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Epidemiology and Economic Impact of Rift Valley Fever: A Brief Review

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ABSTRACT

Rift Valley fever (RVF) is a disease of domestic ruminants, caused by an arbovirus belonging to the *Phlebovirus* genus Bunyaviridae family, a group of enveloped single stranded RNA viruses. It is mosquito-borne viral zoonotic disease that has a significant global threat in devastating economic losses at household and national levels and on human health. Lack of efficient prophylactic and therapeutic measures makes infection a serious public health concern. This review was made with the objective of organizing information on the epidemiology, management and economic impacts of RVF. The disease is characterized by a sudden onset of abortions and high neonatal mortality in ruminants and with self-limiting infection in humans. Many outbreaks are associated with persistent high rainfalls, competent mosquito vectors and susceptible vertebrate species. The transmission of RVF is primarily by the bites of the mosquitoes. Human acquires the infection by contact with the infected animals and insect bites. Diagnosis is confirmed by RT-PCR, culture, serology and histopathology of the liver. Vaccination, destruction of vectors, movement control, surveillance and sentinel herd monitoring can help in the control of RVF. Because of the extended geographical range of the virus, probability of emergence in new areas e.g. East African countries is likely to increase in recent years. By considering cyclical occurrence, forecasting high precipitation events using spatiotemporal epidemiological investigation for up to 4 months that may lead to explosive outbreaks is better. Improving knowledge among herders leads to better practices of management programs to benefit most from the livestock industry and safeguard public health.

Keywords: Epidemiology, Rift Valley Fever, Vector, Economic impact, Virus, RT-PCR, and Zoonotic.

INTRODUCTION:

Rift Valley Fever (RVF) is a viral zoonosis that poses significant threats to both animal and human health, particularly in regions of Africa and the Middle East where it is endemic (Anyango *et al.*, 2020). RVF is caused by the genus *Phlebovirus* under Bunyaviridae, which mainly affects domestic ruminants and some

wild animals (Kimani *et al.*, 2016). 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 (Wright *et al.*, 2019). The Rift Valley Fever virus (RVFV) primarily circulates between mosquitoes and susceptible vertebrate hosts, predominantly livestock

such as sheep, cattle, goats, and camels (LaBeaud *et al.*, 2015). Five primary vectors of the *Aedes* species play a crucial role in the transmission of Rift Valley Fever (RVF), with secondary amplifying vectors including species such as *Culex* and *Anopheles*, along with other biting flies (Hassan *et al.*, 2020).

The propagation of RVF transmission is closely linked to environmental factors, particularly anomalies in sea surface temperatures associated with El Niño events in the eastern equatorial Pacific and the western equatorial Indian Ocean. These events lead to increased rainfall, causing flooding, especially in low-lying areas, which creates ideal breeding conditions for *Aedes* mosquitoes, thus facilitating the occurrence of long inter-epidemic/epizootic periods (IEP) and subsequent outbreaks (Hassan *et al.*, 2020). Livestock outbreaks of Rift Valley fever typically occur following bites from infected mosquitoes, highlighting the critical role of vector control in disease prevention and mitigation efforts (Kimani *et al.*, 2016). Transmission to humans typically occurs through mosquito bites or direct contact with infected animals or their bodily fluids. RVF outbreaks are influenced by various factors including climate, ecology, and human activities, making its epidemiology complex and dynamic (Lumley *et al.*, 2017). Environmental conditions conducive to mosquito breeding, such as heavy rainfall and flooding, often precede outbreaks. Furthermore, human factors such as increased livestock trade and movement can facilitate the spread of the virus. RVF outbreaks not only result in significant morbidity and mortality in both animals and humans but also impose substantial economic burdens on affected communities (Anyango *et al.*, 2020). The economic impact of RVF is multifaceted, encompassing direct losses in agriculture, livestock productivity, human health care costs, and broader societal repercussions (Anyango *et al.*, 2020). Outbreaks of RVF in livestock can lead to devastating consequences, including high mortality rates, reduced fertility and productivity, trade restrictions, and increased veterinary and public health expenditures. Moreover, the loss of livestock assets can severely impact the livelihoods of pastoralist communities, exacerbating poverty and food insecurity (Rich *et al.*, 2017). In addition to direct losses in the agricultural

sector, RVF outbreaks impose considerable financial burdens on healthcare systems, particularly in regions with limited resources and inadequate infrastructure (World Health Organization [WHO], 2018). The costs associated with diagnosing, treating, and controlling RVF in both animals and humans can strain already fragile healthcare systems, diverting resources from other essential health services. Furthermore, the broader societal impacts of RVF outbreaks extend beyond immediate economic losses, encompassing disruptions to trade, tourism, and social cohesion (Pal *et al.*, 2021).

Effective surveillance, prevention, and control measures are crucial for mitigating the impact of RVF outbreaks and reducing the burden on both public health and economies in endemic regions. Addressing RVF requires a multidisciplinary approach, integrating veterinary and public health interventions with broader socio-economic strategies to enhance resilience and mitigate the adverse consequences of outbreaks (Grossi-Soyster and LaBeaud, 2020).

Epidemiology and Economic Impact of RVF

Etiology

Rift Valley fever virus (RVFV) is classified among the viral haemorrhagic fevers (VHFs), a group of pathogens that includes a taxonomically diverse array of RNA viruses belonging to the *Arenaviridae*, *Bunyaviridae*, *Flaviviridae*, and *Phenuiviridae* families (Paweska, 2014). Within the *Phlebovirus* genus, RVFV stands out among nine other viral species, such as Punta Toro, Sand fly fever, and severe fever with thrombocytopenia syndrome (SFTS) virus, despite the genus typically being associated with transmission by *phlebotomine* sandflies. Notably, RVFV primarily utilizes mosquitoes as its main mode of transmission (Hartman, 2017). All *bunya viruses*, including RVFV, possess a tripartite genome composed of three negative-polarity single-stranded RNA segments (Wright *et al.*, 2019). These segments are known as large (L), medium (M), and small (S). The L segment encodes the viral polymerase protein, crucial for replication. Meanwhile, the M segment encodes glycoproteins (Gn and Gc) responsible for viral entry into host cells, along with a nonstructural protein NSm (Javelle *et al.*, 2020). Both the L and M segments utilize a negative-sense coding strategy. In contrast, *phleboviruses*, including RVFV, employ an ambisense

coding strategy for the S segment, providing unique mechanisms for gene expression and regulation (Kwaśnik, Rożek, and Rola, 2021).

Rift Valley fever virus (RVFV) is classified within the *Phlebovirus* genus, belonging to the family *Phenuiviridae*, under the Bunyavirales order (Xu *et al.*, 2023). Its etiology primarily involves transmission through arthropod vectors, predominantly mosquitoes of the *Aedes* and *Culex* genera, as well as various species of ticks. The virus primarily affects domestic livestock such as sheep, goats, cattle, and camels, but it can also infect wildlife and humans. In animals, RVFV causes significant economic losses due to high mortality rates in newborn animals, abortions in pregnant animals, and reduced productivity. In humans, RVFV infection can manifest as a range of clinical symptoms, from mild febrile illness to severe manifestations such as hepatitis, encephalitis, and hemorrhagic fever (Ikegami & Makino, 2011). Additionally, RVFV can lead to severe outbreaks with high morbidity and mortality rates among affected populations, particularly in regions where the virus is endemic. Several studies have documented the epidemiology, transmission dynamics, and clinical manifestations of RVFV infection, contributing to our understanding of its etiology and impact on both animal and human health (LaBeaud *et al.*, 2015).

Occurrence and Distribution

Rift Valley fever (RVF) is endemic to regions of sub-Saharan Africa, but its occurrence has been recorded beyond this area due to factors such as globalization, climate change, and the movement of infected animals. The virus has historically caused outbreaks in countries such as Kenya, Tanzania, Somalia, Sudan, and Egypt. However, over the past few decades, RVF outbreaks have been reported in new areas, including the Arabian Peninsula, such as Saudi Arabia and Yemen, as well as in parts of Madagascar (Nanyingi *et al.*, 2015). Rift Valley Fever (RVF) is regarded as endemic to large parts of sub-Saharan Africa, but it has also been recorded outside the sub-Saharan Africa in Egypt and Madagascar, outside the African continent in the Middle East (Saudi Arabia and Yemen) (Avenonline, 2014). The virus was first identified in 1931 during an investigation into an epidemic among

sheep on a farm in the Rift Valley of Kenya (WHO, 2018; Abedin *et al.*, 2021).

