REVIEW ARTICLE

A systematic review of Rift Valley Fever epidemiology 1931–2014

Mark O. Nanyingi, BVM, MSc[1,2,3]*, Peninah Munyua, BVM, MSc, PhD[4],
Stephen G. Kiama, BVM, MSc, PhD[5], Gerald M. Muchemi, BVM, MSc, PhD[2],
Samuel M. Thumbi, BVM, MSc, PhD[3,6], Austine O. Bitek, BVM, MSc[7,8],
Bernard Bett, BVM, MSc, PhD[9], Reese M. Muriithi, BVM, MSc[8] and
M. Kariuki Njenga, BVM, PhD[3,6]

[1]Department of Biomedical Sciences, Colorado State University, Fort Collins, CO, USA; [2]Department of Public Health, Pharmacology and Toxicology, University of Nairobi, Nairobi, Kenya; [3]Kenya Medical Research Institute, Nairobi, Kenya; [4]Centers for Disease Control and Prevention, Nairobi, Kenya; [5]Wangari Maathai Institute for Peace and Environmental Studies, University of Nairobi, Nairobi, Kenya; [6]Paul G. Allen School for Global Animal Health, Washington State University, Pullman, WA, USA; [7]Zoonotic Disease Unit, Nairobi, Kenya; [8]Directorate of Veterinary Service, Nairobi, Kenya; [9]International Livestock Research Institute, Nairobi, Kenya

Background: Rift Valley Fever (RVF) is a mosquito-borne viral zoonosis that was first isolated and characterized in 1931 in Kenya. RVF outbreaks have resulted in significant losses through human illness and deaths, high livestock abortions and deaths. This report provides an overview on epidemiology of RVF including ecology, molecular diversity spatiotemporal analysis, and predictive risk modeling.

Methodology: Using the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines, we systematically searched for relevant RVF publications in repositories of the World Health Organization Library and Information Networks for Knowledge (WHOLIS), U.S Centers for Disease Control and Prevention (CDC), and Food and Agricultural Organization (FAO). Detailed searches were performed in Google Scholar, SpringerLink, and PubMed databases and included conference proceedings and books published from 1931 up to 31st January 2015.

Results and discussion: A total of 84 studies were included in this review; majority (50%) reported on common human and animal risk factors that included consumption of animal products, contact with infected animals and residing in low altitude areas associated with favorable climatic and ecological conditions for vector emergence. A total of 14 (16%) of the publications described RVF progressive spatial and temporal distribution and the use of risk modeling for timely prediction of imminent outbreaks. Using distribution maps, we illustrated the gradual spread and geographical extent of disease; we also estimated the disease burden using aggregate human mortalities and cumulative outbreak periods for endemic regions.

Conclusion: This review outlines common risk factors for RVF infections over wider geographical areas; it also emphasizes the role of spatial models in predicting RVF enzootics. It, therefore, explains RVF epidemiological status that may be used for design of targeted surveillance and control programs in endemic countries.

Keywords: *Rift Valley Fever*; *spatiotemporal*; *modeling*; *epidemiology*

Responsible Editor: Åke Lundkvist, Uppsala University, Sweden.

*Correspondence to: Mark O. Nanyingi, Kenya Medical Research Institute, Nairobi, Kenya, Email: mnanyingi@kemricdc.org

Rift Valley Fever (RVF) is an arthropod-borne viral zoonosis with evidence of widespread occurrence in humans and animals in Africa and the Arabian Peninsula. Major epidemics have been reported most notably in Egypt (1977, 2003), Kenya (1997–1998, 2006–2007), Tanzania (2007), Somalia (2007), Saudi Arabia and Yemen (2000–2001), Sudan (2007), Mayotte (2008), and Mauritania (2010, 2012) (1–11). The RVF virus (RVFV) is a Phlebovirus belonging to the *Bunyaviridae* family of viruses (12). RVFV has been isolated from over 30 species of mosquitoes in six genera (13, 14). RVF outbreaks are associated with the occurrence of the warm phase of the El Niño/Southern Oscillation (ENSO) phenomenon causing floods, increased greenness of vegetation index, and emergence of mosquito vectors that infect susceptible ruminant hosts (15–17). According to the

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World Organization for Animal Health (OIE), RVFV is an OIE high-impact transboundary pathogen with potential for bioterrorism and a setback to international livestock trade (18). Here, we present a comprehensive review that provides an update on RVF with a focus on understanding the epidemiology of RVF, including ecology and risk factors as well as molecular diversity, spatiotemporal epidemiology, and risk modeling for disease endemic regions.

Methodology

Search strategy
Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) (19) guidelines were used to search for published literature from 1931 up to 31st January 2015 in PubMed, SpringerLink, WHOLIS, Food and Agricultural Organization (FAO), and U.S Centers for Disease Control and Prevention (CDC) databases. To maximize the completeness of the search and reduce selection bias, the search was restricted to English articles using the medical subject heading (MESH) search terms "rift

valley fever" AND "epidemiology" OR "spatio-temporal" OR "modelling". A generalized search was performed using Google Scholar to identify relevant documents not published in peer-reviewed journals using similar terms as above. During the initial search, studies were selected based on a review of titles and abstracts. All abstracts identified from the indexed databases were screened for eligibility, and the full text of relevant articles was reviewed. Review articles on RVF were examined to identify non-indexed articles fitting the eligibility criteria (Fig. 1).

Eligibility and inclusion criteria
Studies included in this review described replicable findings on the epidemiology and geographical extent of RVF, molecular and genetic diversity, human and animal risk factors associated with ecological and climatic conditions, and spatiotemporal and predictive risk modeling for RVF.

