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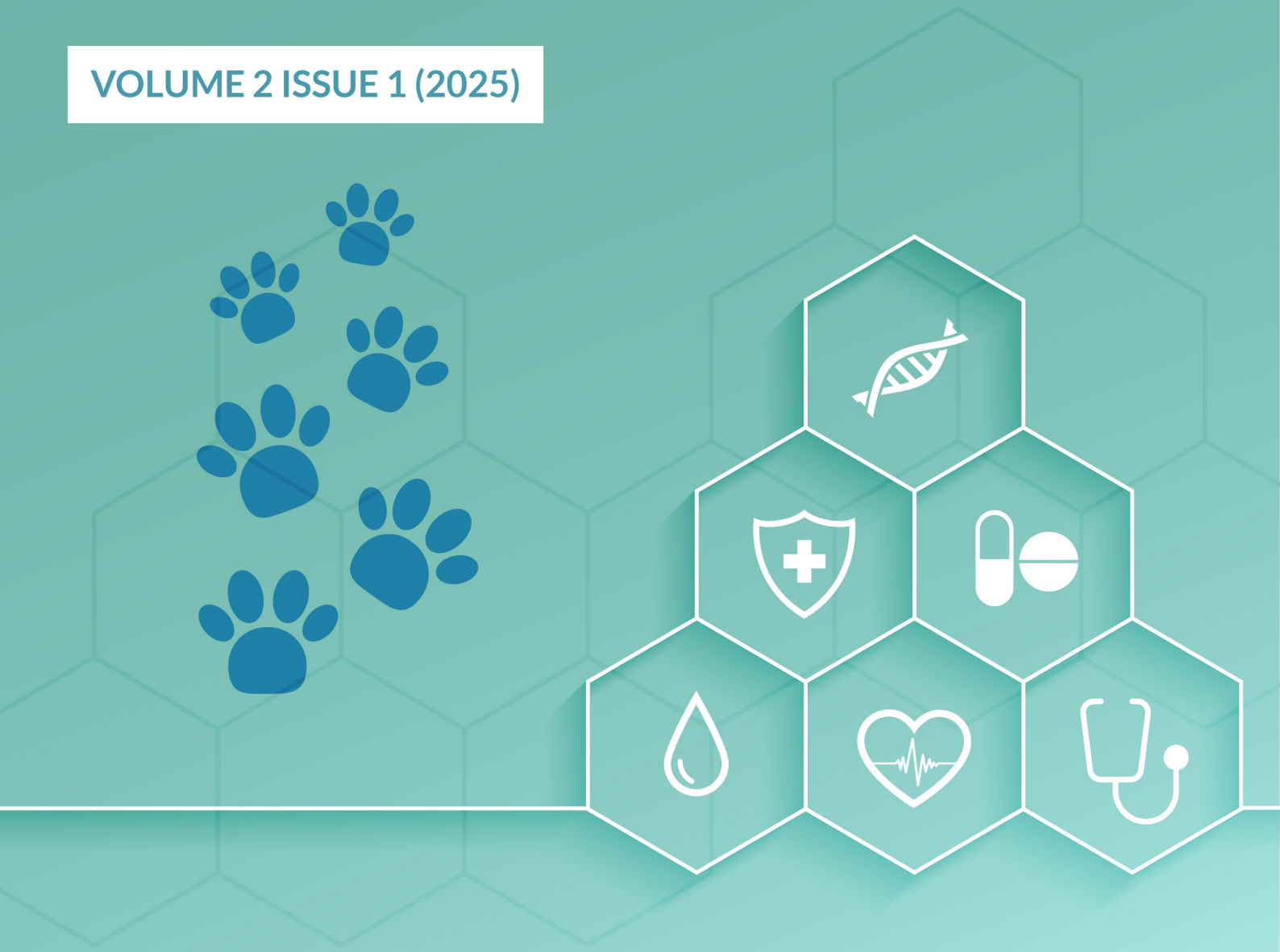

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From Rift to Global Risk: A One Health Perspective on Rift Valley Fever as an Emerging Public Health Threat

Birupaksha Biswas[1*], Damini Joshi[1], Sakshi Thakkar[1], Angel Saluja[1], Md. Bellal Hossain[2], Bongkim Kumar Halder[3]

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ABSTRACT

The zoonotic illness known as Rift Valley Fever (RVF) is spread by mosquitoes and poses a serious risk to public and human health. Within the order Bunyavirales, the genus Phlebovirus and family Phenuiviridae comprise the causal agent, Rift Valley fever phlebovirus (RVFV). The transboundary spread of RVFV is seriously threatened by the substantial presence of competent vectors in regions where the disease is often absent as well as the effects of global climate change. The development of innovative vaccinations, such as DIVA (differentiating infected from vaccinated animals) vaccines, has been greatly aided by advances in the last ten years in our understanding of the molecular biology of RVFV. Despite these developments, non-endemic nations still lack a completely approved vaccination for human or animal use. It is clear that endemic nations lack clear policies or procedures pertaining to the routine or strategic immunization of cattle with the goal of averting or mitigating any RVF disease outbreaks. In addition to offering insights on the best methods for managing the illness, this study aims to give a current summary of the state of RVF vaccine development. This study argues that the most effective way to prevent future disease outbreaks and disease spread. First discovered and characterized in Kenya in 1931, Rift Valley Fever (RVF) is a viral zoonosis spread by mosquitoes. Significant losses have been caused by RVF outbreaks, as seen in the rise in animal abortions and deaths as well as human sickness and mortality. The epidemiology of RVF is thoroughly examined in this research, which covers topics like ecology, molecular diversity, spatiotemporal analysis, and predictive risk modeling. Aedes mosquitoes are recognized as the main cause of outbreaks, whereas Culex mosquitoes act as secondary vectors for the Rift Valley fever virus (RVFV). Nonetheless, the function of Culex species in transmission dynamics could be impacted by environmental change. Our work's objectives were to compile a thorough set of published research from Kenya and Tanzania, pinpoint knowledge gaps on Culex groups, and determine whether there was enough spatiotemporal published data available for a future meta-analysis. This represents a first effort to use the data currently available to gain a deeper comprehension of Culex's function in sustaining RVFV transmission. Using Web of Science, a comprehensive review of the literature was conducted to identify research that sampled Culex in Tanzania or Kenya up until April 28, 2023. In respect to an RVFV risk map, the study identified the major factors impacting the studies, such as their duration and geographic coverage. After that, we evaluated the various methods for identifying species and examined how they could have affected the outcomes. Out of 275 investigations, 17 clearly demonstrated that RVFV served as the catalyst for the inquiry. There was substantial documentation of studies focused on mosquito sampling in regions linked to the risk of RVFV outbreaks, even though different studies examined a variety of topics. Fifty experiments in all were carried out for a minimum of 12 months. Studies on species identification revealed that using a Culex-specific key increased the chance of finding new species outside of the Culex pipiens complex by almost 14 times. We suggest that the data from these broader investigations might potentially provide significant insights into the persistence of RVFV transmission, even though many published studies sampling Culex in Kenya and/or Tanzania did not explicitly specify RVFV as a key study topic.

INTRODUCTION

Rift Valley fever virus (RVFV), which is widespread in several sub-Saharan African nations, is a priority pathogen for the WHO due to the likelihood of outbreaks and the lack of a human vaccine or effective therapies. The virus is zoonotic and can spread by mosquitoes, ruminants like sheep, goats, and cattle, and wildlife. RVFV can infect many host species and mosquito species from the Aedes, Culex, Mansonia, and Anopheles genera (Andrews *et al.,* 2025). In Africa and the Arabian Peninsula, arthropod-borne Rift Valley Fever (RVF) has infected humans and animals. Egypt (1977, 2003), Kenya (1997), Tanzania (2007), Somalia (2007), Saudi Arabia and Yemen (2000-2001), Sudan (2007), Mayotte (2008), and Mauritania (2010, 2012) have had major outbreaks. RVFV is a Phlebovirus in the Bunyaviridae family. Rift Valley fever is carried by around 30 mosquito species from six genera. The warm phase of the El Niño/Southern Oscillation (ENSO) is associated with RVF outbreaks, causing flooding, increased vegetation greenness, and mosquito vectors infecting