Since then, outbreaks have been reported in sub-Saharan Africa and North Africa (WHO, 2018). In 1977, an explosive outbreak was reported in Egypt, the RVF virus was introduced to Egypt via infected livestock trade along the Nile irrigation system (WHO, 2018). In 1997–98, a major outbreak occurred in Kenya, Somalia and Tanzania following El Niño event and extensive flooding (WHO, 2018). Following infected livestock trade from the horn of Africa, RVF spread in September 2000 to Saudi Arabia and Yemen, marking the first reported occurrence of the disease outside the African continent and raising concerns that it could extend to other parts of Asia and Europe (WHO, 2018). Although RVF is mainly affecting large parts of sub-Saharan Africa, there is an increased concern that this vector-borne disease could be introduced into Europe (EFSA, 2013).

The distribution of RVF is closely tied to environmental conditions that favor the breeding of its mosquito vectors and the survival of the virus. The transmission and dispersal of the disease pathogen are affected by many factors, such as climatic, hydrologic and geographic influences, along with impacts from human activities and different forms of virus transmission via different vectors (Xiao *et al.*, 2015). Human and animal movements in territories facing rural movements and civil wars are likely to facilitate RVFV spread and its extension outside its traditional boundaries towards northern Africa (Cêtre-Sossah *et al.*, 2019). Heavy rainfall and flooding create ideal breeding grounds for mosquitoes, leading to increased transmission of RVFV among animal populations. Conversely, drought can concentrate animals around limited water sources, increasing the likelihood of transmission as mosquitoes seek blood meals. Human cases of RVF often follow animal outbreaks, with individuals at highest risk being those involved in livestock farming, animal husbandry, or handling infected animal products (Pepin *et al.*, 2010). Understanding the occurrence and distribution of RVF is crucial for surveillance and control efforts to prevent outbreaks and mitigate their impact on both animal and human populations. Surveillance programs, early warning systems, and vaccination campaigns in high-

risk areas are essential strategies for managing the spread of RVF and reducing its associated morbidity and mortality rates (Rich *et al.*, 2017).

Table 1: Seroprevalence of Rift Valley Fever Virus (RVFV) in different countries and species of animals.

Country	Year	Species and percentages of seropositive					References
		Buffalo	Humans	Goats	Sheep	Cattle	
Senegal	1989		22.3		30.1		(Davies and Martin, 2006)
Madagascar	1990		5.4			29.6	(Clark <i>et al.</i> , 2018)
Mauritania	1998		24.4	16.3	34.8		(Fontenille <i>et al.</i> , 1998)
Central African Republic	2010		16.7	5.0	12.9	7.8	(Nabeth <i>et al.</i> , 2010)
Mayotte	2010		4.1	22.4	22.4	26.8	(Fontenille <i>et al.</i> , 2011)
Kenya	2010		1.4			0.5	(Kahlon <i>et al.</i> , 2012)
Zimbabwe	2008	5.3				12.1	(Paweska <i>et al.</i> , 2008)
Botswana	2010	12.7				5.7	(LaBeaud <i>et al.</i> , 2011)

Source of Infection and Mode of Transmission

Rift Valley Fever (RVF) is a viral zoonosis that primarily affects animals but also has the capacity to infect humans. The majority of human infections result from contact with the blood or organs of infected animals (WHO, 2017; CDC, 2019). This direct contact can occur during slaughter or butchering, while caring for sick animals, during veterinary procedures like assisting an animal with giving birth, and when consuming raw or undercooked animal products (FAO, 2014). Certain occupational groups such as herders, farmers, slaughterhouse workers, and veterinarians are therefore at higher risk of infection (WHO, 2017). The virus infects humans through inoculation, for example via a wound from an infected knife or through contact with broken skin, or through inhalation of aerosols produced during the slaughter of infected animals (WHO, 2017). There is some evidence that humans may become infected with RVF by ingesting the unpasteurized or uncooked milk of infected animals (WHO, 2017). Human infections have also resulted from the bites of infected mosquitoes, most commonly the *Aedes* and *Culex* mosquitoes (WHO, 2017; FAO, 2014). The transmission of RVF virus by hematophagous (blood-feeding) flies is also possible (WHO, 2017). To date, no human-to-human transmission of RVF has been documented, and no transmission of RVF to health care workers has been reported when standard infection control precautions have been put in place (WHO, 2017; CDC, 2019).

The source of infection for Rift Valley fever (RVF) primarily stems from infected animals, particularly livestock such as sheep, goats, cattle, and camels. RVF virus (RVFV) can circulate among these animals either

through direct contact with bodily fluids or tissues of infected animals, or through exposure to contaminated environments such as areas with high concentrations of mosquito vectors (Pepin *et al.*, 2010). Infected animals serve as reservoirs for the virus, amplifying its presence within susceptible populations. The mode of transmission of RVFV to humans predominantly occurs through the bite of infected mosquitoes, particularly species belonging to the *Aedes* and *Culex* genera. These mosquitoes acquire the virus by feeding on viremic animals and subsequently transmit it to humans during subsequent blood meals (Pepin *et al.*, 2010). Additionally, humans can become infected through direct contact with infected animal tissues or body fluids, particularly during slaughtering, handling of aborted fetuses, or assisting with animal births. Moreover, the virus can also be transmitted through the inhalation of aerosols generated during the processing of infected animal products or laboratory accidents (Pepin *et al.*, 2010). Understanding the source of infection and modes of transmission of RVFV is crucial for implementing effective control measures to prevent human infections. Strategies such as vector control, vaccination of livestock, and public health interventions aimed at reducing human-animal contact are essential for mitigating the spread of RVF and reducing its impact on both animal and human populations (WHO, 2017; CDC, 2019).

Host range and susceptibility

Susceptible livestock, primarily including sheep, goats, cattle, and camels, are vulnerable to Rift Valley Fever Virus (RVFV) infection through bites of infected mosquitoes and mechanical transmission by biting flies (Hassan *et al.*, 2020). Natural infections due to RVFV

have been documented in various animal species, including antelope, buffalo, camel, monkey, rodents, and sheep, in addition to humans (Hassan *et al.*, 2020). Furthermore, a wide range of domestic, pet, farm, and laboratory animals are susceptible to RVFV, with certain groups such as children, lambs, puppies, kittens, hamsters, and mice exhibiting high susceptibility (Kasye *et al.*, 2016). However, amphibians and reptiles are generally resistant to RVFV infection (Kasye *et al.*, 2016). Serological positive findings and abortion events have been reported in African buffaloes (*Syncerus caffer*) in South Africa and Kenya, while high prevalence of antibodies against RVFV has been detected in various wild mammal species such as springbok (*Antidorcas marsupialis*), wildebeest (*Connochaetes taurinus*), and black-faced impala (*Aepyceros melampus petersi*) in Namibia, as well as Thomson's gazelle (*Gazella thomsonii*), lesser kudu (*Tragelaphus strepsiceros*), and impala (*Aepyceros melampus*) in Kenya (Nielsen *et al.*, 2020). Additionally, antibodies against RVFV have been identified in black rhinos (*Diceros bicornis*), giraffes (*Giraffa Camelopardalis*), African elephants (*Loxodonta africana*), and warthogs (*Phacochoerus aethiopicus*), suggesting their potential susceptibility to RVFV infection (Nielsen *et al.*, 2020). Even certain bat species, including *Micropteropus pusillus*, *Hipposideros abae*, and *Hipposideros caffer*, have shown serological positivity, with RVFV strains isolated from pooled organs, indicating their involvement in the virus transmission cycle (Nielsen *et al.*, 2020). Limited data exist on the susceptibility of European wild ruminant species to RVFV, with white-tailed deer (*Odocoileus virginianus*) in North America being the only reported indication outside the African continent (Nielsen *et al.*, 2020). It is hypothesized that the virus persists at low levels in forest cycles during the IEP, with ruminants and pseudo-ruminants likely acting as reinforcing hosts (Wright *et al.*, 2019).