Exclusion criteria
We excluded RVF reports on sero-epidemiological surveys that reported negative results and risk factor studies
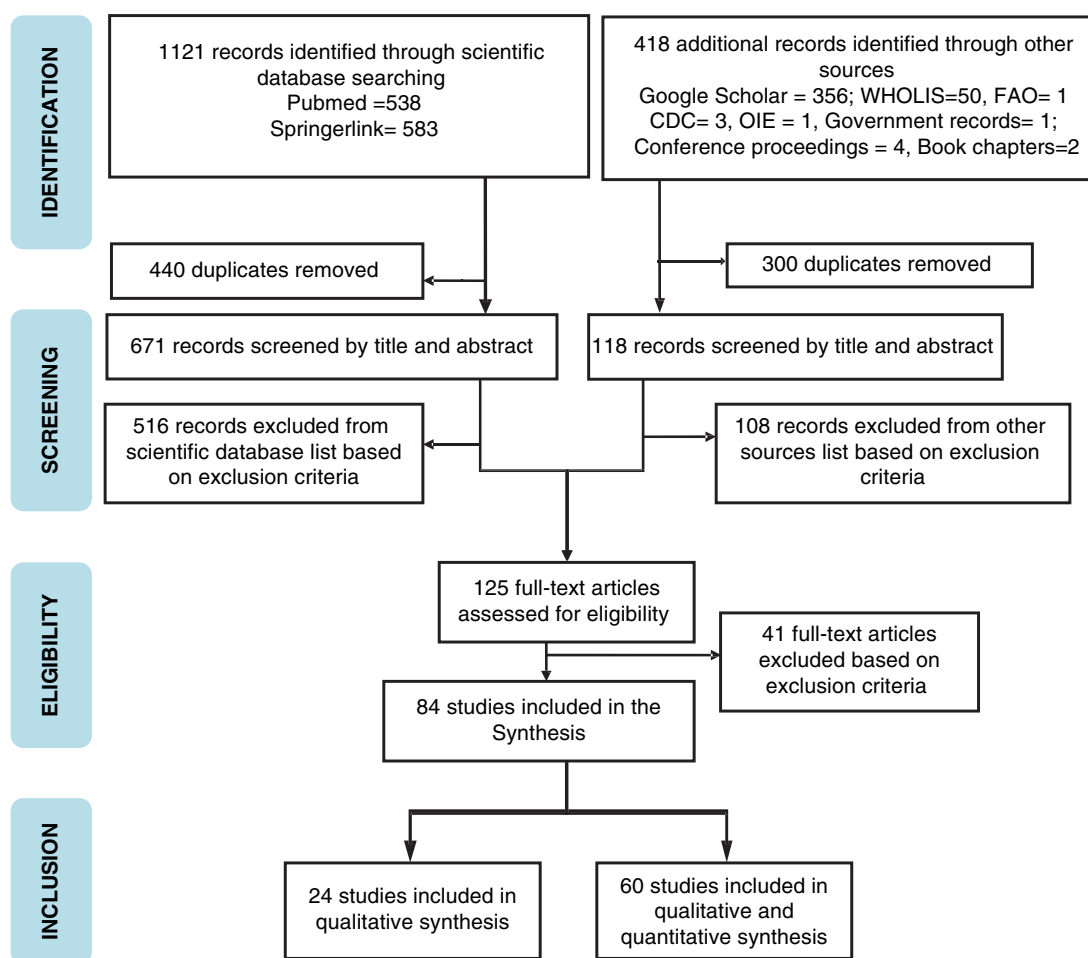

Fig. 1. PRISMA flow chart diagram describing the studies selection process for inclusion in this review [adapted and modified from (19)].

that had no clear case definition or that had an extremely low sample size (<100). We developed a specific criteria based on subject matter expertise which excluded secondary reports, editorial opinions, personal communications, and studies published in scientific conferences that were purely descriptive with no quantitative or qualitative inferences, as illustrated in Fig. 1.

Results

The data
Using the key search terms, 1121 records were retrieved from indexed scientific databases [PubMed (538) and SpringerLink (583)], while 418 non-indexed reports were obtained from generalized searches in Google Scholar (356), WHOLIS (50), FAO/CDC (7), government reports (1), and conference proceedings (4). All records were imported in to Microsoft Excel, and articles presenting duplicate titles/findings were removed to obtain 800 records. Further screening was done by title and abstract focusing on studies reporting the epidemiology of RVF including ecology and risk factors as well as molecular diversity, spatial–temporal epidemiology, and risk modeling. Six hundred and seventy five articles (675) were eliminated based on the general exclusion criteria to remain with a subset of 125 publications that were assessed for eligibility by reading the full text. Furthermore, 41 articles were removed by the subject matter exclusion criteria; limiting this review to 84 publications where 60 had quantitative and qualitative inferences, while 24 had qualitative but replicable findings (Fig. 1).

Maps: spatiotemporal distribution of RVF occurrence
The descriptive geography of the epizootic or endemic status of RVFV was illustrated using GIS software (ArcView® 10.2.2) to produce distribution maps (Figs. 2 and 3). The input data were extracted from the relevant cited publications. The aggregate number of outbreaks in months as reported at country level was confirmed by clinical diagnosis or serological evidence in livestock and humans. From available records, we calculated the cumulative human case fatalities in 13 African and two Arabian countries from 1977 to 2012. More details are available in Fig. 2.

The epidemic focus of RVFV was contained in Kenya in 1912 (20) until three decades later, before spreading to neighboring Tanzania in the late 1940s (21). Intensive outbreaks in Southern Africa in the 1950s were evident in South Africa, Namibia, Zimbabwe, and Zambia forming secondary epidemic foci (22) (Fig. 2). The proximity of the Arabian Peninsula to the Horn of Africa and associated livestock trade may be responsible for the geographical spread of the virus to Saudi Arabia and Yemen (23) as indicated in Fig. 2.

