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National serosurvey and risk mapping reveal widespread distribution of *Coxiella burnetii* in Kenya

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***Coxiella burnetii*, the causative agent of Q fever, is an emerging pathogen that has the potential to cause severe chronic infections in animals and humans worldwide. The detrimental impact on public health is projected to be higher in the low- and middle-income countries given their lower capacity to sustain effective surveillance and response measures. We implemented a national serosurvey of cattle in Kenya to map the spatial distribution of the pathogen. The study used serum samples that were collected from randomly selected cattle in different ago-ecological zones across the country. These samples were screened for the pathogen using PrioCHECK Ruminant Q Fever AB Plate ELISA kit. The laboratory findings were analyzed using INLA package to identify risk factors for *C. burnetii* exposure from herd- and animal-level factors, area, and bioclimatic datasets accessed from online databases. A total of 6,593 cattle were recruited for the study; of these, 7.9% (95% CI; 7.2–8.5) were seropositive. Outputs from the multivariable analysis revealed that the animal age and some of the geographical variables including wind speed, area under shrubs and "petric calcisols" type of soil were significantly associated with *C. burnetii* seropositivity. Being a calf, weaner or subadult was associated with lower odds of exposure compared to being an adult by 0.24 (credibility interval: 2.5% and 97.5%), 0.41 (0.30–0.55) and 0.51 (0.38–0.69), respectively. In addition, a unit increase in the wind speed increased the odds of *C. burnetii* seropositivity by 1.27 (1.05–1.52) while an increase on the land area under shrubs was associated with lower odds of exposure (0.67 [0.47–0.69]). The effect of petric calcisols was non-linear; an increase of the land area with this soil type was associated with an exponential increase in *C. burnetii* seropositivity. This study provides new data on *C. burnetii* seroprevalence, information of its risk factors and a prevalence map that can be used for *C. burnetii* risk surveillance and control. The identification of environmental risk factors for *C. burnetii* exposure, and the increasing awareness of the zoonotic potential of the pathogen, calls for the need to enhance the existing collaborations for the surveillance and control of *C. burnetii* in line with the One Health framework. The evidence generated on the potential role of environmental factors can also be used to design nature-based interventions, such as replacement of vegetation in denuded areas, to reduce potential for the aerosolization of the pathogen. Livestock vaccination in the hotspots would also reduce animal infections and hence the contamination of the environment.**

Keywords *C. burnetii*, Risk factors, Q fever, One health, Kenya

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Coxiella burnetii is a highly contagious zoonotic pathogen with a global distribution, except in New Zealand and Antarctica[1]. The agent causes Q fever, a re-emerging but neglected infectious disease which is listed among the most important zoonotic diseases in several countries. In Africa, Q fever occurs in 24 of 54 countries[2,3], with domestic animals such as cattle, goats, and sheep serving as important reservoirs and sources of zoonotic spillovers[4]. Most of these infections are subclinical; a few often lead to serious reproductive complications such as infertility, abortions, birth of weak, or stillbirths[5]. Infected animals usually shed large amounts of infectious *C. burnetii* material during birth and through milk, faces, urine, and vaginal mucus for several years[6–8]. Human exposures occur through direct or indirect contact with tissues of infected animals or contaminated tick feces, dust, water, soil, and air from infected livestock premises. More than half of the human cases remain asymptomatic. Clinical human cases can cause chronic, persistent and debilitating illness, with serious complications including endocarditis, pneumonia, hepatitis, abortion, premature birth, low birth weight in newborns, chronic fatigue and even death[9–11].

Q fever has caused several outbreaks across the world, the most prominent one being the Netherlands outbreak that occurred between the years 2007 and 2010. That outbreak originated from dairy sheep and goat farms and resulted in >4000 human cases[12–14]. The outbreak caused unprecedented public, veterinary, and economic consequences with massive culling of pregnant goats and sheep. Further studies on this outbreak revealed various exposure pathways in humans that included contact with aborting small ruminants[12–14]. Outbreaks have also been reported in other countries such as Australia where humans got infected from direct or indirect contact with infected livestock, wildlife, and contaminated environments[15].

There is limited information on *C. burnetii* epidemiology in the literature. A recent review showed that *C. burnetii* seroprevalence ranges between 3.0 and 35.8% in humans, 7.4–51.1% in cattle, 6.7–20% in sheep, 20–46% in camel and 20–46% in goats in Kenya[16]. *C. burnetii* infections have also be reported in ticks[17,18]. In general, goats, camels and wild animals in the giraffidae family[19] experience higher levels of exposure compared to other mammals that are found in the same environments.