Morbidity and Mortality

Rift Valley Fever (RVF) is associated with significant morbidity and mortality, particularly in susceptible animal populations and occasionally in humans. The morbidity and mortality rates vary depending on factors such as host species, age, and coexisting health conditions. In susceptible livestock, including sheep,

goats, cattle, and camels, RVF can cause high rates of morbidity and mortality, resulting in substantial economic losses to agricultural industries (Paweska, 2014). Among young animals such as lambs, kids, puppies, and kittens, mortality rates can reach as high as 70% to 100%, highlighting the severity of the disease in vulnerable populations (Musser *et al.*, 2005). Moreover, in severe cases of RVF infection in humans, coexistence with other diseases like acute malaria or HIV infection has been associated with mortality rates as high as 75%, underscoring the importance of considering comorbidities in assessing disease outcomes (Paweska, 2014). Although adult cattle, goats, buffaloes, and humans are generally considered moderately susceptible to RVFV infection, mortality rates typically remain below 10%, with the case fatality rate in humans typically less than 1% (Kasye *et al.*, 2016). Understanding the determinants of severe Rift Valley Fever (RVF) progression has been a challenging endeavor. Retrospective studies have shed light on various factors associated with an increased likelihood of RVFV infection and potentially worse outcomes. These include activities such as touching or handling animals, living in close proximity to animals, and consuming animal products, all of which result in significant exposure to the virus (Javelle *et al.*, 2020). Additionally, fatal cases have documented coinfections such as schistosomal liver involvement or bacterial and fungal infections, further complicating the clinical course (Paweska, 2014). Host susceptibility to RVFV varies depending on age and species. Young animals, including lambs, kids, puppies, and kittens, exhibit extreme susceptibility with mortality rates ranging from 70% to 100% (Musser *et al.*, 2005). Sheep and calves are classified as highly susceptible, with mortality rates between 20% and 70%, while adult cattle, goats, buffaloes, and humans are considered moderately susceptible, with mortality rates typically below 10% (Paweska, 2014). Equines, pigs, dogs, and cats are generally resistant to RVFV infection, with infections often going unnoticed (Kasye *et al.*, 2016).

Risk Factors

Pathogen related factor

The viral Gn/Gc complexes play a crucial role in mediating cell entry and fusion (Ganaie *et al.*, 2021).

Additionally, the non-structural protein NSs acts as a major virulence factor, enabling the virus to evade the host innate immune response by suppressing the type I interferon response (IFRI) (Kwaśnik *et al.*, 2021). Variations in Rift Valley Fever Virus (RVFV) tropism and virulence are thought to be influenced by the involved lineage and the potential accumulation of genetic mutations or genomic re-assortments, with

single nucleotide polymorphisms being associated with severe symptomatology (Javelle *et al.*, 2020). RVFV exhibits broad cellular tropism, with various cell types, including neurons, epithelial cells, macrophages, granulocytes, pancreatic islet cells, adrenal glands, ovaries, testes, and placenta, susceptible to infection (Ganaie *et al.*, 2021).

Table 2: RVF severity in different animal.

Mortality ~100%	Severe illness, Abortion, Low mortality	Severe illness, Viremia, Abortion	Infection, Viremia	Refractive to infection
Lambs	Sheep	Monkeys	Horses	Guinea pigs
Calves	Humans	Camels	Cats	Rabbits
Kids	Cattle	Rats	Dogs	Pigs
Puppies	Goats	Gray squirrels	Monkeys	Hedgehogs
Kitten	Water buffalo			Tortoises
White mice				Frogs
Hamster				Chickens
Field mice				Canaries
Door mice				Pigeons
Field voles				Parakeets

Transmission of RVFV from one mosquito generation to another, known as vertical transmission, has been demonstrated in *Aedes* mosquitoes (Kwaśnik *et al.*, 2021). Furthermore, the virus can persist in *Aedes* eggs, which are resistant to desiccation, allowing for its survival in the environment during inter-epidemic dry/cold periods. These mechanisms, coupled with extreme rainy events during phenomena such as El Niño, contribute to the re-emergence of the disease every 5-15 years, with limited infections during the inter-epizootic period (Kasye *et al.*, 2016). RVFV exhibits low vector specificity and can be transmitted by various vectors, including over 30 species of mosquitoes from seven different genera (*Aedes*, *Anopheles*, *Coquilletidia*, *Culex*, *Eretmapoites*, *Mansonia*, and *Ochlerotatus*), as well as other arthropods such as sand flies (Rolin *et al.*, 2013). This broad vector range suggests that RVF is likely to emerge as an important zoonotic disease worldwide (Balkhy and Memish, 2003).

Host related factor

In areas where Rift Valley Fever (RVF) is endemic, a decline in the herd immunity of livestock populations can lead to widespread virus transmission, culminating in explosive outbreaks (Paweska, 2014). Indigenous livestock species in Africa typically exhibit a high

level of resistance to RVF, contributing to the reduction of enzootic transmission in regions with intense infections (Kasye *et al.*, 2016). The development of massive immunity in recovered animals, transmitted passively through colostrum from mother to offspring, results in enzootic cycles occurring at intervals of 4 to 7 years (Kasye *et al.*, 2016). Long-lived neutralizing antibodies provide protection against RVFV in all species, with sheep and cattle displaying complete resistance to reinfection after previous exposure (Wright *et al.*, 2019).

The innate immune response plays a pivotal role in RVF disease progression, with the NSs protein acting as a major virulence determinant by antagonizing type I interferon (IFN) responses (Ganaie *et al.*, 2021). Mechanisms such as the binding of host protein YY1 to NSs and SAP30 facilitate the downregulation of IFN-β transcription, suppressing the innate immune response within hours of infection (Wright *et al.*, 2019). Additionally, the surface receptor LDL receptor-related protein 1 (Lrp1) is essential for RVFV infection, with direct interaction between Lrp1 and the viral Gn protein facilitating viral entry (Ganaie *et al.*, 2021). Exotic breeds exhibit greater susceptibility to RVF compared to indigenous sheep, goats, and cattle, while recovered animals develop lifelong immunity

(Fyumagwa *et al.*, 2011). Seroprevalence studies have revealed median RVFV seroprevalence rates of 12.9% in sheep, 12.6% in cattle, 11.3% in wildlife, 10.1% in goats, 8.8% in camels, and 5.9% in humans (Clark *et al.*, 2018). Host-related risk factors, such as age and species, influence seropositivity rates, with sheep often at increased risk due to factors such as high population turnover and differences in susceptibility or immune response (Nanyingi *et al.*, 2015). Diagnostic tests such as inhibition ELISA have shown high sensitivity and specificity in sheep and camels, providing reliable estimates of seroprevalence (Clark *et al.*, 2018). Understanding these host-related risk factors and diagnostic methodologies is essential for effective disease management and control strategies.

Vectors

Vector risk takes into account climate (temperature and rainfall) and biotic variables (breeding sites and presence of vertebrate hosts) (Tantely *et al.*, 2015). Mosquitoes have breeding behavior and RVFV transmission dynamics in their respective populations. *Culex* mosquitoes lay their eggs in the inner area of ponds. Unlike the *Culex*, *Aedes* mosquitoes lay their eggs in the moist soil around a body of water rather than on the water surface. Newly laid eggs require minimal time (minimum drying time) to develop to the mature stage. Mature eggs remain in this state until submerged upon hatching. Mature eggs are resistant to desiccation and can be kept in this state for many years (Wanyoike *et al.*, 2021).

Primary vectors such as *Aedes* mosquitoes maintain viability in their eggs, even during dry soil conditions, enabling the virus to persist during inter-epidemic and overwintering periods through vertical transmission (adult to egg) (Van den Bergh *et al.*, 2022). Meanwhile, secondary vector mosquitoes, primarily from the *Culex* species, may migrate to sites with infected animals, facilitating continued virus transmission and potentially widening the geographical spread of the disease, including transmission to humans (Kwaśnik *et al.*, 2021). Epidemic transmission of Rift Valley Fever Virus (RVFV) is often associated with heavy and prolonged rainfall, particularly when stagnant floodwaters are colonized by *Culex* and *Mansonia* species, leading to increased transmission to domestic animals and humans (Kwaśnik *et al.*, 2021).

The flight capabilities of *Aedes* and *Culex* mosquitoes are somewhat limited, spanning from a few hundred meters to over 10 km, yet still sufficient for local spread of RVFV (Chevalier *et al.*, 2010). Wind-assisted transportation of infected mosquitoes has been documented for other arboviruses, further emphasizing the potential for RVFV spread via this mechanism (Chevalier *et al.*, 2010). In South Africa's northern KwaZulu-Natal region, where *Ae. durbanensis* is prevalent, the low minimum infection rate (MIR) of RVFV in tested mosquitoes (0.03%) aligns with detection rates observed elsewhere, typically less than 0.1%, even during outbreaks (Van den Bergh *et al.*, 2022). Studies identifying a species as a potential vector in one geographic area may not extend to members of the same species from a different geographic area. The variation in vector competence appeared to be the result of both a midgut infection and a midgut escape barrier. When genetically similar mosquitoes that separated based on the location were allowed to feed on the same hamsters and handled in the same manner, infection rates were significantly different (Turell *et al.*, 2010).

Environmental factors

For RVF occurrence, the ecology of vectors is highly dependent on environmental conditions (to facilitate host seeking or breeding-site-seeking behavior) or specific vegetation for some species (Pachka *et al.*, 2016). Elevated temperatures, for example, can intensify mosquito feeding frequency and egg production while shortening the duration of their development cycle and the extrinsic incubation period of RVFV in mosquitoes. Furthermore, flooding events contribute to increased animal and human concentrations on dry land areas, thereby amplifying the potential for virus transmission (Paweska, 2014). Studies conducted in regions such as Saudi Arabia and South Africa have explored factors associated with RVF incidence in animals, utilizing epidemic data in conjunction with spatial and time-varying environmental conditions (**Table 3**).