[1] Parul institute of Public Health (PIPH), Parul University, Vadodara, Gujarat, India
[2] Department of Public Health, Faculty of Health and Life Sciences, Daffodil International University (DIU), Dhaka, Bangladesh
[3] Upazilla Livestock Officer, Dacope, Khulna, Bangladesh
[*] Corresponding author's e-mail: 2419381010004@paruluniversity.ac.in

vulnerable ruminant hosts (Nanyingi *et al.,* 2015). Over the past decade, many labs and organisations have tried different methods to develop RVFV vaccines. Several labs and teams have tried different methods to generate effective RVFV vaccines in the previous decade. VLPs, viral replicon particles, virus-vectored immunisations, DNA vaccines, subunit vaccines, modified live vaccines derived from reverse genetically modified recombinant viruses, and live attenuated vaccines are examples. Rift Valley fever epidemics can overwhelm veterinary and public health systems, delaying diagnosis and treatment. Tens or hundreds of thousands may be affected. Veterinarians, doctors, farmers, and abattoir workers risk infection from ill animals and patients. Africa's Rift Valley Fever (RVF) outbreaks often began with veterinarians and assistants contracting infections during necropsies on infected animals. After handling and necropsying RVF-infected animals at a South African veterinary college in 2008, several veterinarians, staff, and students developed illnesses (Faburay *et al.,* 2017). The majority of human infections cause self-resolving febrile symptoms. About 1–2% of cases progress to severe disease with high mortality (Zhang *et al.,* 2025). RVF is unlikely to spread across Europe, but localised outbreaks may occur in humid places with many ruminants. This could lead to human cases among farmers, veterinarians, and abattoir workers. We have many monitoring and diagnostic tools, but few control tools. Vector management is difficult, because vaccinations are limited to ruminants and often ineffective or harmful. To protect Europe and the world from RVF, surveillance and control must be improved and regional monitoring and control coordinated (Chevalier *et al.,* 2010). A domestic animal outbreak in 1977 in Egypt caused the largest and worst Rift Valley fever epidemic in humans (McIntosh *et al.,* 1980). Kenya discovered the Rift Valley fever (RVF) virus in 1931, nearly two decades after the first RVF-like sickness was reported. In 1912 and 1913, animal health experts reported a deadly infection in lambs at the government farm in Naivasha, Rift Valley, Kenya. Disease was reported sometimes, usually during wet seasons, until 1931. A farm in the same location reported a highly contagious epizootic that killed young cattle and aborted adults that year (Murithi *et al.,* 2011). Rift Valley fever, an arboviral disease that mostly affects animals and humans, causes extreme chills, malaise, weakness, nausea, severe headache, and hepatic fullness. Phlebovirus, a Bunyaviridae family member, causes Rift Valley Fever. Mosquitoes and domestic animals spread the infection. RVF takes 4–6 days to incubate (Kaur *et al.,* 2023). The infection incubates in people for 2–6 days. Infected people may have mild to severe symptoms or be asymptomatic. Human clinical signs and symptoms are vague. Mild RVF can cause fever, sudden flu-like symptoms, body weakness, joint and muscle pain, headaches, decreased appetite, vomiting, confusion, neck stiffness, light sensitivity, and dizziness (Komugisha *et al.,* 2025).

LITERATURE REVIEW

Rift Valley fever (RVF) is a zoonotic disease transmitted by mosquitoes, caused by the Rift Valley fever phlebovirus (RVFV; family Phenuiviridae), which has significant economic and public health implications in Africa and other regions. Since its discovery in Kenya in 1931, RVF has led to numerous outbreaks and epidemics, marked by significant livestock abortion rates and a spectrum of human illnesses, from mild febrile disease to severe conditions such as hemorrhagic fever and encephalitis. The ability of the virus to spread across borders, transmit between animals and humans, and respond to ecological changes highlights the significance of RVF as a critical priority in the One Health framework (Rostal *et al.,*2025). Historical and contemporary surveillance indicates that the occurrence of RVF is both spatially and temporally variable, with significant outbreaks associated with unusual rainfall and flooding events (such as those linked to ENSO) that enhance populations of competent mosquitoes. Comprehensive reviews spanning from 1999 to 2021, along with recent reports, illustrate changing outbreak patterns and a consistent trend of under-reporting human cases in various contexts, indicating that the public health impact is probably underestimated. These studies highlight the critical role of integrated livestock–human surveillance in identifying early indicators of viral amplification (Gerken *et al.,*2022). Aedes spp. (floodwater Aedes) are well-known as key vectors capable of vertically sustaining RVFV in transovarially infected eggs, which hatch after flooding, thus triggering outbreaks. Culex spp. generally function as secondary or amplifying vectors, capable of maintaining transmission both during and following outbreaks. Nonetheless, the collection of field data from East Africa reveals a complex interplay in vector community composition and seasonal dynamics; changes in the environment and land use may be influencing Culex abundance and their roles as vectors in ways that current surveillance methods do not fully capture. Field studies conducted in Kenya and Tanzania reveal significant gaps in long-term, species-level data for Culex. Furthermore, they indicate that enhanced identification methods could increase the detection of non-pipiens complex species potentially involved in transmission (Andrews *et al.,* 2025).

Objective

To emphasise the One Health strategy for prevention and control, pinpoint information deficiencies, and deliver a comprehensive and up-to-date evaluation of the ecology, molecular diversity, spatiotemporal patterns, epidemiology, and predictive risk modelling of Rift Valley Fever (RVF).

Etiology

One serotype of the Bunyavirus, belonging to the genus Phlebovirus in the family Bunyaviridae, is responsible for causing Rift Valley fever (OIE, 2004). The virus, which is identified by its single-stranded RNA, has two different

surface glycoproteins, G1 and G2, and is encased in a lipid coat. L (large), M (medium), and S (small) are the three separate segments that make up the genome. The RVF virus replicates in vertebrate hosts as well as mosquito populations.The primary locations for viral replication include the liver, spleen, and brain. Disinfectants like calcium hypochlorite, sodium hypochlorite, and acetic acid can effectively inactivate the virus. It has the potential to remain viable for as long as 8 years when kept at temperatures below 0°C (Pal *et al.,* 2012).

Microbiology

The Bunyaviridae family includes Rift Valley fever virus. RVFV is a Phlebovirus, along with nine other viruses such Punta Toro, Sandfly fever, and SFTS. Phlebotomine sandflies distribute most viruses in this genus, therefore "phleobovirus". RVFV is spread by mosquitoes, making it significant. The tripartite genome of all bunyaviruses, including RVFV, has three negatively polarised single-stranded RNA segments. Genome segments are classified as large (L), medium (M), and tiny. The L segment encodes viral polymerase. M encodes Gn, Gc, and NSm, a non-structural protein. L and M segments use negative-sense coding. Phleboviruses encode the S segment with ambisense. The positive polarity encodes NSs, the second non-structural protein, while the negative polarity encodes Nucleoprotein. Nucleocapsids contain viral nucleoprotein (N) that is tightly bound to RNA segments. Complementary nucleotide sequences at each segment's 3' and 5' ends may help construct circular RNAs (Hartman, 2017).

Relevance of Rift Valley Fever To Public Health In The European Union

Public health implications of Rift Valley fever in the European Union The arbovirus that causes Rift Valley fever (RVF), a zoonotic illness that affects both people and domestic ruminants, belongs to the Phlebovirus genus of the Bunyaviridae family. This results in increased rates of abortion in pregnant women and neonatal ruminant mortality, especially in sheep and goats. The RVF virus (RVFV) can infect humans by mosquito bites, contact with animal bodily fluids, or handling of organs and carcasses during processes such as butchering, slaughtering, and necropsy.

Epidemiology

The epidemiology of RVFV is complex and involves multiple factors, such as mosquitoes, wild animals, domesticated livestock, and humans (Hartman, 2017).

Transmission, Epidemiology and Clinical Symptoms

The transmission cycle of RVFV involves ruminants and mosquitoes, emphasising critical aspects of epidemiology and clinical presentations. Sensitivity to hosts changes with age and type of animal (Table 1). Humans serve as the ultimate hosts. The epidemiological cycle is complicated since diseased ruminants can directly infect healthy ruminants or humans, certain mosquito species can pass the disease through their eggs, and there are many possible vectors with different bio-ecology. The conclusive identification of wild reservoir hosts is still

Table 1: Species susceptibility and sensibility to the Rift Valley fever virus:(Lefèvre *et al.,* 2003)

Mortality >70%	Mortality 10-70%	Severe disease with lowfatalityrate (<10%)	Antibody production	Not susceptible
Lamb	Sheep	Human	Camel	Bird
Kid	Calf	Cattle	Horse	Reptile
Puppy	Rodent	Goat	Cat	Amphibian
Kitten		African Buffalo	Dog	
Mouse		Asian Buffalo	Swine	
Rate		Monkey	Donkey	
			Rabbit	

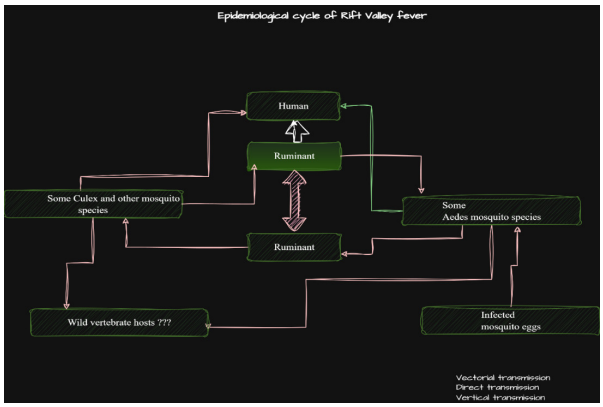

Figure 1: Epidemiological cycle of Rift Valley fever

pending confirmation (Figure 1) (Hartman, 2017; Lefèvre *et al.,* 2003).