C. burnetii can live freely in the environment as a small compact rod that is resistant to drying and UV radiation, allowing it to remain viable in the environment for years[20]. The pathogen can be readily aerosolized and dispersed over long distances by wind. Factors that can facilitate its dispersal include poor vegetation cover, low rainfall, low soil moisture, loose soils and high livestock density[8,21]. These factors, including vegetation cover and ground water levels are thought to influence the distribution of *C. burnetii* in a landscape[23–27]. The high resistance of the free living form of the pathogen to environmental stressors like heat, pressure, and certain antiseptics, along with its ability to spread in aerosolized form makes it a bioterrorism agent[22].

Q fever therefore presents an enormous health and socio-economic risk, particularly in marginalized areas, if no interventions are deployed. The disease has not attracted enough attention in many countries despite the availability of state-of-the-art analytical tools, such as risk mapping, that can be used to inform targeted bio-surveillance measures. We implemented a national serosurvey in Kenya to determine spatial distribution of the pathogen, and to identify risk factors that influence its occurrence. A number of seroprevalence studies have been done in the country[28–35], but the areas that were covered were patchily distributed. The studies were also implemented in different periods and focused on diverse hosts. We believe this is the first comprehensive study on *C. burnetii* epidemiology in the country, which also focused on determining ecological factors that can be associated with the transmission patterns of the disease in cattle, which is an important source of income to a wide range of farmers across the Country.

Materials and methods

Methods

Study design

The study was implemented as a national cross-sectional survey that covered all the five agro-ecological zones including agro-alpine, high and medium potential, semi-arid, arid, and very arid regions. It was implemented between November 2020 to August 2021 to investigate the epidemiology of *Brucella*, *C. burnetii* and Rift Valley fever Virus (RVF) in Kenya. The study used cattle as a representative livestock species given that this livestock species was commonly raised in all the ecological zones in the country, unlike the small ruminants and camels that had limited distribution. This study was part of the *Brucella* seroprevalence study that was published in 2023[36]. All animal procedures performed in this study were in strict accordance with the guidelines and regulations set forth by relevant authorities that provided ethical approval.

Ethics approval and consent to participate

The protocols for this study were reviewed and approved by the ILRI's Institutional Animal Care and Use Committee (IACUC), with an approval REF: ILRI-IACUC2021-18. Similarly, the National Commission for Science, Technology, and Innovation (NACOSTI) also reviewed and approved with reference number NACOSTI REF: 218346. The NACOSTI, IACUC, as well as the ARRIVE guidelines and regulations were strictly observed during the implementation of this study, while only considering cattle from herds where informed consent was issued by the household head.

Sample size determination

The study used the standard algorithm for determining a sample size that is required to estimate a population proportion. *A priori C. burnetii* prevalence of 50% was used due to the limited information on the seroprevalence of the pathogen at the national level. It was also assumed that the *C. burnetii* seroprevalence would be estimated with a precision of 5% at 95% confidence level. A *naïve* sample size of 384 animals was derived based on these assumptions. This was further adjusted to account for potential clustering at the herd level. With an assumed intra-cluster correlation of 0.3 and a maximum of 25 cattle per herd, this adjustment increased the sample size

to 3,150 animals. The sample size was further expanded to accommodate potential confounding variables. We assumed the predictive model for *C. burnetii* would involve at least two continuous predictors, each with a significant correlation of 0.5 with the outcome. To meet these criteria, we calculated a sample size of 6,700 animals which would be distributed across 268 herds or sampling points.

Selection of the sampling units

A two-stage random sampling design was used to select the study subjects as previously described[36]. In the first stage, households were selected using random geographic coordinates (RGCs) whose distribution across the country was weighted by the population of cattle by agroecological zone (Fig. 1). Cattle herds or households that were located closest to a random point were selected for sampling. In the second stage, 25 apparently healthy cattle were selected from each of the herds or location that was chosen for sampling. If the selected herd had fewer than 25 cattle, additional herds around the reference point were recruited until the minimum number of animals per point was obtained. In cases where herds exceeded 25 cattle, a systematic random sampling method was used by first allocating each animal a number and establishing the herd size, before computing a sampling interval for random selection of the 25 sampled animals. Hand-held GPS devices were used to locate the random coordinates generated. The number of animals sampled at each point ($n = 25$) was assumed to strike a balance between statistical reliability, based on the central limit theorem, and practical feasibility with respect to the cost of implementing the survey and processing the samples.