Pathogenesis and Clinical Signs

In both animals and humans, the liver serves as the primary site of Rift Valley Fever Virus (RVFV) replication and the major site of tissue pathology (Kwaśnik *et al.*, 2021). However, during severe

infections, the virus can be detected in virtually all tissues and cell types. Early markers for fatal RVF in humans include hepatic necrosis and increased liver enzymes (Paweska, 2014). Following infection, the virus spreads from the initial site of replication to critical organs such as the spleen and brain, where it causes damage through pathogenic effects or immune-

pathological mechanisms. Alternatively, recovery can occur mediated by nonspecific and specific host responses. The virus is transported from the inoculation site via lymphatic drainage to regional lymph nodes, where replication occurs before dissemination into the circulation, leading to viremia and systemic infections (Kasye *et al.*, 2016).

Table 3: Environmental risk factors.

Study area	Risk factor	Category	HR/OR	Reference
Saudi Arabia	precipitation	increased precipitation	OR=2	(Nanyingi <i>et al.</i> , 2015)
	water bodies	Presence of water bodies	OR= 2.2	(Nanyingi <i>et al.</i> , 2015)
	vector density	High vector density	HR=4.21	(Nanyingi <i>et al.</i> , 2015)
South Africa	vegetation density	Increased vegetation density	HR = 4.20	(Métras <i>et al.</i> , 2015)
	wetlands	Presence of wetlands	HR = 3.52	(Métras <i>et al.</i> , 2015)
	Temperature	above 32°C	HR = 44.35	(Métras <i>et al.</i> , 2015)

Molecular studies suggest that the pathogenicity of the virus in humans may be influenced by widespread vaccination of ruminants in Africa with the live attenuated RVFV Smith-burn neurotropic strain (SNS) (Paweska, 2014). Initial signs of RVF in animals depend on the breed and genotype, but a sudden onset of abortions among sheep, goats, cattle, or camels across a broad area is a significant indicator (Van den Bergh *et al.*, 2022). The concurrent occurrence of influenza-like illness among individuals working with livestock is an additional feature of RVF epizootics. It is worth noting that resistant genotypes of indigenous African cattle and sheep often exhibit no clinical signs of illness, despite experiencing a brief period of viremia (Davies and Martin, 2006).

RVF in livestock

Infection of pregnant domesticated animals at any stage of gestation invariably leads to nearly 100% fetal mortality (Hartman, 2017). Adult livestock are susceptible to peracute disease, which manifests as sudden death without preceding clinical signs. Additionally, they may develop acute disease characterized by symptoms such as weakness, anorexia, diarrhea, bloody nasal discharge, and jaundice (Kasye *et al.*, 2016). Mortality rates vary depending on the species and age of the animal, with sheep and goats generally exhibiting higher susceptibility to death compared to cattle (Kwaśnik *et al.*, 2021). In affected livestock, the liver is the primary target organ of the virus, with characteristic lesions of Rift Valley Fever (RVF) including extensive areas of necrosis and hemorrhage,

resulting in a mottled appearance of the liver (Wright *et al.*, 2019). The severity of the disease correlates with the size of liver lesions, and similar necrosis and hemorrhage are observed in the splenic pulp. Bleeding and the presence of blood in the stomach and small intestine can lead to manifestations such as bloody diarrhea (Hartman, 2017).

RVF in Humans

Among those infected with Rift Valley Fever (RVF), approximately 98% experience subclinical symptoms, while only 2% develop severe complications such as ocular disease, meningoencephalitis, or hemorrhagic fever (Wright *et al.*, 2019). Ocular complications are the most commonly reported, with patients presenting symptoms such as decreased vision (either bilateral or unilateral), blind or black spots, photophobia, and retroorbital pain (Grossi-Soyster and LaBeaud, 2020). Examination often reveals inflammation of the retina and blood vessels, along with bleeding in the retina. While vision defects may not always be permanent, resolution can take weeks to months (Anywaine *et al.*, 2022).

Approximately 1-2% of RVF cases in humans manifest as severe hepatotropic disease, akin to that observed in sheep and other domestic animals (Hartman, 2017). In addition to the nonspecific symptoms mentioned earlier, patients may develop jaundice and hemorrhagic manifestations, such as blood in urine or feces, hematemesis, purpuric rash, and bleeding gums. Autopsies of affected individuals often reveal signs of liver necrosis and prolonged blood

clotting time (Hartman, 2017). Neurological disease represents a third complication associated with RVF, typically presenting with a delayed onset, occurring 5 to 30 days after the initial febrile illness (Anywaine *et al.*, 2022). Clinical signs include severe headache, hallucinations, confusion, dizziness, excessive salivation, and weakness or partial paralysis. This form of RVF can be fatal, with patients experiencing central nervous system complications succumbing to the disease. In survivors, symptoms may persist long-term or even become permanent. While the mechanisms underlying different disease outcomes in humans remain incompletely understood, recent evidence suggests that genetic polymorphisms, co-infections, and comorbidities may contribute to more severe disease outcomes (Javelle *et al.*, 2020). Despite isolated cases of vertical transmission, there appears to be no significant increase in miscarriage rates among pregnant women (Hartman, 2017).

Diagnosis

The International Office of Epizootics (OIE) Manual of standards for diagnostic tests and vaccines contains guidelines on the collection of samples and the diagnostic techniques for diagnosis of RVF infection (Paweska, 2014). Information that is required includes: Sampling site with map reference or full address, Owners name, contact address, telephone, etc., Herds/flocks/breeds/strains affected, numbers and age groups, Date of first case/date sampling, No affected/no dead/no abortions/age groups, Full clinical history, Presence/absence of febrile human disease and Basic ecological characteristics of affected area (Davies & Martin, 2006).

Field diagnosis

Diagnosis of Rift Valley Fever (RVF) may rely on clinical presentation, climatic conditions, and ecological factors, including the presence of large mosquito populations, coupled with the sudden onset and rapid spread of the disease (Kasye *et al.*, 2016). Suspicion of RVF arises when unusually heavy rainfall is followed by widespread abortion and mortality among newborn animals, typically accompanied by necrotic hepatitis. Similarly, suspicion is warranted when hemorrhages and symptoms resembling influenza occur in individuals handling animals or their products (Davies and Martin, 2006).

Laboratory confirmation of RVF

Detection of immunoglobulin G (IgG) antibodies against Rift Valley Fever (RVF) by Enzyme-Linked Immunosorbent Assay (ELISA) indicates previous exposure to the virus, whereas detection of immunoglobulin M (IgM) suggests recent infection (Paweska *et al.*, 2005). Handling of RVF-infected materials should only be conducted under P-2/P-3 conditions or within type II biosafety cabinets and HEPA filtered respirators, ensuring staff safety. Consequently, the choice of diagnostic procedures relies on the availability of appropriate facilities (Davies and Martin, 2006). During specimen collection for RVF diagnosis, tissue samples should ideally be transported in a phosphate-buffered saline/glycerol suspension. Samples preserved in buffered formalin can withstand unfavorable conditions for several days without deterioration. Blood samples in EDTA or heparin, as well as samples of fetal liver, spleen, or lymph nodes, should be transported on ice to maintain integrity (Kasye *et al.*, 2016). A minimum of 10-20 serum samples from recently aborted animals and 10-20 samples from non-aborted animals should be collected for comprehensive testing (Davies and Martin, 2006). Confirmation of RVF diagnosis involves detecting RVF virus/antigen through various methods such as agar gel double diffusion test, antigen capture ELISA, RT-PCR, intraperitoneal inoculation, immunofluorescent or peroxidase staining of fixed cells, Immunohistochemical methods, and histopathology of the liver (Kwaśnik *et al.*, 2021). Additionally, detection of specific antibodies to RVF virus includes ELISA systems for IgM and IgG antibodies, microtitre virus-serum neutralization tests in tissue culture, plaque reduction tests in tissue culture, indirect immunofluorescent tests, and indirect haemagglutination tests (Davies and Martin, 2006).

Differential diagnosis

Differential diagnosis of Rift Valley Fever (RVF) presents challenges due to symptom overlap with other hemorrhagic fevers. While commercially available RT-PCR kits offer case confirmation, the brief viremia period necessitates combining molecular assays with serological tests for reliable detection (Chevalier *et al.*, 2010). Conducting epidemiological studies in cohorts or areas with prior outbreak

evidence is advisable to estimate the true disease burden and validate existing molecular and serological tests, including Rapid Diagnostic Tests (RDTs) where applicable (Petrova *et al.*, 2020).