An epidemiologic shift was evident in the West African pocket in the late 1980s, and may have been associated with climate variability, leading to aggressive emergence and dispersal of competent mosquito vectors of *Aedes* and *Culex* species to the large ruminant populations in Mauritania and Senegal. Southern Africa and West Africa have reported the longest RVF outbreak periods with South Africa and Mauritania (24) sustaining longer outbreaks compared to the rest of the Horn of Africa as detailed in Fig. 3.

Epidemiology, ecology and risk factors for RVF occurrence
RVF epizootics and epidemics in livestock and humans have occurred periodically with the initial geographic range restricted to sub-Saharan Africa, but since 2000 it has spread to the Arabian Peninsula. An enzootic hepatitis in sheep was observed as early as 1912, but the first clinical report was among sheep, cattle, and humans in areas near Lake Naivasha in Kenya in 1930 (25). Since that time, recurrent epidemics have been reported for the last 60 years in South Africa (26), Zimbabwe (27), Mauritania (9–11), Senegal (28), Zambia, Namibia, Gabon, Burkina Faso, Madagascar, East Africa, and more recently in Mayotte, Yemen, and Saudi Arabia (1–7, 29, 30) (Fig. 2 and Table 1).

The South African epizootic of 1951 led to deaths of over 100,000 sheep and half a million livestock abortions (25). In Egypt, a notable human epidemic was reported in 1977 causing an estimated 600 deaths and significant livestock abortions and mortalities. This first report of RVF in Egypt may have been associated with introduction of the virus through livestock trade from the Horn of Africa and aggressive emergence of mosquitoes from the flooded Nile River (32). An additional 45 RVF cases were reported in farmers in Seedy Salim district, where up to 17 human deaths were confirmed in the Egyptian Kafr Al-Sheikh Governorate (33).

A retrospective cohort study conducted in 1989 in Senegal assessed risk factors among 273 people aged >5 years. Increased seropositivity was associated with advanced age. Nursing sick people while being in contact with sick animals had a six-fold risk of infection, and male animal attendants had five-fold risk of contracting RVF as compared to females (34) (Table 1). In the Horn of Africa, epidemics have been closely associated with El Niño-related flooding resulting in a large outbreak in 1997–1998 that led to thousands of livestock deaths and estimated 500 human deaths (2, 35). In 2000, the first RVF outbreak in Saudi Arabia was investigated among 800 patients with a case fatality rate of about 14%. RVF IgM, detected in >50% persons, was indicative of active infection. The majority of cases were males over 40 years old (36). During the same period, another prospective study reported mortalities of up to 34% in 165 seropositive patients (37) (Fig. 2 and Table 1).
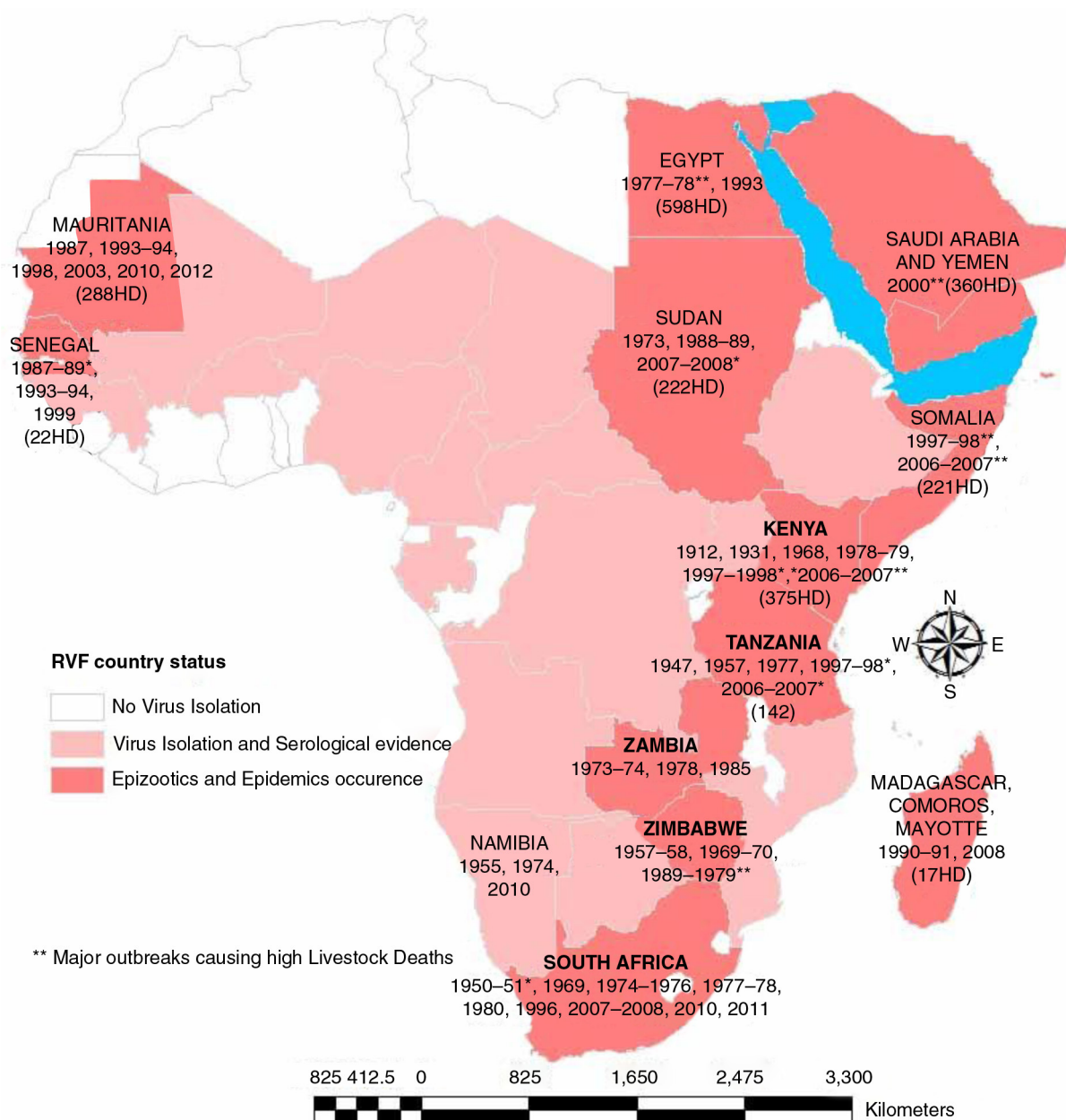
Fig. 2. Map of Africa and Arabian Peninsula illustrating the spatial and temporal distribution of Rift Valley status from the first suspected case in 1912. Total number of human deaths (HD) is indicated for selected countries for all outbreak periods. Based on (2, 5–7, 10–12, 14, 16, 22, 23, 25, 40, 46, 54, 75, 80).