Data collection

Animal-, herd- and area-level data were collected during sampling using a structured questionnaire administered to the household head. The animal-level characteristics that were obtained included age, sex, and reproductive syndromes that an animal had experienced. Those collected at the herd level included herd size, herd composition, and current reproductive syndromes. At the area level, geographic coordinates of the sampling sites as well as the livestock production system that many farmers practiced in the area were recorded.

Blood sample collection and analysis

Selected animals were restrained and approximately 6 ml of blood was collected from the jugular vein using a barcoded plain vacutainer tube. After collection, blood samples were kept for about 15 min to allow clotting; they were then transported in a cool box to the field laboratory. Serum was extracted from the clotted blood through centrifugation at 1300 x g for 10 min. The serum samples were then aliquoted into barcoded cryovials and stored in a motorized freezer at -20 °C. They were then transported to the International Livestock Research Institute (ILRI) laboratories in Nairobi in motorized cool boxes where they were stored at -20 °C until tested.

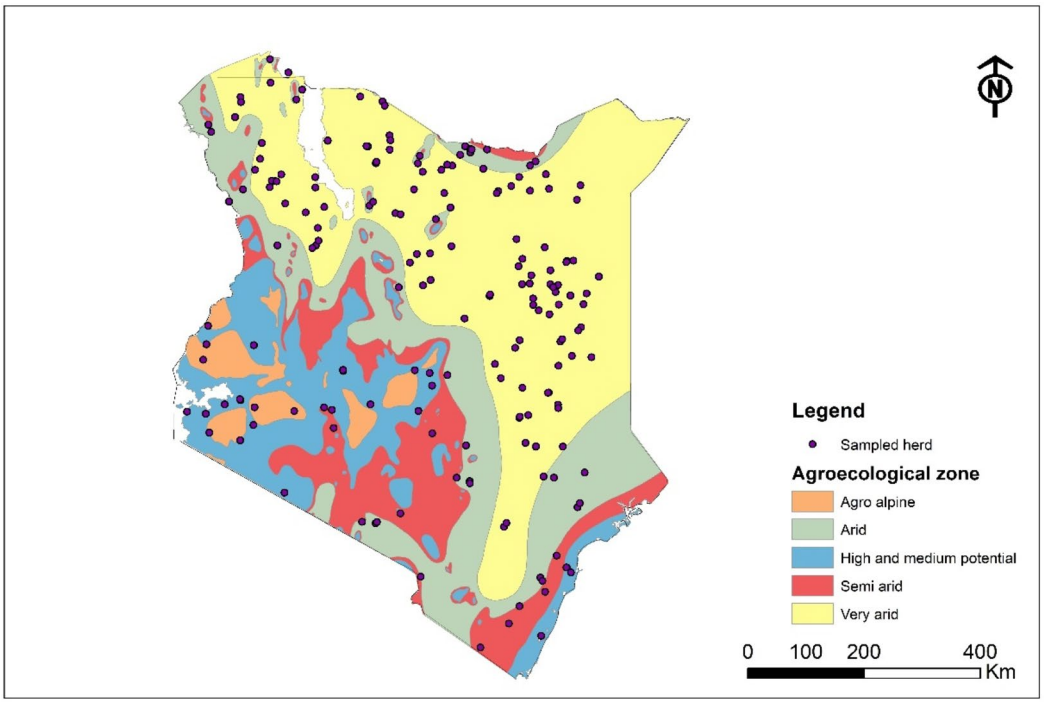

Fig. 1. Map of Kenya showing the randomly generated locations for sampling across the different agroecological zones. The map was prepared using QGIS version 3.30.1. The agroecological zone datasets were retrieved from https://geoportal.icpac.net/layers/geonode:ken_aczones (ICPAC Geoportal).

Laboratory analysis

Enzyme-linked immunosorbent assay (ELISA) was used to test the serum samples for antibodies against *C. burnetii* using the PrioCHECK Ruminant Q Fever AB Plate ELISA Kit (Applied Biosystems, Thermo Fisher Scientific). All the assays were done as per the manufacturer's instructions and all the samples were tested in duplicates. All samples with titers less than 40 were considered negative while those with titers greater than 40 were positive as recommended by the manufacturer.