Single cases of RVF may be mistaken for various viral diseases causing sudden death in sheep, along with generalized lymphadenopathy and petechial and ecchymotic hemorrhages throughout the carcass (Kasye *et al.*, 2016). Diseases manifesting similarly include Nairobi sheep disease (lacks hepatitis and does not affect newborn lambs), Bluetongue (manifests with mouth and foot lesions), Heart water (evidenced by serous fluids in body cavities and neurological signs), Ephemeral fever (characterized by recumbence and rapid recovery), Wesselbron (a rare viral disease less severe than RVF), Toxoplasmosis, Leptospirosis, Brucellosis, Q fever, Salmonellosis, Peste des petits ruminants (causing high mortality in lambs), and Foot-and-mouth disease (associated with neonatal mortality and abortions in small ruminants) (Davies and Martin, 2006).

Prevention and Control

Treatment

Given the significant prevalence of malaria and tick-borne rickettsia disease in Africa, it is recommended that patients be empirically treated with broad-spectrum antibacterial and/or antiparasitic medications until a definitive diagnosis of Rift Valley Fever (RVF) can be established (Paweska, 2014). While the majority of human RVF cases do not necessitate specific treatment, severe instances of the disease require comprehensive supportive care as there is no targeted therapy available (Wright *et al.*, 2019).

For mild to moderate cases of RVF, symptomatic relief with simple analgesics and adequate fluid management is typically sufficient, with a favorable prognosis expected. However, in instances where the disease progresses to severe manifestations such as encephalitis or hemorrhage, prompt identification and aggressive critical care interventions are imperative for any chance of survival (Balkhy and Memish, 2003). Historically, Ribavirin has been considered a potential antiviral treatment for Rift Valley Fever due to its demonstrated efficacy *in vitro* and limited efficacy against other hemorrhagic fever viruses *in*

vivo, such as Lassa fever and Crimean-Congo hemorrhagic fever. However, its intravenous administration may elevate the risk of neurological complications. Newer broad-spectrum antiviral drugs like Favipiravir have shown promise in animal models (Hartman, 2017). It is crucial to avoid the iatrogenic use of medications such as hepatotoxic analgesics (acetaminophen), aspirin, or non-steroidal anti-inflammatory drugs during the early stages of RVF, as they can exacerbate the risk of hemorrhagic complications (Javelle *et al.*, 2020).

Effective communication and Education

Efficient communication stands as a cornerstone for safeguarding both human and animal health, as well as the stability of economies and trade. Messaging must adhere to principles of transparency, evidence, and risk, with a particular emphasis on amplification during the early warning phase to offer timely guidance (Kitandwe *et al.*, 2022). The intended recipients of these messages span across various sectors including animal health personnel, livestock producers, and individuals involved in processing and selling livestock products, urban consumers, and trading partners (Mariner, 2018). Enhancing awareness of Rift Valley Fever (RVF) among both residents and visitors in endemic regions is critical for future outbreak control and prevention (Balkhy and Memish, 2003). Education concerning disease transmission modes and necessary precautions, particularly protection against mosquito bites, holds paramount importance. Simple yet effective measures such as wearing long-sleeved shirts and trousers, utilizing insecticide-treated mosquito nets, and avoiding outdoor sleeping are essential (Balkhy and Memish, 2003). Conducting rapid assessments to ensure message adequacy and appropriateness to public needs, while also utilizing the most efficient communication channels, is imperative (Mariner, 2018).

Rigorous active surveillance and sentinel herd monitoring

Sentinel herds serve as a crucial method for gathering fundamental epidemiological data on Rift Valley Fever (RVF), having been employed across various regions in Africa to monitor viral circulation within susceptible populations. This approach can be strengthened through the additional monitoring of climatic

parameters (Davies and Martin, 2006). Efforts should focus on active disease surveillance to establish baseline information regarding inter-epidemic virus transmission patterns, identify high-risk areas, and provide early warning of increased virus activity or heightened vector mosquito populations (Health *et al.*, 2022). Such surveillance necessitates regular field visits, engagement with livestock farmers and communities, and the implementation of periodically designed and geographically representative serological surveys alongside participatory epidemiological techniques (Davies and Martin, 2006). Utilizing satellite imagery and weather and climate forecasting models to develop early warning systems for RVF prediction represents a sophisticated approach to alerting national authorities promptly, enabling them to implement crucial preventive measures against imminent epidemics (Himeidan, 2016). The advent of satellite remote sensing (RS) data has facilitated more advanced research, enabling national and regional monitoring of precipitation and climate patterns and their environmental impacts. Cold cloud density (CCD) measurements, closely associated with precipitation, contribute to this monitoring (Davies and Martin, 2006). New statistical methodologies derived from satellite data, such as the Basin Excess Precipitation Monitoring Systems (BERMS), assess rainfall in river and wadi system watersheds based on digital catchment and river network maps (Nanyingi *et al.*, 2015). BERMS can forecast potential flooding periods, particularly beneficial for floodplain zones in Horn of Africa countries and the Arabian Peninsula. Early indications suggest BERMS may forecast virus activity up to five months in advance (Clark *et al.*, 2018). Retrospective analysis of RVF outbreaks in Somalia and northeast Kenya during 1997-98 revealed a correlation between RVF virus activity and high NDVI (Normalized Difference Vegetation Index) values. Combining surface sea temperatures (SST) from the Indian and Pacific Oceans with NDVI data demonstrated close to 100 percent accuracy in predicting periods of RVF virus activity (Davies and Martin, 2006).

Vaccinations

Vaccination of ruminants stands out as the primary approach for preventing human Rift Valley Fever

(RVF) infection, representing the most effective strategy against the disease. Early identification of periods with heightened disease risk is feasible and should serve as the foundation for strategic vaccination campaigns (Kwaśnik *et al.*, 2021). Given the trade embargo on animals during outbreaks, it is crucial for commercial livestock vaccine development to differentiate between naturally infected and vaccinated animals (Mandell and Flick, 2011).

Formalin-inactivated vaccines, originally used to safeguard laboratory workers from accidental exposure since the 1960s, have been adapted for veterinary use. These vaccines, produced by passage in BHK-21 hamster kidney cells, offer moderate protection and are more costly to manufacture (Kwaśnik *et al.*, 2021). The formulation of inactivated vaccines mixed with aluminum hydroxide gel as an adjuvant holds the advantage of suitability for use in pregnant ewes. Despite the poor antibody response in cattle, inactivated vaccines are recommended to confer colostral immunity to offspring, requiring multiple inoculations and frequent booster shots for optimal protection (Chevalier *et al.*, 2010). Boosters every three to six months post-initial vaccination, followed by annual boosters, is necessary (Davies and Martin, 2006). To address limitations, live-attenuated vaccines such as MP-12 and Clone 13 strains were developed and tested in the 1980s and 1990s. While these vaccines provide protection against virulent infection, there is a risk of teratogenic effects in pregnant animals, with up to 30 percent experiencing miscarriage or fetal abnormalities (Davies and Martin, 2006). Additionally, live-attenuated vaccines may revert to virulence and facilitate animal-to-animal transmission during epidemics. However, MP-12 is still under research as a potential human vaccine, and reverse genetics have enabled the creation of rationally designed live attenuated vaccines (Kitandwe *et al.*, 2022). Recombinant virus variants with deletions in NSs and NSm proteins show promise in rat and sheep models with no apparent adverse effects on fetuses (Hartman, 2017).

Novel approaches involve removing the NSm protein from the MP-12 virus, allowing for differentiation between infected and vaccinated animals (DIVA) based on antigen-based immune responses detected

via ELISA (Kitandwe *et al.*, 2022). Other strategies include vectored, replicon, and subunit vaccinations, with a replication-defective chimpanzee adenovirus-based vaccine expressing RVFV glycoproteins demonstrating efficacy in various ruminant species. These next-generation candidates hold potential for both veterinary and human use, although significant financial investment from endemic countries is necessary to enable veterinary applications (Hartman, 2017). Routine vaccination outside of pregnancy is advised, while vaccination following confirmation of epizootic virus activity is not recommended due to the risk of needle propagation of the virus (Davies and Martin, 2006).