Mechanisms of Transmission

Mosquitoes are the main way RVF spreads to ruminants between outbreaks. Among the seven RVFV-infected mosquito genera, Aedes and Culex are the most competent vectors (Sissoko *et al.,* 2009; McIntosh *et al.,* 1981). Other genera include Anopheles, Coquillettidia, Eretmapodite, Mansonia, and Ochlerotatus. Transovarian RVFV transmission in Aedes mcintoshi mosquitoes is known. It may occur in other species, such as the common Ae. vexans species complex. If they survive in dried mud during inter-epizootic and dry/cold periods, some Aedes species' diapaused eggs may hatch into infected imagos (Linthicum *et al.,*1985). Mosquito bites can infect people, although ruminant-to-human transmission is most common (Davies *et al.,* 2006). Viraemic ruminant amniotic fluid, foetal membranes, and blood (during slaughter and butchering) can infect humans. Raw and fresh meat can infect people, but the virus soon disappears with age. Despite not being confirmed, empirical field data imply that ruminants can also obtain the virus from foetuses and foetal membranes after abortion (Gerdes *et al.,* 2004).

Table 2: Arthropods naturally infected by Rift Valley fever virus

Genus	Species	Country (year)
Aedes (Aedimorphus)	cumminsii	Kenya (1981-1984)
		Burkina Faso (1983)
	dalzieli	Senegal (1974, 1983)
	dentatus	Zimbabwe (1969)
	durbanensis	Kenya (1937)
	ochraceus	Senegal (1993)
	tarsalis	Uganda (1944)
	vexans arabiensis	Senegal (1993), Saudi Arabia (2000)
Aedes (Neomelaniconion)	circumluteolus	Uganda (1955), South Africa (1955, 1981)
	mcintoshi	Zimbabwe (1969), South Africa (1974-1975), Kenya (1981-1984)
	palpalis	Central African Republic (1969)
Ochlerotatus	caballus	South Africa (1953)
	caspius	Suspected, Egypt (1993)
	juppi	South Africa (1974-1975)
Aedes (Stegomya)	africanus	Uganda (1956)
	demeilloni	Uganda (1944)
Aedes (Diceromya)	furcifer group	Burkina Faso (1983)
Anopheles (Anopheles)	coustani	Zimbabwe (1969), Madagascar (1979)
	fuscicolor	Madagascar (1979)
Anopheles (Cellia)	chrityi	Kenya (1981-1984)
	cinereus	South Africa (1974-1975)
Culex (Culex)	pauliani	Madagascar (1979)
	pharoensis	Kenya (1981-1984)
	spp.	Madagascar (1979)
	antennatus	Nigeria(1967-1970), Kenya (1981-1984)
	neavi	South Africa (1981)
	pipiens	Egypt (1977)
	poicilipes	Senegal (1998, 2003)
	theileri	South Africa (1970), Zimbabwe (1969)
	tritaeniorhynchus	Saudi Arabia (2000)
Culex (Eumelanomya)	vansomereni	Kenya (1981-1984)
	zombaensis	South Africa (1981), Kenya (1981-1984, 1989)
	rubinotus	Kenya (1981-1984)
Eretmapodites	chrysogaster	Uganda (1944)

	quinquevittatus	South Africa (1971), Kenya (1981-1984)
Coquillettidia	fuscopennata	Uganda (1959)
	grandidieri	Madagascar (1979)
Mansonia (Mansoniodes)	africana	Uganda (1959, 1968), Central African Republic (1969) Kenya (1989)
	uniformis	Uganda (1959), Madagascar (1979)

Mosquitoes

Many mosquito species from different genera have RVFV (Table 1). Aedes mosquitoes are the principal reservoir and vector, while Culex, Anopheles, and Mansonia are secondary vectors that amplify epizootics and epidemics. The virus has also been found in ticks, flies, midges, and other mosquitoes, but its role in biological transmission is unknown. RVFV biology is unique in that the virus is maintained via transovarial transmission within Aedes mosquito eggs (Linthicum *et al.,* 1985) (Figure 2). Dry floodplains retain the virus in latent dehydrated Aedes mosquito eggs during inter-epidemic periods; infected mosquitoes emerge after flooding. Naturally, outbreaks are associated to heavy rainfall, especially during ENSO weather patterns (Davies *et al.,* 1985).

Table 3: Genera of insects and their possible function in the RVF life cycle (Linthicum *et al.,* 2016; Clements *et al.,* 2012; Fontenille *et al.,* 1998; Davies *et al.,* 1980).

Genus	**Type of vector**
Aedes	enzootic reservoir and vector primary
Culex	secondary vector
Mansonia	secondary vector
Anopheles	secondary vector
Coquillettidia	secondary vector
Eretmapodites	secondary vector
Culicoides(biting midges)	role unknown
Plebotomus(sand flies)	role unknown
Simulium(other flies)	role unknown

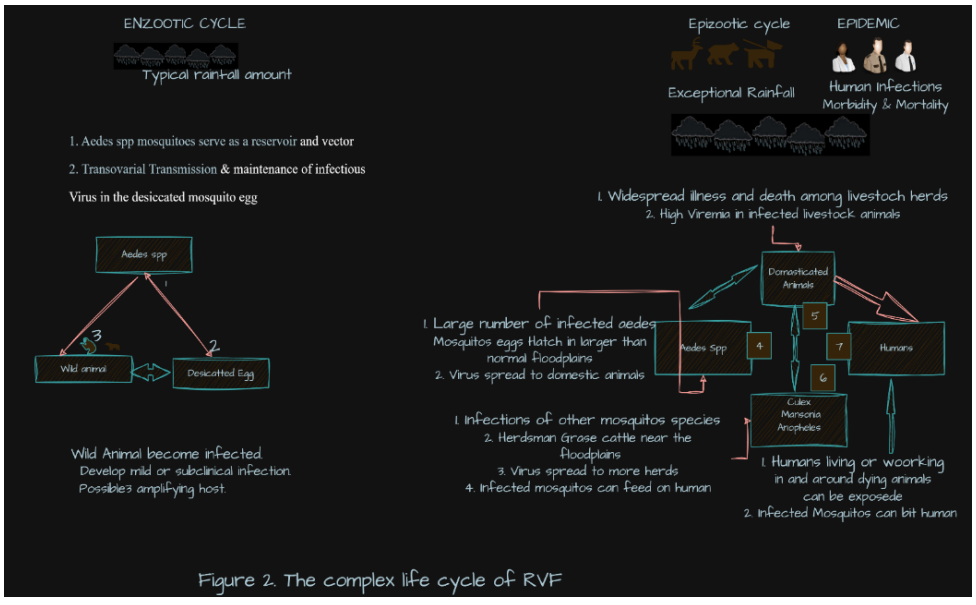

Figure 2: the complex Lifecycle of RVF (Van Velden *et al.,* 1977; LaBeaud *et al.,* 2015).

RVF's complex lifecycle Under normal rainfall, the enzootic cycle (sylvatic or cryptic cycle; green) occurs. Vector and reservoir are infected Aedes mosquitoes (#1). Desiccated mosquito eggs can carry diseases for years (#2). Regular rains create dambos where infected mosquitoes lay their eggs. The transmission cycle of infected adult mosquitoes is unknown, because they feed on and infect wild ungulates (#3). The domestic cycle (epizootic cycle; blue) can be caused by excessive rains or El Nino-Southern Oscillation Events. Larger flood plains

increase the risk of domesticated animals and mosquitoes causing sickness, abortion, and death (#4,5). Culex and Anopheles mosquitoes can transfer the infection to adjacent herds and farther by feeding on affected animals (#6). When many animals are sick and dying, human epidemics occur. Humans can get sick via mosquitoes or infected animals (#7). (Linthicum *et al.,* 1985; Davies *et al.,* 1985).