Statistical analysis

Descriptive and inferential statistical analyses were implemented using the R statistical software (version 4.2.3)[37]. For descriptive analyses, mean *C. burnetii* seroprevalence and its 95% confidence interval were generated. The estimate was further stratified by all the independent variables which included age, sex, herd size, and history of reproductive syndromes captured. Univariable and multivariable modelling was implemented using the *INLA* function from the R-INLA package[38]. It is an approximate Bayesian model that is increasingly being used for disease mapping given its advanced algorithms for fitting hierarchical models, including spatial and spatio-temporal analyses. Independent variables included animal-, herd- and environmental level variables. The animal-level factors were age, sex and history of reproductive problems; the herd level variables included herd size and the herd composition (by species), while the environmental variables included soil type, digital elevation index, aridity index, spatial distribution of goats, cattle, sheep, and camels based on census data from the Department of Veterinary Services of Kenya, and predictions from the gridded livestock of the world project[24], mean annual temperature, precipitation and wind speed.

Multivariable analyses were guided by a causal web model that identified independent predictors for building the models. Data on historical reproductive disorders were classified as being endogenously correlated with *C. burnetii* exposure and therefore could not be fitted as independent predictors. Similarly, herd level variables such as herd size and production system were considered as intervening variables for environment factors in the *C. burnetii* exposure pathway. Environmental variables such as soil types, temperature and rainfall were used as antecedent factors.

C. burnetii exposure levels were assumed to be randomly distributed across the target region, with a mean and variance that could be estimated using the Gaussian Markov random process. The spatial dependence of these observations was accounted for using stochastic partial differential equations which provide an intensive approach for approximating gaussian random field. The spatial component of the model was developed over five consecutive stages. The first involved creating a mesh for indexing the spatial domain, while the second step entailed the establishment of a projector matrix that connected the observed data with the nodes within the mesh. The Kenya shape file was downloaded from https://www.diva-gis.org/gdata and used to construct the mesh. The third step involved setting up the SPDE model to capture spatial autocorrelation. The model used non-informative priors. The two subsequent stages involved the construction of data stacks for fitting the model and projecting the predictions to the spatial domain.

Two multivariable models were fitted to the data, the first prioritized animal-, herd- and environment level variables to determine risk factors for *C. burnetii* exposure, while the second used environmental variables only. The second model was specifically fitted for risk mapping purposes. This enabled the projection of predicted *C. burnetii* seroprevalence to the entire spatial domain, including the unsampled locations.

A combination of backward and forward variable selection procedures was used to identify variables that were significant in these models. A variable was considered significant if its 95% credible intervals excluded zero. Any continuous variable that was found to be significant was tested for its linearity assumption by fitting a quadratic function. The significance of the SPDE model (that was included to account for the spatial effect) was assessed deviance information criteria (DIC). The spatial component was retained in the model only if it reduced the DIC estimates.

The second multivariable model (with environmental variables) was used to predict *C. burnetii* seroprevalence across the spatial domain. The first step in this process involved the construction of a 5 km grid across the spatial domain. Centroids from the grids were then generated and used to extract environmental predictor variables from raster files. The extracted data were subsequently offered to the model for the generation of the posterior distribution of *C. burnetii* seroprevalence. The mean of this distribution was finally mapped to show the expected seroprevalence of the pathogen across the country.

Results

Descriptive analysis

A total of 6,593 cattle were sampled in 468 sampling sites or herds across 34 counties. The overall median herd size of selected herds or sites (including all the livestock species) was 63 (range; 1–3770). A large percentage of the animals sampled were from herds or sites with multiple livestock species of herds (81.6%). A median number of 14 cattle (with a range of 1–28) was sampled.

Table 1 gives the number of animals that were sampled and *C. burnetii* seroprevalence that was estimated by animal- and herd level factors. A larger proportion of the animals comprised females compared to males, adult animals compared to weaners and calves, and animals that were in arid and semi-arid areas compared to those that were sampled in other ecological regions in the country. The study found an overall *C. burnetii* seroprevalence of 7.9% (95% CI; 7.2–8.5, $n = 6593$). In general, weaners and adults had significantly higher seroprevalence estimates than calves (Table 1). Similarly, females had significantly higher seroprevalence compared to males, while cattle-only herds had higher seroprevalences compared to herds that had multiple livestock species. *C. burnetii* seroprevalence steadily increased with herd size but animals that came from large herd sizes (> 200) had low seroprevalences.