Vector control

Enhanced mosquito control measures are imperative in regions affected by both epizootic and human Rift Valley Fever (RVF) activity (Kasye *et al.*, 2016). Larvicide treatments offer a viable control approach, particularly in areas where mosquito breeding sites are well-defined and cover limited surface areas. Commercially available *larvicides* such as Methoprene, a hormonal larval growth inhibitor, and *Bacillus thuringiensis israeliensis* (BTI) preparations, a microbial larvicide, have demonstrated success in treating temporary ponds and watering places where mosquitoes thrive (Chevalier *et al.*, 2010; Davies and Martin, 2006). However, adulticide treatments, such as those employing pyrethroids, pose challenges due to their costliness and logistical complexity. The widespread application of ultra-low volume insecticide sprays via vehicle or aerial methods often yields limited effectiveness in reducing RVF transmission rates or targeting adult mosquito species, particularly in areas with expansive floods (Davies and Martin, 2006). Moreover, the environmental and ecological ramifications of treating large areas with insecticides must be carefully considered (Chevalier *et al.*, 2010).

Movement controls

The migration of infected vectors, individuals, and animals can facilitate the spread of Rift Valley Fever (RVF) into non-endemic regions. A clinical epizootic of RVF in the Sahel region was linked to the movement of nomadic cattle and seasonal migrations of herdsmen (Alhaji *et al.*, 2020). Preventive measures should encompass restrictions on animal movements, UniversePG | www.universepg.com

controlling or avoiding the slaughter and butchering of ruminants, employing insect repellents and bed nets during outbreaks, conducting information campaigns, and enhancing targeted surveillance of animals, humans, and vectors (Chevalier *et al.*, 2010). While these measures may not alter the course of an outbreak within an infected country, they hold relevance for managing the movement of animals for trade from areas where RVF virus transmission is ongoing (Davies and Martin, 2006).

The Economic Consequences of Rift Valley Fever **A Global Perspective Economic Impact of Rift Valley Fever**

Rift Valley Fever (RVF) inflicts substantial economic burdens across various sectors globally. Primarily affecting livestock, RVF outbreaks result in significant losses due to animal morbidity, mortality, and reduced productivity (OIE, 2019). These losses extend to farmers through decreased fertility, milk production, and veterinary costs for control measures like vaccination campaigns. Moreover, trade restrictions imposed on countries grappling with RVF outbreaks lead to further economic strain by disrupting export markets (WTO, 2010). On the human front, RVF outbreaks strain public health systems, driving up healthcare expenditures due to hospitalizations, treatment costs, and loss of productivity (Anyamba & Linthicum, 2016). Investments in surveillance, vector control, and public awareness campaigns divert funds from other health priorities, compounding economic challenges. Consequently, the cumulative impact of RVF on agriculture, public health, and trade underscores the urgent need for coordinated efforts to mitigate its economic ramifications and safeguard livelihoods and economies globally.

Impact on Agriculture

Rift Valley Fever (RVF) outbreaks have profound implications for agriculture, particularly in regions where the virus is endemic. The economic impact on agriculture stems from various factors, including direct losses due to animal morbidity and mortality, as well as indirect effects such as decreased productivity and trade restrictions. RVF primarily affects livestock, causing abortions, reduced fertility, and decreased milk production in infected animals (OIE, 2019). These consequences result in significant financial

losses for farmers who rely on livestock for their livelihoods. Additionally, control measures implemented during RVF outbreaks, such as animal movement restrictions and vaccination campaigns, entail additional costs for both farmers and governments (OIE, 2019). Furthermore, the imposition of trade restrictions on livestock and animal products from regions experiencing RVF outbreaks exacerbates the economic impact on agriculture by disrupting export markets and diminishing revenue opportunities for producers (WTO, 2010). Thus, RVF poses substantial challenges to the agricultural sector, highlighting the need for comprehensive strategies to mitigate its effects and protect agricultural economies.

Impact on Public Health Systems

Rift Valley Fever (RVF) outbreaks exert significant pressure on public health systems, resulting in increased healthcare expenditures and resource allocation. The impact on public health is multifaceted, encompassing both direct consequences of human infections and indirect effects on healthcare infrastructure and services. Human cases of RVF can lead to hospitalizations, treatment costs, and loss of productivity due to illness or death (Anyamba and Linthicum, 2016). The management of RVF outbreaks necessitates surveillance activities, vector control measures, and public awareness campaigns, all of which require substantial financial resources and personnel. These efforts divert funds from other health priorities, potentially compromising the ability of public health systems to respond effectively to other disease threats. Furthermore, long-term health complications in survivors of RVF infections can impose additional healthcare burdens, including ongoing medical care and rehabilitation services. Thus, RVF outbreaks strain public health systems, highlighting the importance of robust preparedness and response strategies to mitigate their impact on human health (FAO, 2013).

Impact on Trade

Rift Valley Fever (RVF) outbreaks have far-reaching implications for international trade, particularly in regions where the virus is endemic. The impact on trade stems from various factors, including trade restrictions imposed on livestock and animal products from affected regions. During RVF outbreaks, impor-

ting countries often enact stringent measures such as bans or limitations on the importation of livestock and animal products to prevent the spread of the virus (WTO, 2010). These trade restrictions disrupt supply chains, leading to market instability and reduced revenue for producers in the affected regions. Moreover, importing countries may incur higher costs for sourcing alternative products or implementing quarantine measures, further complicating trade dynamics (WTO, 2010). Additionally, the loss of consumer confidence in products originating from RVF-affected areas can significantly impact trade volumes and market demand. Consequently, RVF outbreaks pose significant challenges to international trade, highlighting the need for coordinated efforts to facilitate trade while safeguarding public health (OIE, 2019).

CONCLUSION AND RECOMMENDATIONS:

Rift Valley Fever (RVF) presents formidable economic challenges, impacting both human health and livestock productivity. The disease imposes direct losses through animal morbidity and mortality, while indirect costs arise from trade restrictions, healthcare expenses, and disruptions to livelihoods, particularly in agriculture and livestock-dependent regions. RVF's cyclical epidemics, reliant on vectors for transmission, affect various animal species, including humans, with individuals in occupational roles like livestock handlers, slaughterhouse workers, and veterinarians being particularly vulnerable. Vaccination and vector control serve as primary strategies for managing RVF outbreaks, yet the absence of effective prophylactic and therapeutic measures underscores its gravity as a public health concern. Ethiopia's geographical proximity to RVF-endemic countries, coupled with cross-border trade and pastoralist movements, heightens the risk of clinical RVF outbreaks in East Africa during epizootic periods. Despite positive attitudes toward RVF prevention among pastoralist communities, knowledge gaps persist, hindering effective preventive practices. Hesitancy to report RVF suspicions due to compensation system shortcomings further complicates control efforts, fragmenting community involvement in disease management and increasing transmission risks. Countries affected by RVF often exhibit high dependence on livestock and face economic,

infrastructure, and capacity challenges, hindering effective prevention, detection, and response measures. Political focus on livestock producers' financial losses overlooks downstream impacts, such as those on butchery and slaughterhouse operations. Comprehensive disease impact studies are crucial for informing decision-makers and guiding efficient resource allocation, yet limited research exists on RVF seroprevalence and economic impacts. Existing studies suffer from biases and data limitations, hampering efforts to identify vulnerable groups and develop targeted protective measures.

Based on the above conclusions the following recommendations are forwarded:

- Strengthen Surveillance Systems: Implement robust surveillance systems to monitor RVF outbreaks in both human and animal populations. Early detection is crucial for timely intervention and containment measures.
- Enhance Veterinary Services: Invest in veterinary infrastructure and capacity-building programs to improve disease management and prevention strategies among livestock populations.
- Promote Vaccination Programs: Implement widespread vaccination campaigns targeting susceptible livestock species to mitigate the spread of RVF and minimize economic losses associated with disease outbreaks.
- Improve Public Health Education: Raise awareness among communities about RVF transmission, symptoms, and preventive measures to reduce human infection rates and healthcare costs.
- Foster International Collaboration: Facilitate cross-border cooperation and information sharing to prevent the spread of RVF across regions and mitigate its economic impacts on a global scale.
- Develop Contingency Plans: Develop and regularly update contingency plans for RVF outbreaks to ensure swift and coordinated responses, minimizing economic disruptions and maximizing containment efforts.

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CONFLICTS OF INTEREST:

The authors declare that there is no conflict of interest.