In 2006–2007 the outbreak that affected Sudan, Kenya, Somalia, and Tanzania led to substantial losses of livestock and over 900 human deaths (6, 38–41). In 2000, sustained heavy rainfall in the Arabian Peninsula led to flooding and the first outbreak of RVF in Saudi Arabia and Yemen (42), which resulted in over 200,000 human infections with an estimated 250 human deaths and thousands of livestock deaths (5, 43, 44) (Fig. 2 and Table 1).

In East Africa, high-risk areas for RVFV activity have been identified based on ecological receptiveness for the vector, historical presence of virus or proximity to known infected areas, and areas experiencing increased rainfall and flooding. High incidence has been reported in areas having soil with poor drainage and flat landforms with low altitudes below 500 m (21, 45, 46). Human and animal prevalence studies focus on description of risk factors for outbreak using OR as a measure of association of factors in prevalence studies. Most of the studies examined human–animal contact or consumption of animal products (47). After the 2006–2007 RVF outbreak in Kenya, a randomized household cluster survey was conducted in two RVF outbreak foci areas – one among 248 individuals
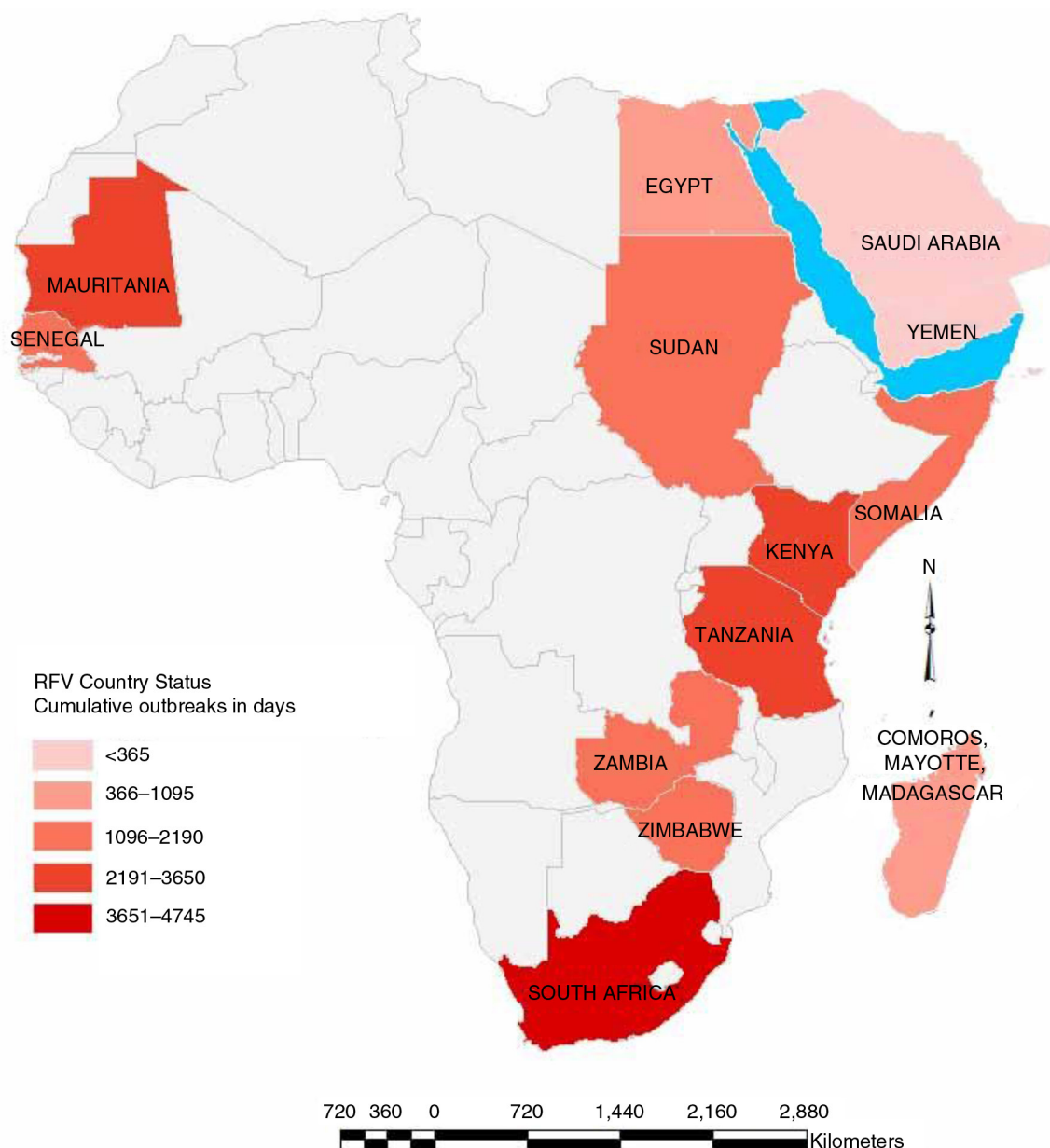
Fig. 3. Map of Africa and the Arabian Peninsula illustrating the spatial and temporal distribution of Rift Valley cumulative outbreaks in days from 1977 to 2012 (2, 5–7, 10–12, 22, 23, 25, 40, 46, 80).