Mammals

Rift Valley fever virus (RVFV), similar to many arboviruses, cycles between mosquito vectors and vertebrate hosts. Evidence of RVFV infection, as determined by hemagglutinin inhibition or plaque-reducing neutralizing antibody titers, has been documented in various wild mammalian species in Africa, including camels, bats, lions, and elephants (see Table 2) (Boiro *et al.,* 1987: Capobianco *et al.,* 2016: Davies *et al.,* 1985). The virus leads to mild or asymptomatic illness in these species. The potential function of wild animal species as amplifying hosts is still not clearly defined. Unlike wild animals, RVFV demonstrates significant pathogenicity in domesticated ruminants, which act as amplifying hosts. The hosts exhibit considerable viremia, which allows them to infect mosquitoes that feed on them, thereby promoting additional transmission. The most severe manifestation of the disease occurs in developing fetuses and very young animals right after birth; older animals show increased resistance. Table 3 provides a summary of the diseases affecting the three most prevalent domestic animals: cattle, sheep, and goats. Infected animals demonstrate elevated viral levels in their bloodstream for about a week after the onset of illness. Transmission of RVFV directly between animals does not occur within herds or in controlled laboratory environments. Culex and Mansonia mosquitoes are thought to play a role in the horizontal transmission of viruses between viremic animals and humans (Figure 2)(McIntosh *et al.,* 1972).

Table 4: Wild and domesticated animals with evidence of RVF infection (McIntosh *et al.,*1973)

Susceptible wild animals	**Domesticated animals**
springbok	cattle
wildabeest	sheep
impala	goats
lions	buffalo
gazelles	camels
African buffalo	horses
warthogs	donkeys
elephants	
bats	
rhinocerous	
murine rodents	

Table 5: Species susceptibility and sensibility to the Rift Valley fever virus:(Lefèvre *et al.,* 2003)

	Adult Sheep or Goats	**Lambs or Kids**	**Adult Cattle**	**Calves**
Peracute disease?	Yes	No	Yes	No
Acute clinical illness	Leukopenia, raised liver enzymes, fever, weakness, diarrhoea, jaundice, anorexia, blood in the nose, and elevated breathing	elevated respiration rate, anorexia, listlissness, lethargy, and high temperature; abdominal discomfort; ruminant stomach bleeding and hemorrhage; intestinal blood	weakness, anorexia, hypersalivation, bloody nasal discharge, and diarrhea	fever, weakness, anorexia, jaundice, and bloody diarrhea
Macroscopic pathology	Hemorrhage and cell necrosis cause the liver to look mottled, and swollen lymph nodes	Friable, enlarged liver (mottled from hemorrhage and necrotic foci); spleen: capsular hemorrhage with moderate enlargement	Hemorrhage and cell necrosis cause the liver to look mottled, and swollen lymph nodes	swollen lymph nodes; enlarged, friable liver; enlarged spleen with some bleeding

Microscopic pathology	hepatic and splenic necrosis (less severe and extensive than lambs)	Hepatic necrosis, lung congestion, abomasal mucosal bleeding, splenic necrosis, pyknotic/ karyorrhexic kidney, and intermittent small intestinal necrosis	Necrosis of the liver and spleen (less severe and widespread than calves)	Lung congestion; necrosis of the liver and spleen
Time frame	3 days	before 36 hours	2–3 days	2–8 days
Mortality rate	30–50%	90%	5–10%	20%
Fetal effects	frequent abortion (up to 100%)	Comparatively less frequent abortion (up to 85%)		

(Daubney et al., 1931; Coetzer et al., 1977; Van der Lugt et al., 1996; Davies et al., 2006; Coetzer et al., 1982; Food and Agriculture Organization of the United Nations (2016, August 24).

Human

When domesticated cattle experience widespread disease and mortality, humans can get RVFV. While mosquito bites can infect humans, the main way the virus spreads to humans is believed to be by exposure of mucosal membranes or inhalation of viral particles while handling infected animals and corpses. Numerous retrospective studies indicate that consuming, living near, and handling animal products are all linked to a higher risk of contracting RVFV and potentially more serious consequences. (van Velden *et al.,*1977; LaBeaud *et al.,* 2015; Anyangu *et al.,* 2010; Madani *et al.,* 2003; Gear *et al.,* 1951; McIntosh *et al.,* 1980. Nine RVFV outbreaks occurred between 1997 and 2010, resulting in 1,220 verified human fatalities and over 500,000 projected cases (Dar *et al.,* 2013). The most recent human RVF cases, linked to a goat epidemic, were in Uganda in March 2016. One patient claimed to have dealt with ill animals, while the other was a butcher. 35 Only a small number of vertical transmission instances have been reported, and the virus has not demonstrated direct human-to-human transmission (Niklasson *et al.,* 1987; Adam *et al.,* 2008; Arishi *et al.,* 2006).

Diagnostic Methods

Techniques for diagnosis For cattle producers, veterinarians, butchers, workers in slaughterhouses, and lab personnel handling contaminated biological materials, RVFV poses a serious biohazard. For facilities handling the virus in Europe, international public health organisations have set a bio-safety level (BSL) of BSL3, whereas facilities in the US are assigned a BSL4. Peripheral blood obtained in EDTA, plasma or serum from infected people or animals, and liver, brain, spleen, or lymph nodes from dead animals are examples of samples appropriate for diagnosis. When it is possible to transport samples quickly to a diagnostic laboratory (within 48 hours), they must be kept at temperatures lower than +4 °C. If the samples are not already frozen at -20 °C or lower, they must be. It is possible to maintain small organ fragments in a solution that contains 10–20% glycerol. Numerous cell cultures, such as Vero, BHK21, or mosquito line cells, as well as suckling or weaned mice with intracerebral or intraperitoneal injections, can be used to isolate viruses. Effective methods for identifying RVFV in cell cultures include reverse transcriptase polymerase chain reaction (RT-PCR), viral neutralisation test, immunofluorescence, and genome sequencing.The most reliable method for diagnosing RVF is virus isolation. Its sensitivity is quite modest, though, as RVFV isolation is difficult to accomplish. As an alternative, RT-PCR on RNA isolated straight from biological materials can be used to identify RVFV ribonucleic acid (RNA) (Sall *et al.,* 2001). Since results may be obtained in a matter of hours, RT-PCR is the test of choice when RVF is suspected. Enzyme-linked immunosorbent assays (ELISA) and the viral neutralization test (VNT) are serological methods used to identify antibodies against RVFV. Cross-reactions with other phleboviruses are rare, and VNT is highly specific (Tesh *et al.,* 1982; Xu *et al.,* 2007). The gold standard serological test is this one. Nevertheless, it necessitates a BSL3 or 4 laboratory and is expensive and time-consuming. ElISAs for the detection of (indirect) immunoglobulin (Ig) are rapid, sensitive, and specific. VNT is gradually being replaced by them (Paweska *et al.,* 1995). IgG and IgM may also be detected commercially using a competition ELISA (cELISA). It enables both human and ruminant serological diagnostics. It can identify antibodies as early as four days after infection or vaccination in animals that react quickly, and eight days after immunization in all animals (Paweska *et al.,*2003). An further indirect ELISA based on a recombinant RVFV nucleoprotein has been created more recently. It has a 99.4% specificity and a 98.7% sensitivity (Jansen van Vuren *et al.,* 2007; Fafetine *et al.,* 2007; Paweska *et al.,* 2009).

Virology: Host Cell Entrance And Structure

RVFV is classified within the Phenuiviridae family, previously referred to as Bunyaviridae. The sandfly fever virus (SFV), which includes the sandfly Naples virus, sandfly Sicilian virus, and Toscana virus, along with the severe fever with thrombocytopenia syndrome virus (SFTSV), are notable viruses of biomedical significance within the Phenuviridae family. The tripartite RNA genome of RVFV consists of short (S), medium (M),

and large (L) segments, predominantly exhibiting negative-sense polarity (Mansfield *et al.,* 2015)(Figure 3). The only segment employing an ambi-sense strategy is the S segment, which encodes a nonstructural protein (NSs) as well as the nucleoprotein (N) (Figure 3). The L segment encodes the viral polymerase. The M segment encodes two structural glycoproteins, Gn and Gc, as well as a 78 kDa glycoprotein and an additional non-structural protein (NSm). The precise function of the 78 kDa protein remains unclear; however, it appears to play a critical structural role in virions derived from mosquito cells, while not influencing those produced in human cells (Weingartl *et al.,* 2014; Kreher *et al.,* 2014). To facilitate host-cell entry, RVFV Gn and Gc proteins form heterodimers that subsequently assemble into pentamers and hexamers exhibiting T=12 icosahedral symmetry on the surface of the mature virus envelope (Halldorsson *et al.,* 2018; Freiberg *et al.,* 2008; Sherman *et al.,* 2009). RVFV Gc adopts a class-II fusion protein structure similar to those of other phleboviruses, hantaviruses, alphaviruses, and flaviviruses, facilitating the pH-dependent fusion of the host and virion membranes (Huiskonen *et al.,* 2009; Halldorsson *et al.,* 2016; de Boer *et al.,* 2012; Harmon *et al.,* 2012; Guardado-Calvo *et al.,* 2016; Willensky *et al.,*2016. The N-terminal ectodomain region of RVFV Gn has been shown to shield hydrophobic fusion loops on the associated Gc protein and exhibits the same fold topology as the Gn of SFTSV (Zhu *et al.,* 2018; Wu *et al.,*2017; Lozach *et al.,* 2011; Phoenix *et al.,* 2016). Oligomannose-type glycans displaying Gn and Gc can interact with the C-type lectins DC-SIGN and l-SIGN, facilitating the adhesion of virions to cells prior to endocytosis. In cutaneous dendritic cells, where RVFV may proliferate, there is a strong expression of DC-SIGN (Léger *et al.,*2016). Heparan sulfate proteoglycans represent another important component involved in host-cell attachment (Figure 4) (Lozach *et al.,* 2011; Riblett *et al.,* 2016).