REFERENCES:

- 1) Abedin MZ, Jarin L, Hasan R, et al. (2021): Analysis of the Dengue Infection, Occurrence and Hematological Profile of Dengue Patients in Dhaka City, *J Microbiol Pathol*. **5**: 115. www.hilarispublisher.com/open-access/analysis-of-the-dengue-infection-occurrence-and-hematological-profile-of-dengue-patients-in-dhaka-city.pdf
- 2) Abdi, I.H., Affognon, H.D., Wanjoya, A.K., Onyango-Ouma, W., Sang, R. (2015). Knowledge, Attitudes and Practices (KAP) on rift valley fever among pastoralist communities of Ijara District, North Eastern Kenya. *PLoS Neglected Tropical Diseases*, **9**, e0004239.
- 3) Adamu, A.M., Allam, L., Sackey, A.K., Nma, A.B., Simon, A.Y. (2021). Risk factors for Rift Valley fever virus seropositivity in one-humped camels (*Camelus dromedarius*) and pastoralist knowledge and practices in Northern Nigeria. *One Health*, **13**.
- 4) Alhaji, N.B., Aminu, J., Lawan, M.K., Odetokun, I.A. (2020). Seropositivity and associated intrinsic and extrinsic factors for Rift Valley fever virus occurrence in pastoral herds of Nigeria: a cross sectional survey. *BMC Veterinary Research*, **16**.
- 5) Anyamba, A., & Linthicum, K. J. (2016). Rift Valley Fever Virus: A Historical Overview and Recent Epizootics in Southern Africa. *American Journal of Tropical Medicine and Hygiene*, **94**(2), 269–273. <https://doi.org/10.4269/ajtmh.15-0757>
- 6) Anyamba, A., Chretien, J. P., Small, J., & Breiman, R. F. (2009). Prediction of a Rift Valley fever outbreak. *Proceedings of the National Academy of Sciences*, **106**(3), 955-959.
- 7) Anyango, V. O., Gould, L. H., Omolo, J. O., Mutonga, D., & Njenga, M. K. (2020). Rift

- Valley fever in Kenya: History of epizootics and outbreaks. *Open Veterinary Journal*, **10**(3), 226-236.
- 8) Anywaine, Z., Lule, S.A., Hansen, C., Warimwe, G., Elliott, A., 2022. Clinical manifestations of Rift Valley fever in humans: Systematic review and meta-analysis. *PLoS Neglected Tropical Diseases*, **16**, e0010233.
 - 9) Anywaine, Z., Lule, S.A., Hansen, C., Elliott, A., 2022. Clinical manifestations of Rift Valley fever in humans: Systematic review and meta-analysis. *PLoS Neglected Tropical Diseases*, **16**, e0010233.
 - 10) Avensonline, (2014). Change on the Epidemiology of Rift Valley Fever.
 - 11) Balkhy, H.H., Memish, Z.A. (2003). Rift Valley fever: an uninvited zoonosis in the Arabian peninsula. *International journal of antimicrobial agents*, **21**, 153-157.
 - 12) Bird, B. H., Ksiazek, T. G., & Nichol, S. T. (2009). Rift Valley fever virus. *Journal of Virology*, **85**(8), 3717–3725.
 - 13) Centers for Disease Control and Prevention (CDC), (2019). Rift Valley Fever (RVF).
 - 14) Cêtre-Sossah, C., Pédarrieu, A., Paweska, J., Cardinale, E. (2019). Development and validation of a pen side test for Rift Valley fever. *PLoS Neglected Tropical Diseases*, **13**.
 - 15) Chevalier, V., Pépin, M., Plee, L., Lancelot, R., 2010. Rift Valley fever-a threat for Europe? *Eurosurveillance*, **15**.
 - 16) Clark, M.H., Warimwe, G.M., Di Nardo, A., Lyons, N.A., Gubbins, S. (2018). Systematic literature review of Rift Valley fever virus seroprevalence in livestock, wildlife and humans in Africa from 1968 to 2016. *PLoS Neglected Tropical Diseases*, **12**, e0006627.
 - 17) Davies, F.G., Martin, V. (2006). Recognizing rift valley fever. *Vet Ital*, **42**, 31-53.
 - 18) European Food Safety Authority (EFSA), (2013). Systematic literature review on the geographic distribution of rift.
 - 19) Fontenille, D., Fournet, F., & Zeller, H. G. (2011). Rift Valley fever in the Comoros Islands. *Emerging Infectious Diseases*, **17**(7), 1319–1320.
 - 20) Fontenille, D., Traore-Lamizana, M., Diallo, M., & Zeller, H. G. (1998). New vectors of Rift Valley fever in West Africa. *Emerging Infectious Diseases*, **4**(2), 289–293.
 - 21) Food and Agriculture Organization of the United Nations (FAO), (2013). Rift Valley Fever: A Strategy for the African Continent. Retrieved from – <http://www.fao.org/docrep/017/aq236e/aq236e.pdf>
 - 22) Food and Agriculture Organization of the United Nations (FAO). (2014). Rift Valley Fever.
 - 23) Fyumagwa, R.D., Ezekiel, M.J., Moshiro, C., Keyyu, J.D. (2011). Response to Rift Valley fever in Tanzania: challenges and opportunities. *Tanzania journal of health research*, **13**.
 - 24) Ganaie, S.S., Schwarz, M.M., Hartman, A.L. (2021). Lrp1 is a host entry factor for Rift Valley Fever Virus. *Cell*, **184**, 5163-5178. e5124.
 - 25) Grossi-Soyster, E.N., LaBeaud, A.D. (2020). Rift Valley fever: Important considerations for risk mitigation and future outbreaks. *Tropical medicine and infectious disease*, **5**, 89.
 - 26) Hartman, A. (2017). Rift Valley Fever. *Clinics in laboratory medicine*, **37**, 285-301.
 - 27) Hassan, A., Muturi, M., Mwatondo, Munyua, P. (2020). Epidemiological Investigation of a Rift Valley Fever Outbreak in Humans and Livestock in Kenya, 2018. *The American journal of tropical medicine and hygiene*, **103**, 1649-1655.
 - 28) Health, E.P.O.A., Welfare, Nielsen, S.S., Gortázar, C. (2022). Assessment of the control measures for category A diseases of Animal Health Law: Contagious Bovine Pleuropneumonia. *EFSA Journal*, **20**, e07067.
 - 29) Health, E.P.o.A., Welfare, Nielsen, S.S., Gortázar Schmidt, C. (2020). Rift Valley Fever–assessment of effectiveness of surveillance and control measures in the EU. *EFSA Journal*, **18**, e06292.
 - 30) Health, E.P.o.A., Welfare, Nielsen, S.S., Gortázar, C., 2022. Assessment of the control measures for category A diseases of Animal Health Law: Contagious Bovine Pleuropneumonia. *EFSA Journal*, **20**, e07067.

- 31) Health, E.P.o.A., Welfare, Nielsen, S.S., Gortázar Schmidt, C. (2020). Rift Valley Fever—assessment of effectiveness of surveillance and control measures in the EU. *EFSA Journal*, **18**, e06292.
- 32) Himeidan, Y.E. (2016). Rift Valley fever: current challenges and future prospects. *Research and Reports in Tropical Medicine*, 1-9.
- 33) Ibrahim, M., Schelling, E., Tschopp, R. (2021). Sero-prevalence of brucellosis, Q-fever and Rift Valley fever in humans and livestock in Somali Region, Ethiopia. *PLoS Neglected Tropical Diseases*, **15**.
- 34) Ikegami, T., & Makino, S. (2011). The pathogenesis of Rift Valley fever. *Viruses*, **3**(5), 493–519.
- 35) Javelle, E., Lesueur, A., Texier, G., Simon, F. (2020). The challenging management of Rift Valley Fever in humans: literature review of the clinical disease and algorithm proposal. *Annals of Clinical Microbiology and Antimicrobials*, **19**, 4.
- 36) Kahlon, S. S., Peters, C. J., LeDuc, J. W., & Muchiri, E. M. (2012). Rift Valley Fever in Kenya: History of Epizootics and Identification of Vulnerable Districts. Research Gate.
- 37) Kasye, M., Teshome, D., Abiye, A., Eshetu, A. (2016). A Review on Rift Valley Fever on Animal, Human Health and its Impact on Live Stock Marketing. *Austin Virology and Retrovirology*, **3**, 1020.
- 38) Kifaro, E.G., Kasanga, C.J., Yongolo, M., Dautu, G. (2014). Epidemiological study of Rift Valley fever virus in Kigoma, Tanzania: proceedings. *Onderstepoort Journal of Veterinary Research*, **81**, 1-5.
- 39) Kimani, T., Schelling, E., Bett, B., Ngigi, M., Randolph, T., Fuhrimann, S. (2016). Public Health Benefits from Livestock Rift Valley Fever Control: A Simulation of Two Epidemics in Kenya. *Ecohealth*, **13**, 729-742.
- 40) Kitandwe, P.K., McKay, P.F., Kaleebu, P., Shattock, R.J. (2022). An Overview of Rift Valley Fever Vaccine Development Strategies. *Vaccines*, **10**, 1794.
- 41) Kwaśnik, M., Rożek, W., Rola, J. (2021). Rift Valley fever—a growing threat to humans and animals. *Journal of Veterinary Research*, **65**, 7-14.
- 42) LaBeaud, A. D., Cross, P. C., Sall, A. A. (2011). Rift Valley fever virus infection in African buffalo (*Syncerus caffer*) herds in rural South Africa: evidence of interepidemic transmission. Research Gate.
- 43) LaBeaud, A. D., Kazura, J. W., & King, C. H. (2015). Advances in Rift Valley fever research: Insights for disease prevention. *Current Opinion in Infectious Diseases*, **28**(5), 437–445.
- 44) LaBeaud, A. D., Kazura, J. W., & King, C. H. (2015). Rift Valley Fever: An Emerging Mosquito-Borne Disease. *Annual Review of Medicine*, **67**, 399-413.
- 45) Linthicum, K. J., Davies, F. G., Kairo, A., Bailey, C. L., & Kalluri, S. (2016). Rift Valley fever virus (family Bunyaviridae, genus Phlebovirus). In *Handbook of Zoonoses*, CRC Press, pp. 149-159.
- 46) Lumley, S., Horton, D.L., Fooks, A.R., Hewson, R. (2017). Rift Valley fever virus: strategies for maintenance, survival and vertical transmission in mosquitoes. *Journal of General Virology*, **98**, 875-887.
- 47) Machalaba, C. (2020). Impacts of Rift Valley Fever virus: a One Health approach to assess burden and inform prevention and control options.
- 48) Małgorzata K, W.R., Jerzy Rola, (2021). Rift Valley fever – a growing threat to humans and animals. *J Vet Res*, **65**, 7-14.
- 49) Mandell, R., Flick, R. (2011). Rift Valley fever virus: a real bioterror threat. *J Bioterr Biodef*, **2**.
- 50) Mariner, J. (2018). Rift Valley Fever Surveillance; FAO Animal Production and Health Manual No 21; Food and Agriculture Organization of the United Nations: Rome, Italy, 2018 (ISBN 978-92-5-130244-6).
- 51) Métras, R., Jewell, C., Collins, L.M., White, R.G. (2015). Risk factors associated with Rift Valley fever epidemics in South Africa in 2008–11. *Scientific reports*, **5**, 1-7.
- 52) Michaely, L.M., Rissmann, M., Gutjahr, B., Baumgärtner, W., Groschup, M.H. (2022). Rift Valley Fever Virus Non-Structural Protein S Is Associated with Nuclear Translocation of