and another population-based serological survey in three foci among 861 individuals in over 400 households (39, 48).

Both studies reported higher infection with RVF among male herders, and common risk factors were touching or disposing aborted fetuses or being exposed to mosquitoes. Other risk factors include consuming or handling products from sick animals, contact with livestock as herdsmen and handling of aborted fetuses, milking, skinning, slaughtering, sleeping with animals, touching blood, and caring for animals during birthing (39, 48).

Movement of livestock during the viremic phase of infection to areas with high mosquito density and naïve livestock populations poses a high risk of human infection especially among persons handling livestock (39, 49). A multispecies sero-epidemiological survey was conducted in the Sahrawi territory which is a cluster of refugee camps in Tindouf province of Algeria. Less than 1% of the 982 ruminant samples tested positive for IgG antibodies against RVFV with two clusters of high seroprevalence in Mehaires (7.14%) and Tifariti (7.69%) regions. The proximity of this region to RVF endemic countries like Mauritania and Senegal that engage in large livestock trade may have been responsible for the frequency of RVF outbreaks in 2008, where goats and older animals had higher seropositivity (50).

Table 1. Characteristics of the eligible studies investigating RVF epidemiology, ecology, risk factors, and spatial modeling 1931–2012

| | | | | Morbidity and mortality estimates | | | | | |
| | | | | Animals | | Humans | | | |
Year(s)	Geographic distribution	Study design	Research questions/objectives	Reported cases	Deaths	Reported cases	Deaths	Estimated impact in US$ ($\times 10^6$)	References
1931	Kenya	Cross-sectional	Risk factors and ecology	nd	4,700	nd	nd	nd	(25)
1950–1951	South Africa	Cross-sectional	Epidemiology and spatial modeling	600,000	100,000	nd	nd	nd	(22, 26, 31)
1977–1978	Egypt	Cross-sectional	Epidemiology and socioeconomics	nd	nd	200,000	598	115	(1, 32, 81)
1978	Zimbabwe	Cross-sectional	Risk factors	70,000	10,000	nd	nd	nd	(27, 31)
1988	Mauritania	Case–control	Risk factors	nd	nd	nd	224	nd	(9)
1987–1989	Senegal	Cohort	Molecular epidemiology	1,715	nd	273	16	nd	(28, 34)
1997–1998	Kenya	Cross-sectional	Ecology, risk factors and socioeconomics	89,000	478	160,000	450	250^c	(2, 35, 82)
	Somalia			nd	nd	28,000	170	378^c	
	Tanzania			nd	nd	89,000	478	nd	
1998	Mauritania	Cross-sectional	Sero-epidemiology, entomology and virology	343	nd	90	1		(30)
2000	Saudi Arabia	Cross-sectional	Risk factors and ecology	>10,000	1,000	883	245	10	(36, 37)
2000–2001	Yemeni	Cross-sectional	Risk factors and socioeconomics	22,000	6,000	1,328	166	107	(5, 42, 81)
2003	Egypt	Cross-sectional	Risk factors and virology	nd	nd	45	17	nd	(33)
2007–2008	Sudan	Cross-sectional	Risk factors and ecology	nd	nd	75,000* (698)	222	nd	(6, 79, 80)
2008–2009	Madagascar	Cross-sectional	Epidemiology and socioeconomics	nd	nd	10,000 (712)	26	nd	(84)
2006–2007	Somalia	Cross-sectional	Risk factors, predictive modeling and ecology	nd	nd	35,000* (114)	51	541^c	(21, 29, 39, 40, 48, 49, 52)
	Tanzania			32,000	4,200	40,000* (264)	109		
	Kenya			nd	nd	75,000* (684)	158		
2010–2011	South Africa	Cross-sectional	Spatial and predictive modeling	14,342	8,877	242	26	nd	(22, 75, 84)
2012	Mauritania	Cross-sectional	Risk factors, molecular diversity	nd	343	41	17	nd	(8, 10, 11)

nd = no documented estimates; c = combined estimates.
*Estimated cases in brackets are the reported.

In 2007–2008, a large cross-sectional seroprevalence survey among 17,000 people in Tanzania revealed a higher seroprevalence of 29.3% in persons living near water bodies. Seropositivity was also associated with increased age, owning livestock, and poverty. It further showed a high correlation of increased risk in areas with dense vegetation, hence, favoring mosquito emergence and large cattle populations (51). Similarly a clinical epidemiological study of 511 RVF suspect cases in Tanzania during the 2007 outbreak revealed major signs of infection such as fever, encephalopathy, retinopathy, and hemorrhages with a high case fatality rate of about 30% in 186 laboratory-confirmed cases. Increased infection was highly associated with contact or consumption of foods of animal origin (52) (Table 1).