Variable	Category	Number sampled	Number positive	% Seropositive (95% CI)
Animal sex*	Male	1707	155	8.7 (7.5–10.0)
	Female	4886	336	7.0 (6.3–7.7)
Age group*	Calves	2197	92	4.2 (3.4–5.0)
	Sub-adults (weaners)	984	62	6.3 (4.9–7.7)
	Adult	3412	364	10.7 (9.7–11.7)
Herd type	Cattle only herd	130	39	30.0 (22.3–37.9)
	Cattle with other animals	338	156	46.2 (40.8–51.8)
Herd size*	< 10 animals	186	22	11.8 (7.5–16.2)
	10 to 25	66	32	48.5 (37.9–61.8)
	26 to 100	114	76	66.7 (58.8–76.0)
	100 to 199	74	60	80.6 (72.9–89.3)
	Above 200	30	7	23.3 (10.0-37.3)
Sampled regions*	Non-arid	203	36	17.7 (12.8–22.9)
	Arid	265	159	60.0 (54.3–66.4)

Table 1. *Coxiella burnetii* seroprevalence in cattle by animal- and herd level factors from the national serosurvey in Kenya. CI: confidence interval, * significant variables.

Variable	Category	Mean β (SD)	Quantile range	
			2.5%	97.25%
Animal sex*	Male	0.00		
	Female	0.56 (0.13)	0.32	0.81
Age group*	Calves	0.00		
	Weaners	0.44 (0.18)	0.08	0.79
	Adult	1.11 (0.13)	0.86	1.37
Herd type	Cattle only herd	0.00		
	Mixed herds	0.03 (0.20)	-0.363	0.43
Wind speed		0.19 (0.10)	-0.00	0.37
Altitude [a]		-0.03 (0.03)	-0.08	0.02
Annual mean temperature		0.09 (0.04)	0.00	0.17
Annual precipitation [a]		-0.10 (0.04)	-0.18	-0.01
Petric calcisols		0.39 (0.09)	0.38	0.58

Table 2. Outputs from univariable analyses of selected animal-, herd- and environmental variables. The mean and quantile ranges of the model coefficients (β) represent unadjusted log odds of *C. burnetii* exposure. [a]Variable divided by 100 to obtain perceptible parameter estimates. *Significant animal-level variables.

Up to 23.8% ($n = 1570$) of the animals were from herds that had at least one reproductive disorder including retained placenta, infertility, abortions, weak calves, and swollen testicles. A higher *C. burnetii* seroprevalence was observed in herds that reported at least one of these reproductive problems (9.9%, 8.5–11.3) compared to those that did not (7.2%, 6.5–7.9). Stratifying the observed numbers further by the syndrome reported, cattle from herds that had a history of retained placenta had a mean *C. burnetii* prevalence of 15% (11.1–19.7) compared to 7.6% (6.9–8.2) that did not report the syndrome. Similarly, cattle from herds with reported infertility had a mean *C. burnetii* seroprevalence of 14.2% (10.5–18.1) compared to 7.6% (6.9–8.2) that did not report. However, *C. burnetii* seroprevalence did not vary significantly with the other syndromes recorded, such as abortion, weak calves and swollen testis.

Univariable analysis

Table 2 illustrates results from univariable analysis. This analysis identified sex and age of an animal and annual mean temperature, annual mean precipitation and proportion of an area with petric calcisols as having crude independent associations with *C. burnetti* exposure. Older animals had a higher risk of exposure to the pathogen compared to the younger ones and herds. Similarly, higher temperatures, lower precipitation and areas increasing concentrations of petric calcisols were associated with increasing risk of *C. burnetti* exposure. In all the models used, the spatial term improved the fit of the model; models with the spatial component had consistently lower DIC estimates compared to those without.

Table 3 gives results of a multivariable model that was used to analyze animal (age and sex) and environment predictors. In general, the results show that *C. burnetti* exposure significantly (i) increased with the age of an animal, (ii) increased with wind speed, and (iii) declined with the proportion of an area under shrubs. The

Variable	Mean (β)	SD	Quantile range (β)	
			2.5%	97.5%
Intercept	-3.47	0.34	-4.13	-2.80
Age group	-1.42	0.19	-1.80	-1.04
Calf	-0.90	0.16	-1.21	-0.60
Weaner	-0.67	0.15	-0.97	-0.37
Subadult	0.00			
Adult				
Wind speed	0.24	0.10	0.05	0.42
Shrubs	-0.40	0.18	-0.76	-0.05
Petric calcisols	0.05	0.21	-0.36	0.46
Petric calcisols^2	0.14	0.07	0.01	0.27

Table 3. Outputs of a multivariable model for analysis of *C. burnetii* exposure patterns in cattle based on predictors drawn from both animal- and environmental levels. The mean and quantile ranges of the model coefficients (β) represent log odds of *C. burnetii* exposure. Marginal log-likelihood: -1659.15.