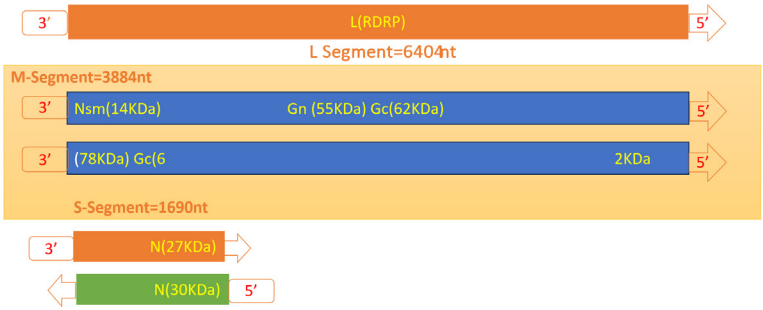

Figure 3: (Wright *et al.,*2019)

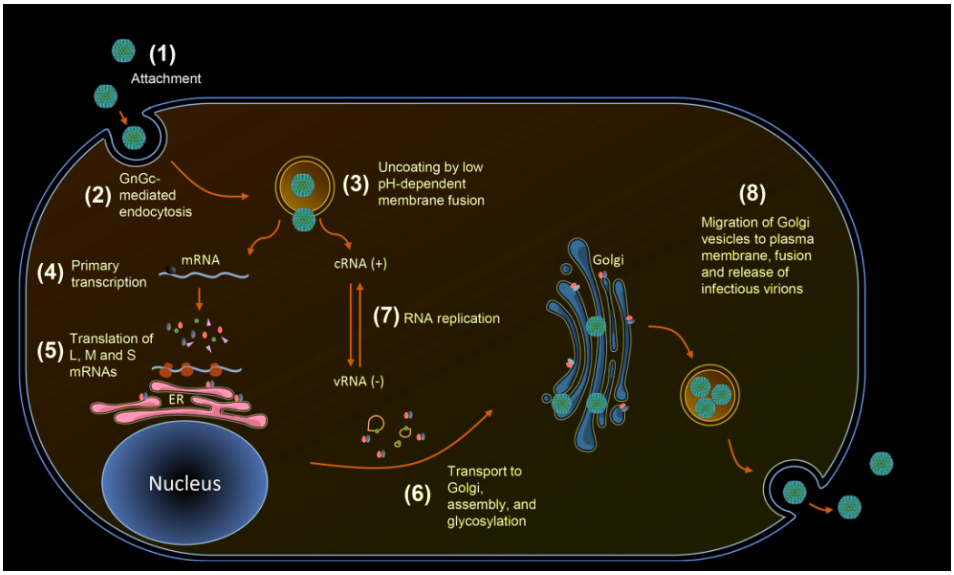

Figure 4: RVFV replication.

Figure 4 shows RVFV replication. Host membrane viral attachment. Endocytosis made easier by GnGc. Endocytic vesicles acidify and viral and endosomal membranes fuse to uncoat. Primary mRNA transcription by viral RNA polymerase. Endoplasmic reticulum processes viral protein translation, M-segment polyprotein cleavage, and GnGc dimerisation. Gn and Gc glycosylation, Golgi cisternae budding, and GnGc heterodimers trafficking.

RNA replication produces positive-sense complementary RNA from negative-sense viral RNA or sub-genomic mRNA for the ambisense S segment. Vesicles with viral content travel to the cell surface, merge with the plasma membrane, and release virions (Knipe *et al.,* 2013). The authors and copyright holder approved figure alteration.

MATERIALS AND METHODS

Data Sources and Search Strategy

The primary data source for this study is the Scopus database. A popular tool for scholarly literature investigations, Scopus is a top global platform for literature retrieval and analysis that excels in integrating academic resources and assessing scientific research. These search phrases were used to retrieve literature from scopus:"RVFV," "Rift Valley fever virus," "Rift Valley fever," "Rift Valley fever phlebovirus," or "RVF." All of the material published between January 1, 1936, and October 25, 2024, was found using a search of the Scopus database. The following were the search criteria: (1) English-language literature categories, such as reviews, research letters, and articles; and (2) information specifically pertaining to RVFV. To guarantee the timeliness and correctness of the data, two separate researchers coordinated the literature search and screening procedure. A full-text examination of possibly pertinent literature was carried out to make sure it suited the study subject, and the search technique was founded on a core screening of titles, abstracts, and keywords. Publications with substantial citation data should receive special attention during the literature screening stage. To suit the requirements of further study and analysis, the particular screening process consists of: (1) exporting datasets of the chosen literature in CSV format from the Scopus database; and (2) using CiteSpace analysis software to transform the data into TXT format text files. Figure 5 shows the whole technological path. There was no need for further Ethics Committee permission because all of the data used in this investigation came from publicly accessible databases.

Analyzing and Visualizing Data

The primary strengths of bibliometrics lie in its data-driven objectivity and trend prediction capabilities. Utilizing citation networks, keyword co-occurrence, and various methodologies, it can identify the evolution of disciplines and the emergence of new fields. The widespread application of databases Platforms like Scopus and the Web of Science have greatly enhanced their reliability. In this study, four professional analysis tools were utilized in the knowledge graph creation process: CiteSpace (version 6.4. R1), and the Bibliometrix online analysis platform. The distinct characteristics and supportive functions of these tools greatly improve the effectiveness of knowledge graph development and advanced analytical abilities. CiteSpace is a visual analysis tool that utilizes set theory to process data in a standardized way. The program combines clustering algorithms with knowledge unit similarity metrics to create a scientific foundation for examining current trends in RVFV research, systematically uncovering its evolution and developmental path (van Eck *et al.,* 2010). The fundamental aspects of VOS viewer include the analysis of keyword co-occurrence and the examination of institutions. The patterns of development, collaborative features, and associated aspects in the RVFV study area can be depicted through networks of collaboration and co-occurrence among authors. VOS viewer presents three distinct visualization patterns. Network visualization provides a structured method for presenting scientific research data in multiple dimensions, highlighting the overarching correlation framework. The overlay visualization effectively illustrates the characteristics of temporal evolution, whereas the density visualization represents the distribution of research hotspots. During the data preparation phase of this investigation, the Scopus literature dataset underwent a systematic de-duplication process using the data cleaning feature of CiteSpace. A comprehensive and systematic protocol has been implemented for all documentation to guarantee the integrity of the data. Specific operations encompass: (1) producing standard text files according to the "thesaurus_authors" specification by employing VOSviewer's term management system; (2) performing term consolidation in the TXT format file, which entails (a) combining synonyms, (b) organizing related terms into broader classifications, and (c) standardizing regional administrative units to align with the national level.

RESULTS AND DISCUSSION

Trends of Publications

This study included a systematic review of 820 documents that satisfied the inclusion criteria. The number of published articles in the RVFV field showed two major peaks, as seen in Figure 6, in 1978 and 2016. There was a dramatic rise in output around 2010, followed by a continuously high output in the following years. While the frequency of reference citations has been rising annually, the total number of publications has been steadily increasing. The yearly number of cited references, in particular, showed a notable increasing peak from 2005 to 2019, demonstrating the ongoing academic interest in RVFV research. Research gaps still exist in important areas such viral pathogenesis, host–pathogen interactions, the creation of innovative vaccines, and particular antiviral treatments, despite tremendous advancements in fundamental virology and the creation of preventative and control measures. In order to improve worldwide readiness against RVFV, this systematic review emphasizes the necessity of targeted research in these areas (Di Nardo *et al.,* 2014).