In 2011, a random cross-sectional survey was conducted on about 1600 livestock in Kilombero Valley, Tanzania. It showed that younger animals born after the 2007 outbreak had a lower prevalence (5.5%) compared to older animals (> 22.7%), but a large difference was observed in female animals having a three-fold increased risk compared to male animals (53). Grazing within 5 km of water bodies had little influence on the presence of antibodies compared to animals grazing > 15 km from water bodies; this is in contrast to the expected increase in vector density at breeding sites where hosts aggregate. However, a related human study in Mbeya region of Tanzania reported a higher correlation between altitude and increased seroprevalence among 1,228 residents (51).

Despite the proximity of Uganda to Kenya and Tanzania, RVF outbreaks have never been reported in Uganda in either humans or animals. Magona et al. (54) tested 2,700 European and local goat breeds in 30 farms across four Ugandan districts and recorded a 10% seroprevalence of antiRVF IgG antibodies. Similarly in Djibouti located in the Horn of Africa, Andayi et al. (55) assessed the risk factors and sero-epidemiology of arbovirus and detected an RVF prevalence of 2.2% in 1,000 humans. This confirms the presence of subclinical virus circulation in non-epidemic areas and calls for continued virus and vector surveillance in anticipation of outbreaks.

In Mayotte, a risk factor assessment conducted in 2011 among 1,420 individuals and 198 seronegative ruminants in 33 herds (56) found that high human seroprevalence was associated with increased age (≥ 15 years), being male, proximity to water bodies, farming, and low education levels. While animal risk factors were similar to reports in East Africa, a major risk factor for both humans and animals was exposure to competent mosquito vectors (56). The 2012 outbreak in southern Mauritania reported 17 human deaths among 41 confirmed cases (10, 11). Phylogenetic analysis indicated that outbreaks may have remerged from enzootic foci with isolates having a close relationship with the strains responsible for the 2010 outbreak in the northern part of Mauritania (8, 10).

Analysis of ecological factors associated with RVF infection in Gabon from 2005 to 2007 was conducted in 4,323 individuals covering 10% of national households. Despite the lower overall seroprevalence (3.3%), individuals living around the lake region had higher seropositivity (8.3%) than those residing in forested and savannah areas (2.9 and 2.2%, respectively). This study reaffirms the role played by water bodies leading to rapid turnover of competent vectors (57) to establish RVF endemic status as also evidenced in Mbeya, Tanzania (51). The influence of sex on RVF infection rates was described by a hospital based case control study that was conducted on 290 febrile patients during the 2007 outbreak in Sudan. One hundred and twenty two (82%) of 149 patients were RVFV IgG seropositive with a reported three-fold risk of being seropositive for males (OR = 2.8, 95% CI = 1.0–7.6) (58).

In Saudi Arabia, a seroprevalence survey of 275 small ruminants examined the contribution of environmental and animal risk factors to RVF outbreaks and demonstrated a positive association of disease occurrence and increased precipitation (OR = 2), presence of water bodies (OR = 2.2), high vector density (OR = 4.2), with high disease occurrence (59); the findings were closely related to other studies in Gabon and Tanzania (51, 57). Caminade et al. (60) investigated the relationship between rainfall and greening vegetation triggering RVF outbreaks in Mauritania from 1990 to 2012. Whereas intra-season variability of weeklong rainless periods, then heavy precipitation was observed to be critical in the onset of outbreaks, this finding was contrary to the effect of total seasonal levels precipitation or normalized differential vegetative index (NDVI) responsible for East African outbreaks (15, 61).

Molecular epidemiology and genetic diversity of RVFV

Phylogenetic analysis of samples isolated from 1944 to 2000 in 10 African countries and Saudi Arabia revealed that seven main viral lineages were categorized as A, B, C, D, E, F, and G among the 33 viruses (62). Despite the A–D lineages existing in most African countries, enzootic circulation was consistent in Central Africa. There is potential of virus transportation outside endemic regions which may have been associated with the outbreak in Egypt (1977–1979) due to introduction of genotypes from endemic sub-Saharan Africa. Five more lineages (B, F, H, I, and M) were described; this high variability may be due introduction of virus by the live Smithburn vaccines used to combat the outbreaks of South Africa (1951–1968) and Zimbabwe (1969–1970) (63).

There is evidence of widespread circulation of multiple lineages B, C, K, and L, with C being isolated from outbreaks in Zimbabwe (1978), Madagascar (1991), Horn of Africa 1997–1998, Saudi Arabia (2000–2001), Kenya (2007), and South Africa (2008–2009). However, the

genetic similarity in isolates of 1977, 1983, and the 1997–1998, 2006–2007 in Kenya may indicate virus introduction through vaccination using live vaccines (63). Genetic analysis of S, M, and L segments of RVF isolates from the Arabian Peninsula outbreak in 2001 indicated striking similarity to isolates associated with RVF epidemics in Kenya (1997) and Madagascar (1991). This may rule out the genetic reassortment associated with the emergence of RVFV in the Arabian Peninsula but suggest concurrent virus circulation and genetic evolution of during the inter-epidemic period (64). Phylogenetic analysis of 2007 and 2010 outbreak samples from Sudan revealed a closer lineage with the Kenyan RVFV variants during the 2006–2007 outbreak, suggesting a common ancestry and multiple viral introductions from East Africa (65).