Variable	Mean (β)	SD	Quantile range (β)	
			2.5%	97.5%
Intercept	-3.76	0.34	-4.42	-3.11
Wind speed	0.22	0.10	0.03	0.41
Shrubs	-0.43	0.18	-0.77	-0.08
Petric calcisols	-0.04	0.21	-0.44	0.37
Petric calcisols^2	0.16	0.07	0.03	0.29

Table 4. Results of a multivariable model that analysed *C. burnetii* exposure in cattle based on environmental factors. The mean and quantile ranges of the model coefficients (β) represent log odds of *C. burnetii* exposure. Marginal log-likelihood: -581.66.

analysis also showed a non-linear association between *C. burnetti* exposure and the proportion of an area that had petric calcisols. Further analysis of the quadratic term suggests that an increase in the area under petric calcisols led to an exponential increase in the log odds of *C. burnetti* exposure. The random effect that was used to account for spatial autocorrelation was also significant; respectively, the DIC estimates for the models with and without the spatial terms were 3149.70 and 3469.65.

Table 4 provides outputs of a final multivariable model that used environmental factors to predict *C. burnetii* exposure. Wind speed, shrubs and petric calcisols type of soil were retained as the main environmental predictors for *C. burnetii* exposure and the nature of their association with the outcome (positive or negative) did not change. Slight differences in the values of their parameter estimates, as expected, was noted. of. The spatial effect also improved the fit of the model as the DIC estimates with and without the spatial effect were 3239.49 and 3556.97, respectively.

Figure 2 gives a *C. burnetii* seroprevalence map that was developed using the mean and 2.5% and 97.5% quantile values of the posterior distribution from the model presented in Table 4. The entire northern and eastern regions and parts of southern regions associated with semi-arid, or arid agro-ecological zones were predicted to have a high to moderate prevalence of the pathogen. However, the Western regions and the Central highlands that receive moderate to high levels of precipitation showed low seroprevalences (Fig. 2).

Discussion

This study analysed the distribution of *C. burnetii* in Kenya based on a national serosurvey of cattle, serological analysis of samples collected and analysis of data using spatial models. The findings revealed that *C. burnetii* exposure in cattle is associated with animal (age), herd (herd size) and area (meteorological and geographic) variables.

Our investigation revealed a national prevalence of 7.9%. This closely mirrors a previous estimate of 7.4% that was reported through a systematic review of literature on *C. burnetii* in cattle in Kenya[16]. Other studies conducted in smaller geographical areas across Kenya showed diverse estimates of 89.7%, 13%, 10.5% and 3%[3,39–41]. These estimates vary largely with agro-ecological zones where the studies were implemented.

At the individual level, the age of an animal was a significant determinant of risk for *C. burnetii* exposure. Our results were concurrent with previous studies that have demonstrated an increase in seroprevalence with age of cattle[17,42,43]. The increase of seroprevalence with age may be due to the multiple and possibly persistent exposure of cattle to *C. burnetii* from birth through to adulthood. An infected cattle can shed the bacteria in