Description of PRISMA Flow Diagram (Figure 6)

The PRISMA flow chart illustrates the systematic process of identifying, screening, and including studies in this review. A total of 820 records were initially identified through scientific databases, including PubMed (n = 438)

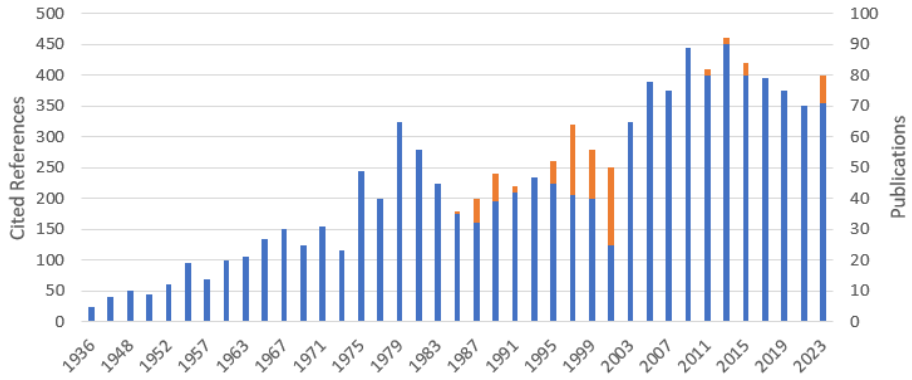

Figure 5: Number of publications and cited Reference

and SpringerLink (n = 382). Additionally, 518 records were retrieved from other sources such as Google Scholar (n = 356), WHO-ILS (n = 140), FAO (n = 2), CDC (n = 2), OIE (n = 2), government records (n = 1), conference proceedings (n = 5), and book chapters (n = 10). After removing 520 duplicates from the database search and 318 duplicates from other sources, 300 and 200 records respectively were screened based on titles and abstracts. Subsequently, 116 records from databases and 110 records from other sources were excluded according to predefined exclusion criteria. A total of 114 full-text articles were then assessed for eligibility. Following further evaluation, 34 studies were included in the synthesis, of which 24 were part of the qualitative synthesis. From other sources, 29 studies were included in both qualitative and quantitative synthesis, while 81 full-text articles were excluded for not meeting eligibility criteria. This process ensured a rigorous and transparent approach to study selection, minimizing bias and enhancing the reliability of the review findings (Sissoko *et al.,* 2009; Imam *et al.,* 1979; Al-Afaleq *et al.,* 2011).

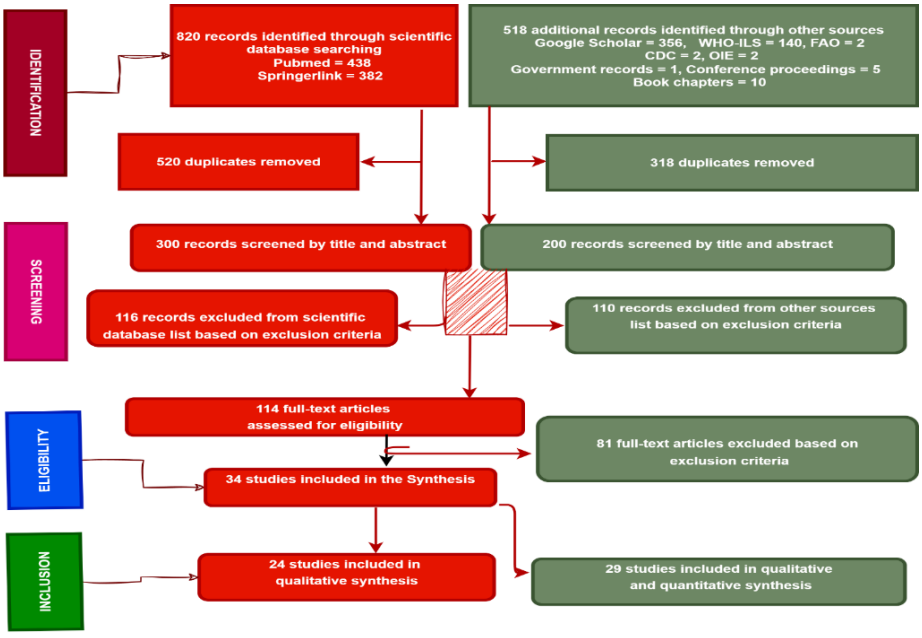

Figure 6: PRISMA flow chart diagram describing the studies selection process for inclusion in this review.

RVFV was originally identified as an epidemic center in Kenya in 1912 (Davies *et al.,*2010), and it stayed there for over thirty years until moving to Tanzania in the late 1940s (Sindato *et al.,*2013). Secondary epidemic foci were established in Southern Africa during the 1950s as a result of severe outbreaks that were recorded there, especially in South Africa, Namibia, Zimbabwe, and Zambia (Pienaar *et al.,*2013). The Arabian Peninsula's near proximity to the Horn of Africa and the resulting movement of cattle are believed to have contributed to the virus's geographic expansion outside of Africa, resulting in epidemics in Saudi Arabia and Yemen (Bird *et al.,*2009). More than 900 people died and significant livestock losses occurred in Sudan, Kenya, Somalia, and Tanzania during the 2006–2007 epidemic (Hassan *et al.,*2011). The first known RVF epidemic outside of Africa occurred in Saudi Arabia

and Yemen in 2000 as a result of abnormally high rainfall and the ensuing floods in the Arabian Peninsula (Ahmad *et al.,*2000). Over 200,000 human infections, roughly 250 human fatalities, and the loss of thousands of livestock were all caused by this outbreak(Abdo-Salem *et al.,*2006; Balkhy *et al.,* 2003; Centers for Disease Control and Prevention (CDC). (2000)(Table 4). The risk of human infection is greatly increased when animals are moved to regions with

Table 6: Characteristics of the eligible studies investigating RVF epidemiology, ecology, risk factors, and spatial modeling 1931 to 2012

Year(s)	Geographic distribution	Study design	Research questions/ objectives	Reported cases	Deaths	Reported cases	Deaths	Estimated impact in US$ (106
1931	Kenya	Cross-sectional	Risk factors and ecology	nd	4700	Nd	nd	nd
1950 1951	South Africa	Cross-sectional	Epidemiology and spatial modeling	600000	100000	Nd	nd	nd 115
1977 1978	Egypt	Cross-sectional	Epidemiology and socioeconomics	nd	nd	200000	598	nd nd
1978	Zimbabwe	Cross-sectional	Risk factors	70000	10000	Nd	Nd	nd
1988	Mauritania	Case control	Risk factors	nd	nd	Nd	224	250
1987 1989	Senegal	Cohort	Molecular epidemiology	1715	nd	273	16	378 nd
1997 1998	Kenya	Cross-sectional	Ecology, risk factors and socioeconomics	89000	478	160,000 28,000 89,000	450	10 107
1997-1998	Somalia Tanzania Mauritani	Cross-sectional	Sero-epidemiology, entomology and virology	nd	nd	90	170	Nd nd
2000	Saudi Arabia	Cross-sectional	Risk factors and ecology	nd	nd	883	478	nd
2000 2001	Yemeni	Cross-sectional	Risk factors and socioeconomics	343	6000	1328	1	541
2003	Egypt	Cross-sectional	Risk factors and virology	>10000	45	45	245	
2007 2008	Sudan	Cross-sectional	Risk factors and ecology	22000	nd	75000 10000	166 17	
2008 2009	Madagascar	Cross-sectional	Epidemiology and socioeconomics	nd	nd	10000	222 26	
2006 2007	Somalia Tanzania Kenya	Cross-sectional	Risk factors, predictive modeling and ecology	32000, nd	Nd, 4200, nd	35000 40000	51 109	
2010 2011	South Africa	Cross-sectional	Spatial and predictive modeling	14342	8,877	75000 242	158 26	nd
2012	Mauritania	Cross-sectional	Risk factors, molecular diversity	nd	343	41	17	nd

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

high mosquito densities and naïve animal populations during the viremic phase of illness, especially for those who handle livestock. (Sindato *et al.,* 2013; Hightower *et al.,* 2012; Sindato *et al.,* 2014; Nicholas *et al.,* 2014)