Common genetic ancestry was demonstrated by Faye et al. (66) from human and animal outbreak strains in Mauritania (2003) whose phylogenetic lineages were similar to isolates from Madagascar (1991), Kenya (1997), Chad (2001), and Saudi Arabia (2001). Whole genome sequencing using 12 strains of viruses isolated for over 50 years (1944–2001) from six African countries and Saudi Arabia indicated that *Aedes vexans arabiensis* was responsible for the 2000 in Saudi Arabia. These cases occurred after heavy rainfall, hence, agreeing with East African dynamics of disease emergence based on favorable ecological variables for vector emergence (66, 67). Genomic analysis of over 3,000 animal specimens collected in Kenya during the 2006–2007 outbreak demonstrated concurrent circulation and genetic reassortment of multiple virus lineages in 31 RVFV isolates. The 2006/2007 outbreak viruses had the same ancestry as the 1997/1998 outbreak strains. The coverage of all genome segments was confirmed by serology and, therefore, confirms enzootic circulation and wider geographical distribution of similar RVFV strains (38).

In West Africa, phylodynamics of RVFV has been evaluated by Bayesian models using 48 RVFV isolates collected over 65 years from 18 sites in Senegal and Mauritania, and 15 other countries (24). They demonstrated a geographic pattern with high temporal coherence matching the earlier reported cases in East Africa. There were five distinct introductions routes in Senegal and Mauritania, stretching from South Africa, Zimbabwe, hence, confirming the serological and entomological surveys that Barkedji coastal area of West Africa may be a vital entry point of RVFV in Senegal and Mauritania (24).

In East Africa, a whole genome phylogenetic analysis of 16 RVF viruses isolated from humans, livestock, and mosquitoes during the 2006–2007 outbreak revealed three distinct lineages, which in comparison had similarity to the Kenyan isolates of 1980, 1998 and the Saudi Arabia isolate of 2000 but had no genetic similarity with other isolates including Entebbe 44 and Kenya 1965 (68). This strongly suggest the possibility of focal 'de-novo' reemergence of viruses and spontaneous release of resident virus maintained inter-epidemically in desiccated *Aedes* eggs which hatch during flooding.

During the Kenyan 2006–2007 RVF outbreak, virus isolates were analyzed for genetic diversity in three outbreak regions. The eight human isolates had up to 99.6% nucleotide sequence identity with each other across the M segment of the genome. They had high homology with the RVF strains involved in the 1996–1997 RVF outbreak in Kenya and 2000 outbreak in Saudi Arabia (49). The outbreaks of RVF in Arabian Peninsula in 2000 may be associated virus introduction from disease endemic areas through transboundary livestock trade of infected animals and migration of sick persons, these may be supported by the genetic similarity of the Arabian Peninsula strains to Kenyan isolates during the 1997/1998 RVF outbreak (23).

Spatial temporal epidemiology and predictive modeling of RVF

Earlier predictive studies by Linthicum et al. (69) used satellite derived NDVI and rainfall to assess the potential for RVFV activity in two ecologically distinct RVF enzootic areas in Kenya which indicated a positive correlation between NDVI and high mosquito population, hence the high likelihood of RVF occurrence. Using a 19-year-old NDVI data, a mask was created to identify areas where RVF was more likely to occur in the savannah ecosystems of Africa. In East Africa, there was a strong correlation between predicted risk areas and observed outbreaks of RVF from 1981 to 2000; these outbreaks were more likely to occur during warm ENSO events in East Africa while cold ENSO events in Southern Africa triggered RVF outbreaks (70).

A climatic RVF risk-monitoring model was proposed in predicting the potential spatial and temporal distribution by using NDVI anomaly and elevated sea surface temperature (SST). The model retrospectively detected previous three outbreaks (1982–83, 89 and 1997–98) which correlated with positive SST and NDVI anomalies and also predicted, almost accurately, the areas for 2006–2007 outbreak areas in East Africa (13). Despite high success in predicting outbreak areas times and areas, the coarse resolution of 8 km may overgeneralize the risk and it was, therefore, useful for small-scale areas as countries and not continental scales.

Spatiotemporal analysis employing climatic and environmental variables was used in combination with vector surveillance data to predict potential outbreaks in Horn of Africa, Sudan, and Southern Africa with suitable areas of eminent epidemics identified with a lead time of 2–4 months. Accurate prediction timelines by optimal model performance were enhanced with precise animal and human disease data (14). Using time series analysis of NDVI from September 1997 to April 1998, RVF suitable

areas were identified in Kenya with a lead time of 5 months, these received anomalous rainfall which led to favorable conditions for emergence of RVFV vectors. This has been supported by recent studies by Anyamba et al. whose improved methods led to first the prospective prediction of RVFV circulation enabling accurate prediction with lead times of 2–4 months before outbreaks (14, 15, 71).

A large-scale continental estimate of RVF prevalence in Africa using serological data has been explored by a spatially explicit Bayesian logistic-regression model (72). There were correlated high-prevalence RVF clusters in areas that had previously experienced epidemics including Kenya's North Eastern region and Somalia. This study and others form a framework for estimating the seroprevalence where no accurate serological data are available (72, 73).

Using knowledge driven spatial modeling, RVF endemicity suitability maps for Africa were developed by Clements et al. (74). Most of sub-Saharan Africa had high predicted suitability of RVF occurrence; moderate suitability was predicted for Morocco, Algeria, and Tunisia while the whole of the Sahara desert was unsuitable. This was corroborated with overlay of observed serological prevalence for suitability for RVF in Senegal, thus providing wider applications where serological data are available.

Metras et al. (75) reported five major cycles of RVF outbreaks in South Africa from 2008 to 2011 with the 2010 cases having elaborate spatiotemporal interactions and supporting the role of other factors in the spread of disease beyond active vector dispersal. This study, however, described human patients who may have acquired infection by close contact with infected animals or who were infected directly from mosquito bites (74, 75). A comprehensive temporal and spatial distribution of RVF outbreaks in South Africa from 1950 to 2011 was described by Pienaar et al. (22) using more accurate records from smaller but extensive outbreaks in livestock and humans. These studies indicated higher cumulative outbreaks periods in South Africa despite low human and livestock mortalities (Figs. 2 and 3 and Table 1).

