within and between the strains based on the 260 bp of *htpB* gene.

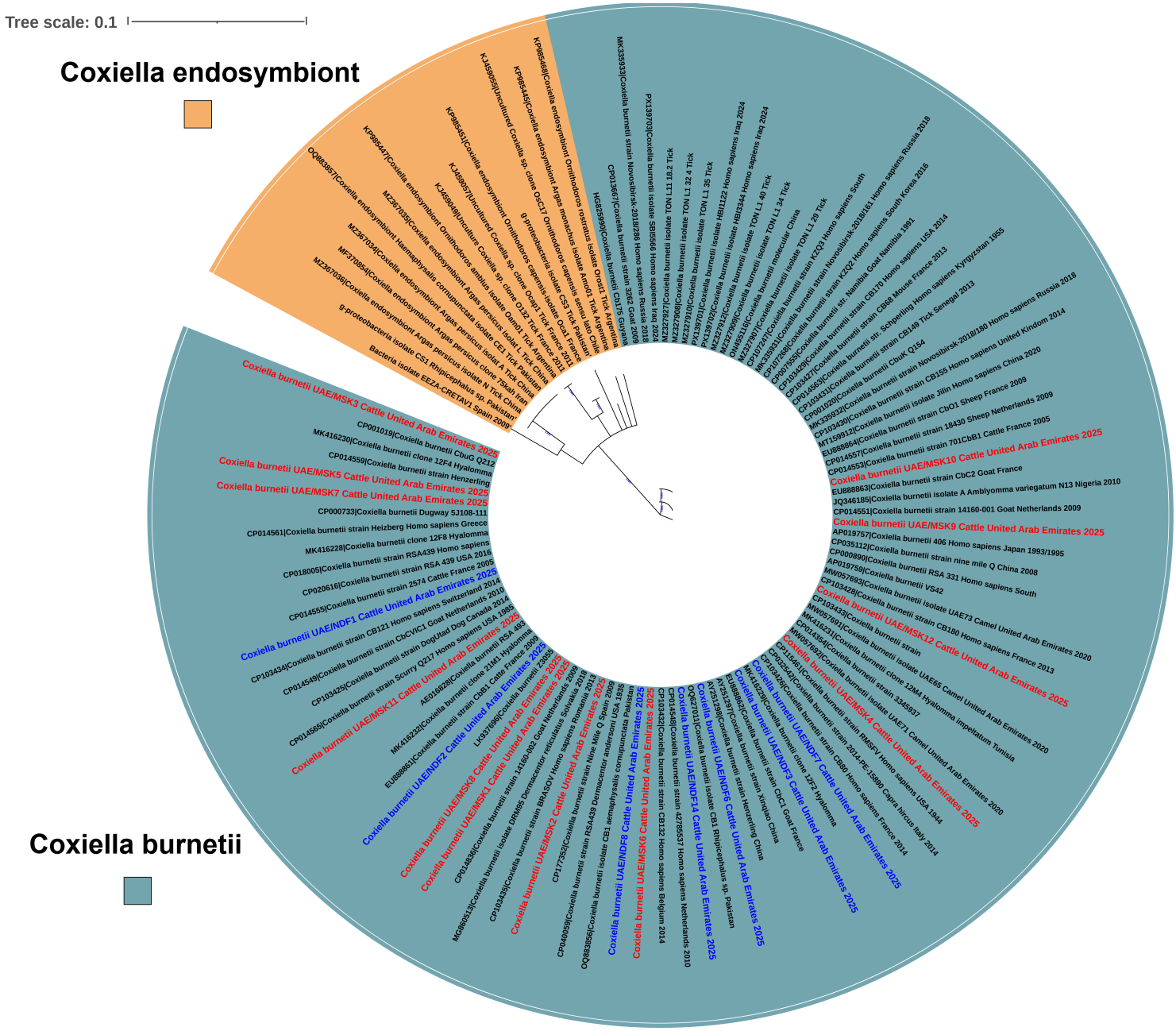


Figure 3

Phylogenetic tree based on partial *htpB* gene sequences (260 bp) of *Coxiella burnetii* strains from Farm 11 indicated in blue fonts (n = 7) and Farm 7 indicated in red fonts (n = 12), together with reference sequences from GenBank (black). The tree was constructed using the “Maximum Likelihood Method” in MEGA 11 with 1,000 bootstrap replicates. UAE strains clustered within the *C. burnetii* clade (light green fill), with Farm 11 and Farm 7 isolates shown in red and blue, respectively, while *Coxiella*-like endosymbionts formed a distinct clade (orange). GenBank accession numbers are indicated.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryfilesS1PCRGel.pdf
- PROOFOFSEQUENCESUBMISSIONTOGENBANK.pdf