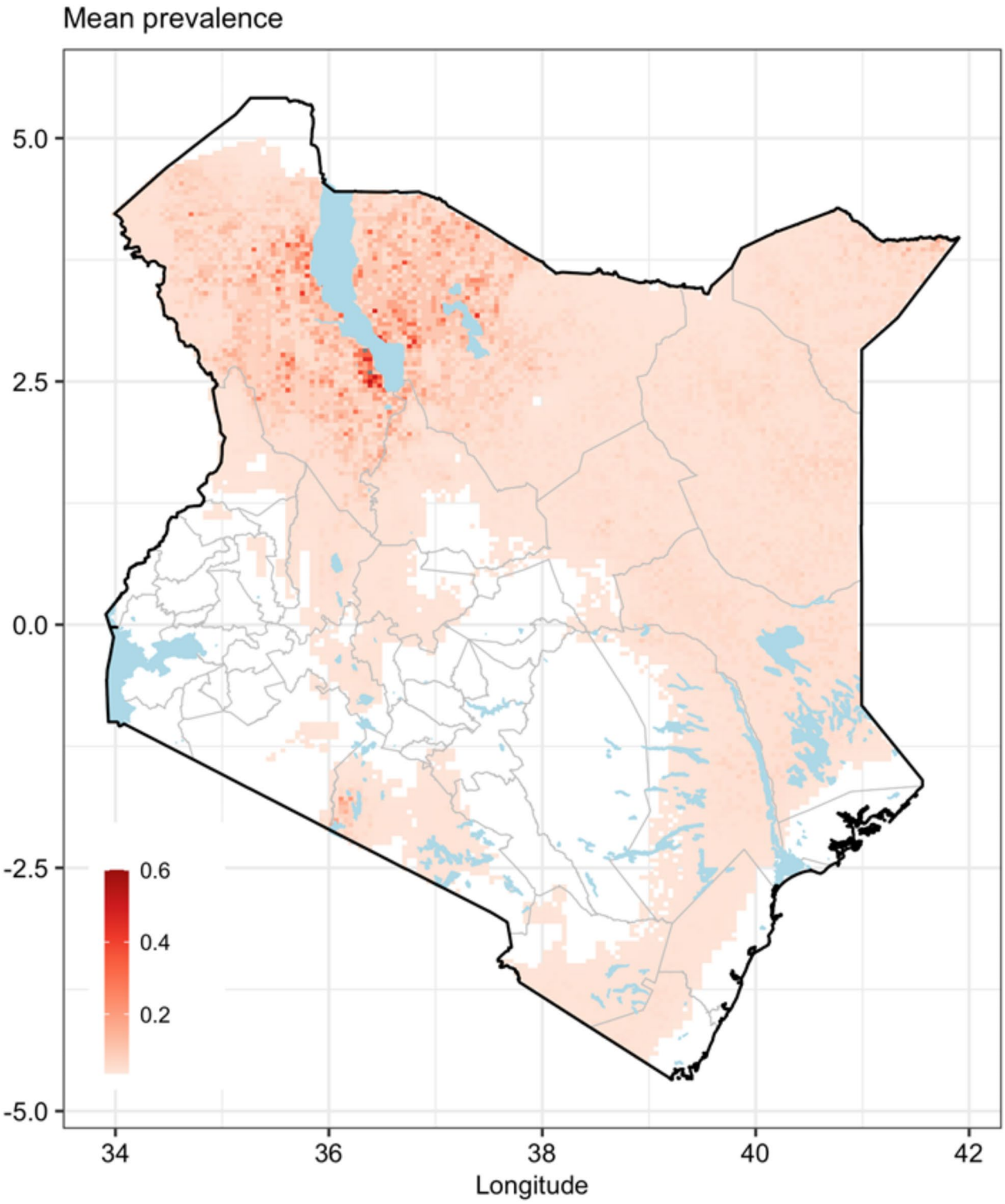

Fig. 2. Predicted *C. burnetii* seroprevalence in Kenya based on a multivariable model that analysed *C. burnetii* exposure in cattle based on environment factors.

parturient fluids, vaginal mucus, milk, and faeces for extended periods coupled with the ability of the bacteria itself to persist in contaminated environments[7,44].

We detected a crude association between reproductive disorders and the presence of *C. burnetii*. Our findings indicate a positive association of *C. burnetii* exposure in herds with a history of reproductive disorders including history of still births, abortion, retained placenta. This aligns with some conclusions from earlier studies, although some variations were noted[5,45,46]. The extent to which *C. burnetii* impacts reproductive disorders in cattle

remains debatable. From the descriptive analysis, individual reproductive problems, i.e., infertility, abortion, weak calves, and retained placenta, were not significant risk factors. It is important therefore to consider multiple reproductive disorders as being indicators for *C. burnetii* exposure in cattle[43,46,47].

At the population or herd-level, we observed that the risk of *C. burnetii* seropositivity was highest in large cattle herds (with 26–99, and 100–199 animals), corroborating the findings of previous studies[42,46,48], which established association between herd size and increased risk of *C. burnetii* exposure. The study by Sadiki et al.[46] demonstrated that herds of > 150 cattle had higher levels of *C. burnetii* shedding and seropositivity, while the study by Agger and Paul, showed 18 times higher odds of testing positive for *C. burnetii* in herds of > 150 cattle, relative to smaller herds[49]. The positive association between herd size and *C. burnetii* positivity could suggest that the pathogen gets transmitted much more efficiently in bigger herds where more frequent contacts between animals can be achieved[23,27,48]. Large herds were found in the pastoral arid and semi-arid areas where communal grazing and watering was routinely practiced.