- Active Caspase-3 and Inclusion Body Formation. *Viruses*, **14**, 2487.
- 53) Musser, J., Coetzer, J., Burnham, S. (2005). Rift Valley Fever Symptoms. *Sciences*.
- 54) Nabeth, P., Kane, Y., Ba, K., McCormick, J. B. (2010). Rift Valley fever outbreak, Mauritania, 1998: seroepidemiologic, virologic, entomologic, and zoologic investigations. *Emerging Infectious Diseases*, **8**(12), 1–6.
- 55) Nanyingi, M. O., Munyua, S. M., Bitek, A. O. (2015). A systematic review of Rift Valley Fever epidemiology 1931-2014. *Infection Ecology & Epidemiology*, **5**(1), 28024.
- 56) Nielsen SS, Alvarez J, B.D., Calistri P, and, Y, V.d.S. (2020). Scientific Opinion on Rift Valley Fever – assessment of effectiveness of surveillance and control measures in the EU. *EFSA Journal*; **18**(11): 6292, 75.
- 57) Pachka, H., Annelise, T., Véronique, C., Janusz, P., Ferran, J. (2016). Rift Valley fever vector diversity and impact of meteorological and environmental factors on *Culex pipiens* dynamics in the Okavango Delta, Botswana. *Parasites & Vectors*, **9**.
- 58) Pal, M., Gutama, K.P., Tolawak, D. (2021). Rift Valley fever: A tropical emerging viral zoonosis of public health concern. *Community Medicine and Health Education Research*, **2**, 52-59.
- 59) Paweska, J. T., Smith, S. J., Wright, I. M., Swanepoel, R. (2008). Indirect Fluorescent Antibody Test for the Detection of Rift Valley Fever Virus in Blood Specimens from Wild and Domestic Ruminants. *Journal of Virological Methods*, **149**(1), 45–51.
- 60) Paweska, J.T. (2014). Rift valley fever, In: *Emerging Infectious Diseases*. Elsevier, 73-93.
- 61) Pepin, M., Bouloy, M., Bird, B. H., Kemp, A., Paweska, J. (2010). Rift Valley fever virus (*Bunyaviridae: Phlebovirus*): an update on pathogenesis, molecular epidemiology, vectors, diagnostics and prevention. *Veterinary Research*, **41**(6), 61.
- 62) Petrova, V., Kristiansen, P., Norheim, G., Yimer, S.A. 2020. Rift valley fever: diagnostic challenges and investment needs for vaccine development. *BMJ Global Health*, **5**, e002694.
- 63) Peyre, M., Chevalier, V., Thiry, E., Roger, F. 2015. A systematic scoping study of the socio-economic impact of Rift Valley fever: research gaps and needs. *Zoonoses and public health*, **62**, 309-325.
- 64) Rich, K. M., Wanyoike, F. (2010). An assessment of the regional and national socio-economic impacts of the 2007 Rift Valley fever outbreak in Kenya. *The American Journal of Tropical Medicine and Hygiene*, **83**(2_Suppl), 52-57.
- 65) Rich, K. M., Wanyoike, F., & Ankers, P. (2017). Rift Valley fever: strategies for prevention and control. *Epidemics*, **18**, 32-37.
- 66) Rolin, A.I., Berrang-Ford, L., Kulkarni, M.A. 2013. The risk of Rift Valley fever virus introduction and establishment in the United States and European Union. *Emerging microbes & infections*, **2**, 1-8.
- 67) Routray, A., Rath, A.P., Panigrahi, S., Lambe, U.P., Sahoo, S., Ganguly, S. 2017. RIFT Valley Fever: An Update. *International Journal of Contemporary Pathology*, **3**, 20.
- 68) Sindato, C., Karimuribo, E., Mboera, L.E. 2011. The epidemiology and socio-economic impact of Rift Valley fever in Tanzania: a review. *Tanzania journal of health research*, **13**.
- 69) Smith, D. R., Steele, K. E., Twenhafel, N. A., & Van Wyke, K. L. (1991). Rift Valley Fever Virus: The Northern Extension of Its Range in Africa. Research Gate.
- 70) Tantely, L.M., Boyer, S., Fontenille, D. 2015. A Review of Mosquitoes Associated with Rift Valley Fever Virus in Madagascar. *The American journal of tropical medicine and hygiene*, **92**, 722-729.
- 71) Turell, M.J., Wilson, W.C., Bennett, K.E. 2010. Potential for North American mosquitoes (Diptera: Culicidae) to transmit rift valley fever virus. *Journal of medical entomology*, **47**, 884-889.
- 72) Van den Bergh, C., Thompson, P.N., Venter, E.H. 2022. Detection of Rift Valley Fever Virus in *Aedes* (*Aedimorphus*) *durbanensis*, South Africa. *Pathogens*, **11**, 125.
- 73) Wanyoike, F.N., Dizyee, K., Bett, B.K., Rich, K.M. 2021. Application of system dynamics

- modelling in the analysis of economic impacts of Rift Valley fever: A case study of Ijara County, Kenya. *Tropical Diseases*, **9**, e0004239.
- 74) World Health Organization (WHO), (2017). Rift Valley fever.
- 75) World Health Organization (WHO), (2018). Rift Valley Fever – Kenya. Disease outbreak news.
- 76) World Organization for Animal Health (OIE), (2019). Rift Valley Fever: Technical Disease Card. Retrieved from – https://www.oie.int/fileadmin/Home/eng/Animal_Health_in_the_World/docs/pdf/Disease_cards/RIFT_VALLEY_FEVER.pdf
- 77) World Organization for Animal Health (OIE), (2020). Rift Valley fever. OIE Technical Disease Cards.
- 78) World Trade Organization (WTO), (2010). Trade and Animal Disease: Economic Impacts and Responses. Retrieved from – https://www.wto.org/english/res_e/reser_e/ersd201002_e.html
- 79) Wright, D., Kortekaas, J., Bowden, T.A., Warimwe, G.M., 2019. Rift Valley fever: biology and epidemiology. *The Journal of general virology*, **100**, 1187-1199.
- 80) Xiao, Y., Beier, J.C., Ruan, S., 2015. Modelling the Effects of Seasonality and Socioeconomic Impact on the Transmission of Rift Valley Fever Virus. *PLoS Neglected Tropical Diseases*, **9**.
- 81) Xu, Y., Wang, X., Jiang, L., Zhou, Y., Liu, Y., Wang, F., Zhang, L., 2023. Natural hosts and animal models for Rift Valley fever *Phlebovirus*. *Frontiers in Veterinary Science*, **10**.

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