Furthermore, a multispecies sero-epidemiological investigation in the Sahrawi area, a group of refugee camps in Algeria's Tindouf Province, revealed that less than 1% of the 982 ruminant samples had positive IgG antibody tests against RVFV. However, Mehaires (7.14%) and Tifariti (7.69%) showed two clusters of high seroprevalence (Di Nardo *et al.,* 2014). The return of RVF outbreaks in 2008 may have been caused by the region's closeness to RVF-endemic nations like Mauritania and Senegal, both of which have significant animal traffic. Significantly, older animals and goats showed increased seropositivity, indicating vulnerability related to age and species.According to a major cross-sectional seroprevalence study conducted in Tanzania in 2007–2008 with 17,000 participants, the prevalence among those who live close to bodies of water was 29.3%. Additionally, there was a strong correlation between seropositivity and poverty, animal ownership, and advanced age. Furthermore, regions with thick vegetation, ideal mosquito emerging circumstances, and high cow numbers were shown to have greater incidence (Heinrich *et al.,* 2012). With a case fatality rate of almost 30% among 186 laboratory-confirmed cases, a clinical epidemiological analysis of 511 probable RVF patients during the 2007 epidemic in Tanzania also revealed fever, encephalopathy, retinopathy, and hemorrhagic symptoms as important clinical indications. Contact with cattle or eating items produced from animals was highly linked to an increased risk of infection (Mohamed *et al.,* 2010) (Table 1).A random cross-sectional study of over 1,600 cattle in Tanzania's Kilombero Valley in 2011 showed that the seroprevalence was lower in younger animals born after the 2007 epidemic (5.5%) than in older animals (22.7%). Interestingly, compared to males, female animals were three times as likely to get infected (Sumaye *et al.,* 2013). Despite higher vector densities close to breeding sites, grazing within 5 km of water bodies had little effect on antibody prevalence when compared to animals grazing 15 km away. However, among 1,228 inhabitants in Tanzania's Mbeya Region, a comparable human investigation discovered a significant association between elevated seroprevalence with altitude (Heinrich *et al.,* 2012). Uganda has not documented human or animal RVF outbreaks, despite being geographically close to Kenya and Tanzania. Nonetheless, Magona et al (Magona *et al.,* 2013. Found a 10% seroprevalence of anti-RVF IgG antibodies in 2,700 goats (including domestic and European varieties) from 30 farms spread over four districts, indicating quiet circulation. Similarly, Andayi et al (Andayi *et al.,* 2014). Examined sero-epidemiology and arboviral risk factors in 1,000 people in Djibouti and found a 2.2% RVF prevalence. These results highlight the significance of ongoing viral and vector surveillance by pointing to subclinical transmission in non-epidemic areas.According to a 2011 risk factor evaluation conducted in Mayotte with 1,420 people and 198 seronegative ruminants from 33 herds (Lernout *et al.,* 2013), human seroprevalence was substantially correlated with male sex, age (>15 years), farming, closeness to water bodies, and poor educational attainment. The greatest risk factor for both humans and animals, in line with research conducted in East Africa, was exposure to skilled mosquito vectors. An epidemic in southern Mauritania in 2012 resulted in 41 confirmed cases and 17 fatalities (10, 11). With isolates closely linked to strains from the 2010 epidemic in northern Mauritania, phylogenetic analysis indicated that these outbreaks most likely re-emerged from enzootic foci (Jäckel *et al.,* 2013; Sow *et al.,* 2014). Similarly, an overall seroprevalence of 3.3% was observed by ecological studies carried out in Gabon between 2005 and 2007 with 4,323 participants (10% of national households). Reiterating the importance of water bodies in maintaining vector populations and RVF endemicity, higher incidence was found in lake regions (8.3%) as opposed to wooded (2.9%) and savannah areas (2.2%). Similar correlations were seen in the Mbeya Region of Tanzania (Heinrich *et al.,*2012). During the 2007 epidemic in Sudan, the impact of sex on infection risk was also shown. Males had a threefold greater risk (OR 2.8, 95% CI 1.0–7.6), and 82% (122/149) of the 290 febrile patients in a hospital-based case-control study were RVFV IgG seropositive (Hassanain *et al.,*2010). Significant correlations between RVF outbreaks and environmental parameters, such as higher precipitation (OR 2.0), closeness to water bodies (OR 2.2), and high vector density (OR 4.2), were found in a seroprevalence assessment of 275 small ruminants conducted in Saudi Arabia (Elfadil *et al.,* 2006). These results aligned with data from Tanzania and Gabon (Heinrich *et al.,* 2012; Pourrut *et al.,* 2010). The association between rainfall and vegetation greening as indicators of outbreaks in Mauritania between 1990 and 2012 was further investigated by Caminade (Caminade *et al.,* 2014). They came to the conclusion that one of the main causes of outbreaks was intra-seasonal variability, which is defined by week-long dry spells followed by intense rains. In contrast, the normalized difference vegetation index (NDVI) and total seasonal rainfall are better indicators of RVF outbreaks in East Africa (Linthicum *et al.,* 1999; Soti *et al.,* 2013).

Disease Control and Treatment

Diagnosis

Since the majority of human infections are asymptomatic or result in flu-like symptoms, "abortion storms," or almost simultaneous abortions in herds of pregnant sheep, are sometimes the first indication of an RVF epidemic. Acute RVFV infection in humans and cattle can be diagnosed using a variety of techniques, but all of them need laboratory testing (Mansfield *et al.,* 2015). ELISA detection of IgM against RVFV antigens is one way to diagnose acute or recent infection (Williams *et al.,* 2011). Real-time reverse transcriptase (RT)-PCR is a

highly sensitive and specific molecular technique that may also be used to identify viral RNA. Viruses can also be recovered by cell culture from postmortem organ tissues or blood samples obtained during the feverish stage. Finally, histological techniques, such as post-mortem liver evaluation for hepatic lesions, may be employed. Because of their great sensitivity and specificity, viral neutralization tests are the gold standard for identifying prior exposure to RVFV. These, however, call a specialized lab space that can handle live RVFV safely. There are also commercial ELISA kits (ID-VET, Montpellier, France) that may detect antibodies against the N protein.

Treatment

The majority of human RVF cases don't need to be treated. Other than general supportive care, there is no specialized treatment for people with severe RVF illness. Potential RVF treatments are being researched by a number of groups; some, like ribavirin and favipiravir, have demonstrated effectiveness in rodent models (reviewed in (Atkins *et al.*,2017). Intravenous ribavirin therapy to RVF patients was discontinued during the 2000 Saudi Arabian epidemic because of the apparent rise in neurological illness in these individuals (Hartman.,2017. Further study is necessary in order to create an effective therapy for RVF illness, which is of great relevance.

Vaccine

Vaccines Currently, the only way to stop animals from contracting RVFV is by vaccination. Nonetheless, there is a lot of space for improvement in the current animal vaccinations. In addition, the lack of approved human vaccinations hinders methods to prevent spillover into people (Faburay *et al.,* 2017). An appealing objective is to replicate the long-lasting protection that comes from natural exposure. MP-12 and TSI-GSD-200 are the two vaccinations that are now classified as Investigational New Drugs in the USA (Dungu *et al.,* 2018). The strain, MP-12, was developed in the 1980s and has 23 nucleotide alterations spread throughout its three genomic regions (Ikegami*et al.,* 2015). Despite possible teratogenic effects in pregnant ruminants (Dungu *et al.,* 2018; Mansfield *et al.,* 2015), it is safe and immunogenic in both people and animals, and it has a conditional license for animal immunization (Ikegami, 2017). The US army developed the formalin-inactivated TSI-GSD-200 vaccination to safeguard people whose jobs may expose them to infection risks. Although it has a great safety record, it takes many booster shots to become effective, and even then, about 10% of recipients have low nAb titres or do not seroconvert (Pittman *et al.,* 1999). Due to the irregularity of outbreaks or the dearth of documented cases, the majority of nations in Africa and the Arabian Peninsula do not employ the RVF vaccination for cattle (Dungu *et al.,* 2018). Using a few approved vaccinations, the vaccination rate varies depending on the ecological context in nations that do vaccinate, like South Africa, Egypt, Kenya, and others(Faburay *et al.,* 2017). Named for its creator Smithburn, the most popular commercial vaccination for cattle is a live-attenuated RVFV that provides sustained protection following a single injection (Botros *et al.,* 2006). However, due of the increased risk of miscarriage caused by residual virulence, pregnant animals cannot receive the Smithburn vaccination (Ahmed Kamal., 2011). Furthermore, there is a chance that the vaccination will revert to virulence, as it has in the past (Muller *et al.,* 1995). Genetic reassortment with wildtype RVFV is another possibility, however it is unlikely to lead to an increase in pathogenicity over that of the wild-type virus. The capacity to distinguish between vaccinated and infected animals (DIVA) would be an added benefit of a new RVFV vaccine. The antibody profile produced by live-attenuated RVFV vaccines, like Smithburn, is comparable to that of spontaneous infections, making it challenging to map outbreaks in the face of vaccination. One benefit of subunit vaccinations is that they do not include all of the RVFV antigens. It is feasible to distinguish between animals that have been vaccinated and those that have been naturally exposed by assessing reactions to the N protein, if it is absent from the vaccine. Several viral clones were identified from a human patient infected with the 74HB59 strain in the Central African Republic, including clone 13, another livestock vaccination. A significant loss in the NSs gene, the main virulence factor, was shown to naturally decrease it; subsequent infection in mice revealed that it did not induce illness (Dungu *et al.,* 2010). Cattle, lambs, and goats have shown that Clone 13 is safe and immunogenic following a single dosage (von Teichman *et al.,* 2011; Njenga *et al.,* 2015; Dungu *et al.,* 2018). However, clone 13 can get through the placental barrier and produce teratogenic consequences, according to overdose trials conducted on pregnant ewes (Dungu *et al.,* 2018). In Senegal and Mali, animals have been vaccinated with a thermostabilized variant that was chosen from viable clones following incubation at 56 °C (Dungu *et al.,* 2018).