Previous retrospective sero-epidemiological studies in Kenya using wildlife data correlate well with spatial predictions and the epidemiological sequence of RVFV transmission providing a future window of improving RVF transmission risk models (76). Sindato et al. (46) in Tanzania examined the distribution of RVF outbreaks from 1930 to 2007. Heterogeneity in outbreaks was observed as well as increased likelihood of occurrence in areas receiving rainfall above 400 mm and areas with clay or loam soils. Previous spatial analysis of non-clinical RVF cases in Tanzania indicated the existence of multiple ruminant hosts that were potential reservoirs of RVFV during inter-epidemic period and increased the likelihood of outbreaks in animals located at elevations less than 1,000 m (21), confirming observations of influence of elevation on outbreaks in Kenya (45).

In Kenya, Hightower et al. (45) utilized geo-referenced locations of human RVF cases during the 2006–2007 outbreak combined with geological and climatological attributes to estimate incidence of RVF disease. A correlation of altitude and disease outbreak was evident with areas at ≤1,100 meters consistently having epidemics. This finding was in agreement with the Tanzanian study that indicated high intra-village seroprevalence of IgG in domestic and wild ruminants and higher risk at low altitudes (53).

Discussion and conclusion

This review describes RVF epidemiologic characteristics, predisposing factors, and potential geographical spread of RVFV that should be considered in light of other favorable parameters influencing outbreaks with emphasis on host susceptibility, environmental and climatic conditions. It describes the impacts of outbreaks such as case fatality and morbidity and also highlights socioeconomic factors that may elevate the risks of infection among vulnerable populations.

Molecular epidemiology is a useful tool in understanding the genetic diversity of concurrent circulation of RVF related virus lineages between Africa and Arabian Peninsula, and there may be two modes of circulation: distant spread across countries or continents and endemic circulation as observed in Senegal. The role of wildlife maintenance of the RVFV in the interepizootic period has since been demonstrated in Kenya (62, 77, 78) (Fig. 2).

Spatiotemporal evidence indicates a progressive spread of RVFV from initial foci in East Africa to the entire continent and further geographical spread to Arabian Peninsula. This may be attributed to the movement of viremic livestock through transboundary livestock trade. It may be precipitated by favorable climatic conditions for aggressive vector emergence and virus dispersal. Despite the scarce data available for livestock and human mortalities, here we have attempted to report human deaths associated with RVF outbreaks over specific time periods where records were available. These maps should, therefore, be interpreted with caution as they may not accurately represent the true burden of disease (Figs. 2 and 3).

Predictive models and mapping potential risk of RVF outbreaks in endemic areas using disease occurrence data, vector population dynamics, anomalous climatic conditions, and surrogate environmental variables provide a foreseeable public health strategy for RVF surveillance (13, 14, 69, 72). The use of seroprevalence data in spatial and temporal prediction of RVF risk provides an opportunity to validate previously suggested prospective models that map areas at risk of RVFV transmission in endemic regions (76).

Despite climatic factors being a major contributor to outbreaks, other ecological and host parameters should be considered for use of spatiotemporal predictive models for cost-effective surveillance. The combination of spatial and temporal predictions of RVF risk with targeted serological and entomological surveillance can be useful and cost-effective tool capable of identifying the areas with the highest probability of an outbreak.

A comparison of RVF outbreaks in Saudi Arabia (2000) and Sudan (2007) by Hassan et al. (79) described a 'One Health' framework with similar economic impacts related to the outbreaks in both countries, and suitable ecological or environmental variables for disease emergence despite the large geographic separation (>1,900 km). The findings of this study can be replicated in other endemic regions by improving epidemiological surveillance systems which will result in better preparedness, earlier detection, and mitigate spread of the outbreak.

RVF episodes have a predictable cyclic occurrence based on climatic forecasts and models with precise applications to the East African and Horn of Africa scenarios (14). However, recent models have failed to accurately pinpoint the location and timing of the next epidemic. This calls for collaborative research efforts that attempt to combine serological, climatic, and ecological data for greater area prediction models that will bridge the uncertainty in RVF spatial and temporal patterns in East, South, and West Africa (22, 72, 75, 76).

While this review focuses on reports of major epidemics and illustrates the geographical pattern of the disease with pockets in East, West, and Southern Africa, RVF endemic status may be underreported due to lack of surveillance and diagnostic capacity in other countries as shown in Fig. 2. The unexpected RVF epidemiological shift to Egypt, Sudan in 1988–89 (80), Arabian Peninsula in 2000, and isolated landmasses like Madagascar, Mayotte, and Comoros may be driven by climatic and ecological factors conducive for robust vector emergence that coincides with the presence of circulating virus dispersed by livestock trade and human migration. Due to unreliable national livestock disease surveillance systems, there is a dearth of livestock mortality figures; thus, our attempt to aggregate the sparse human studies to provide mortality and outbreak maps may be severely underestimated (Figs. 2 and 3).

We have highlighted epidemiological and ecological studies focusing on humans and animals as well as risk factors, molecular and genetic diversity, spatiotemporal and predictive risk mapping for RVF. However, there is a need to contextualize socioeconomic impacts and to quantify long-term disease burden of RVF as evidenced in Table 1 (81–83). Clearly, there is an urgent need for global collaboration and prioritized research funding to tackle RVF and other emerging zoonotic diseases (84).

Disclaimer

The findings and conclusions in this paper are by the authors and views expressed in this publication do not necessarily represent the decisions, policy, or views of their institutions.

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