Our analysis revealed that spatial distribution of *C. burnetii* in Kenya is associated with environmental factors, mainly soil type and wind speed. Petric calcisols are characterized by shallow soils with fine-medium texture that may aid formation of dust, thereby facilitating the spread of *C. burnetii* aerosols from contaminated farms or herds[50,51]. It is prevalent in the northwestern region of Kenya and parts of the southeastern and southwestern coast of the country that were associated with highest risk of *C. burnetii* exposure. Wind speed, on the other hand, is a good predictor for *C. burnetii* given that it aids the dispersals of aerosols and the free living small variant forms of the pathogen. During parturition for example, the dispersal of many infective particles of *C. burnetii* can be transmitted between animals much more readily in areas that experience higher windy conditions. Windy conditions can also increase the amount of dust in air, which can further facilitate the spread of *C. burnetii* in environments where the bacterium is present.

This current national serosurvey and risk map of *C. burnetii* in Kenya brings to the fore several considerations for the pathogen in Kenya. Firstly, the findings confirm the endemicity of *C. burnetii* in cattle across Kenya, while highlighting potential hotspots in the northwestern, southeastern, and coastal areas of the country. This necessitates the development of surveillance and control measures, especially in the hotspots to avert the potential outbreaks of Q fever. An inactivated *C. burnetii* vaccine is only available in parts of Europe for use in animals[52] and current vaccine for use in humans is faced with limitations in safety and effectiveness[53]. None of these vaccines are available or in use in Kenya.

The other major finding is the establishment of the environmental factors that are associated with *C. burnetii*. The findings obtained corroborate previous studies on the potential contributions of soil types and wind speed on *C. burnetii* risk. They also provide critical evidence that can be used, not only to design One Health research and control of the disease but also to quantify the potential benefits of environmental conservation practices that involve reinstatement of vegetation in denuded areas. We hypothesize that replacement of vegetation cover in *C. burnetii* hotspots would reduce aerosolization of infection particles by checking wind flow and formation of dust plumes. We also argue that climate factors including increased frequency of droughts and sporadic rains, which are becoming more pronounced in the pastoral areas in the Horn of Africa, are likely to expand the spatial range of the pathogen.

When the results of this study are interpreted together with published studies that show that *C. burnetii* is more prevalent in goats, camels and wild animals in the giraffidae family compared to the other animal raised in the same environments[19], a better understanding on the components of the pathogen's ecological niche is realized, albeit with several unanswered questions. Even though the current study used cattle to determine the spatial distribution of the pathogen, the other animals that have been associated with high *C. burnetii* exposure levels in previous studies are well adapted to the harsh environments which were identified as being the hot spots for the pathogen.

This study had some limitations. The investigations were based on serological analysis done on cattle only. It can be argued however that any biases that could have arisen from using only one livestock species in the study can be classified into the non-differential misclassification errors that often biases the observed statistical tests to the null. The reported measures of association are therefore conservative. The role of ecological and climatic factors may be further strengthened by analysis of environmental samples such as soil, water, and air, which were beyond the scope of the current study. Molecular studies are still required to elucidate the genotypic characteristics of *C. burnetii* in the country to contribute towards understanding the genetic epidemiology of *C. burnetii* in Kenya.

Conclusion

The present study contributes to the nation-wide understanding of *C. burnetii* epidemiology in Kenya. By undertaking national sero-survey of *C. burnetii* in cattle, followed by geospatial modelling, we demonstrate that *C. burnetii* is endemic in Kenya, with specific hotspots in the northwestern, southeastern, and coastal areas of the country. The current risk map demonstrates the epidemiological complexity of *C. burnetii*, in which risk profile is dictated by an array of factors including age of animal, herd size, as well as environmental factors including soil type and wind speed. These findings provide a basis for directing financial and human resources to focus interventions on the hotspots including biosurveillance, risk communication, and risk management. This study also highlights the collective advantage of a One Health approach in the prevention and control of Q fever in Kenya.

Data availability

All the data are included in this article. Upon reasonable request, the raw datasets are available from the corresponding authors.

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Concept development; J.A., B.B., M.M., A.M., J.G., R.N., Formal analysis; J.A., B.B., D.N., A.B., E.M., A.B., Funding acquisition; B.B., A.M., K.N., J.F. Investigation; J.A., A.M., M.M., L.W., J.G., K.L., R.N., B.B., E.K.,L.K.,H.A., Methodology; J.A., B.B., J.G., A.M., M.M., F.G., N.K., L.W., R.N.,H.A., Supervision; B.B., K.N., J.F., F.G., J.A., Writing original draft; J.A., L.W., B.B., A.M., M.M., E.M., H.A., Writing review & editing; All Authors. All the authors have read and approved the manuscript.

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Declarations

Competing interests

The authors declare no competing interests.

Additional information

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