Animal & Human Prevention

Early animal case discovery by strict active surveillance and sentinel herd monitoring is the first step in preventing serious human disease (Davies *et al.,* 2006) Controlling mosquitoes, regulating animal movement, prohibiting the slaughter of livestock, or at the at least using gloves, masks, and gowns while handling carcasses or aborted fetuses helps stop the spread of the disease once affected animals and/or herds have been identified. To stop more animal sickness from spreading to humans, public education and awareness on the warning signs, symptoms, and risk factors are essential. In high-risk locations, targeted animal immunization might be useful as a control approach. Nosocomial transmission is avoided by standard measures; there is little risk to healthcare personnel (Al-Hamdan *et al.,* 2015).

Discussion

With a focus on host susceptibility, environmental and

climatic circumstances, this study outlines the epidemiologic features, predisposing variables, and possible geographic expansion of RVFV that should be taken into account in light of other favourable aspects impacting outbreaks. In addition to highlighting socioeconomic variables that may increase the risk of infection among vulnerable people, it details the effects of outbreaks, including case fatality and morbidity. The genetic diversity of concurrent circulation of RVF-related virus lineages between Africa and the Arabian Peninsula can be understood through molecular epidemiology. There may be two modes of circulation: endemic circulation, as seen in Senegal, and distant spread across nations or continents. Since then, Kenya has shown the importance of the preservation of the RVFV during the interepizootic era (Bird *et al.,* 2007; Sall *et al.,* 1998; Evans *et al.,* 2008). From its original centres in East Africa throughout the entire continent and then to the Arabian Peninsula, the RVFV has gradually expanded, according to spatiotemporal data. This might be explained by the transboundary cattle trade, which involves the transportation of viremic livestock. Favourable weather conditions for aggressive vector emergence and viral spread may cause it. Here, we have attempted to describe human deaths linked to RVF outbreaks throughout specific time periods when records were available, notwithstanding the paucity of data available for cattle and human mortalities. Therefore, care should be used when interpreting these maps since they could not fully reflect the actual burden of disease. A predictable public health approach to RVF surveillance is provided by predictive models and mapping the risk of RVF outbreaks in endemic locations prioritize disease occurrence data, vector population dynamics, abnormal meteorological circumstances, and surrogate environmental factors (Linthicum *et al.,* 2007; Anyamba *et al.,* 2010; Linthicum *et al.,* 1987; Clements *et al.,* 2007). It is possible to evaluate previously proposed prospective models that map areas at risk of RVFV transmission in endemic locations by using seroprevalence data in the geographic and temporal prediction of RVF risk (Hassan *et al.,* 2014). The use of spatiotemporal prediction models for cost-effective surveillance should take into account other ecological and host characteristics, even if climatic conditions are a primary contributor to outbreaks. Targeted serological and entomological monitoring combined with geographical and temporal projections of RVF risk can be a practical and economical approach that can pinpoint the regions most likely to see an epidemic. A "One Health" framework with comparable economic impacts associated with the outbreaks in both countries and appropriate ecological or environmental variables for disease emergence despite the great geographic distance (1,900 km) was described by (Anyamba *et al.,* 2010). In their comparison of RVF outbreaks in Saudi Arabia (2000) and Sudan (2007). By enhancing epidemiological monitoring systems, the results of this study can be repeated in other endemic areas, strengthening readiness, detecting the epidemic early, and preventing its spread. Based on accurate climate projections and models applied to East African and Horn of Africa scenarios, RVF events have a predicted cyclical recurrence (Pienaar *et al.,* 2013. The next epidemic's location and timing, however, have not been precisely predicted by contemporary models. In order to bridge the ambiguity in RVF spatial and temporal patterns in East, South, and West Africa, this necessitates cooperative research efforts that try to integrate serological, climatic, and ecological data for broader area prediction models (Clements *et al.,* 2007; Métras *et al.,* 2012; Britch *et al.,* 2013; Pienaar *et al.,* 2013. RVF endemic status may be underreported in other countries due to a lack of monitoring and diagnostic capabilities, even though this analysis focusses on reports of large epidemics and shows the disease's geographic distribution with pockets in East, West, and Southern Africa. A combination of climatic and ecological factors that favour robust vector emergence and the presence of circulating virus spread by livestock trade and human migration may have contributed to the unexpected RVF epidemiological shift to Egypt, Sudan in 1988 89 (Clements *et al.,* 2007), the Arabian Peninsula in 2000, and isolated landmasses like Madagascar, Mayotte, and Comoros. Our attempt to compile the few human research to create mortality and outbreak maps may be significantly understated because there are few animal death statistics due to inadequate national livestock disease surveillance systems. Our focus has been on human and animal epidemiological and ecological research, risk factors, genetic and molecular diversity, spatiotemporal risk mapping, and predictive risk mapping for RVF. However, as Table 4 shows, it is necessary to assess the long-term illness burden of RVF and to contextualise the socioeconomic implications. To address RVF and other emerging zoonotic illnesses, it is evident that international cooperation and financing for research must be prioritized immediately (Dar *et al.,* 2013; Rostal *et al.,* 2025; Gerken *et al.,* 2022; Andrews *et al.,* 2025)

CONCLUSION

Several reviews funded by national and EU authorities have analysed the potential introduction and dissemination of Rift Valley fever (RVF) in Europe. The overall calculated risk is still fairly low as of now. Recent epidemiological trends in East Africa, particularly outbreaks in Sudan, the Nile Valley, and Indian Ocean areas, indicate that the Rift Valley fever virus (RVFV) continues to exhibit significant dynamism. Changes in the weather, the environment, and the economy all have a big effect on its activities.

Both legal and unregulated livestock migration are happening because the population is growing quickly and the demand for animal protein is going up. Because of this, the probability of RVF spreading to the Middle East, central Europe, and the Mediterranean basin is likely to rise in the next several years. It will be very important to improve risk assessment frameworks that include accurate predictions of livestock trade and movement across borders between areas with RVF and those without it.

At the same time, high-risk ecological zones should be constantly watched and updated with new environmental data and forecasting technologies. In addition to existing monitoring efforts, such as those started by the EU-funded EDEN project, further research is needed to better understand the biology and vector ecology of RVFV in Europe. Making strong models that predict the chances of a virus getting in, setting up shop, and spreading will make us more ready, help us get early warnings, and help us make smart decisions.

It is still very important to improve vector and disease control measures. A rigorous assessment of current vector management strategies, bolstered by laboratory and field efficacy testing, can facilitate enhanced contingency planning. At the same time, new ideas should be promoted, such looking at genetic control methods that lower the number of mosquitoes or make it harder for RVFV to spread.

Partnerships with the drug industry are also very important. The Smithburn strain, which is widely used and cheap, is one of the current vaccinations that raises safety concerns because it could cause infections in both animals and people. To protect both livestock and high-risk professional groups like farmers, veterinarians, abattoir workers, and butchers, new-generation vaccines, especially recombinant or reverse-genetics platforms, should be given priority. Because there aren't many ways for people to get sick in Europe, tailored immunisation efforts will be more practical and cost-effective than covering the whole population.In the end, long-term RVF prevention must focus on lowering the disease burden in areas where it is common. Long-term strategies should include:

- Better understanding of RVFV reservoirs, vector populations, and environmental drivers in Africa
- Risk-based and community-inclusive surveillance approaches
- Use of predictive spatial models and real-time risk mapping
- Stronger cross-border collaboration and policies that focus on One Health.

The global public health community can better prepare for RVF threats and lessen their effects on both human and animal health by using better scientific knowledge, new technologies, and proactive policies.

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