Risk Evaluation of Rift Valley Fever virus (RVFV) lineages and their Public Health Impact, 27 May 2026

This risk evaluation summarizes current evidence on Rift Valley fever (RVF) virus, provides a risk evaluation based on the latest evidence, assesses the public health risk posed by currently circulating RVF viruses, highlights key evidence on control measures, identifies existing knowledge gaps, recommends priority studies. This risk evaluation draws on knowledge gained through recent outbreaks and analyses from the World Health Organization (WHO), Food and Agriculture Organization of the United Nations (FAO), World Organisation for Animal Health (WOAH), and partners.

In summary, currently, there is no available evidence to suggest that RVFV evolution is causing increased virulence, immune escape, vaccine failure, or diagnostic escape. The main challenges for RVFV remain in efforts for preparedness, surveillance, sustained preventive vaccination of livestock in hotspot areas, and early warning rather than viral evolution itself.

Emergence, epidemiology and transmission

RVF virus (RVFV) is a mosquito-borne *phlebovirus* (family *Phenuiviridae*, order *Hareavirales*) that primarily infects domestic ruminants—such as sheep, goats, cattle, and camels—resulting in significant economic losses, particularly through its impact on livestock production and trade. It can also cause disease in humans.[1]

Rift Valley fever has a well-documented history of causing significant human health crises. The disease was first identified in Kenya in 1930 and remained largely confined to East and Central Africa for decades.[2] However, in 2000, RVFV crossed a major geographical barrier when it was identified outside Africa for the first time on the Arabian Peninsula,[3] marking a critical shift in its global distribution. The most devastating outbreak occurred in Egypt in 1977–1978, with an estimated 200,000 infection and 600 deaths.[4] In recent decades, Eastern Africa has experienced repeated large outbreaks. The 2006–2007 outbreak affected Kenya, Tanzania, and Somalia with particularly severe consequences.[5] Southern Africa, including Namibia, South Africa, Madagascar and Mayotte have also experienced RVF outbreaks in humans.[6–9] Uganda reported a striking transition from 50 years without RVF to 10 sporadic outbreaks between 2016 and 2018 with high case fatality ratios.[10] More recently, South Sudan documented an outbreak in 2017-2018, [11] and Rwanda documented large outbreaks in 2018 and 2022 .[12,13] These outbreaks underscore RVFV's erratic emergence pattern and expanding geographic footprint,[14,15] with regular evidence of inter-epidemic transmission documented by human and animal serosurveys.[16]

A Joint WHO/FAO/WOAH rapid risk assessment published in October 2025 reported a major multicountry Rift Valley fever (RVF) outbreak affecting Mauritania and Senegal.[17] By the end of December 2025, Mauritania had recorded 53 human infections, including 15 deaths (CFR 28%), along with 68 animal outbreaks across 12 regions. In Senegal, 558 human cases had been reported as of 28 December 2025, including 51 severe cases and 31 deaths (CFR 5.5%), with transmission documented in 11 of the country's 14 regions; no consolidated national totals beyond December 2025 were available at the time of reporting.[17] Although early data suggested high severity—particularly in Mauritania—this pattern was likely driven by early detection bias, delayed access to clinical care, and limited availability of supportive treatment, rather than evidence of increased viral virulence. Transmission was concentrated along the Senegal River basin, where shared ecological and economic systems facilitate cross-border spread, and extended into eastern Senegal near the border with Mali, raising concerns about possible regional expansion. However, no human or animal cases were reported outside Mauritania and Senegal. Genomic analyses conducted during the event indicated no major mutations in known virulence-associated markers, supporting the interpretation that the outbreak's impact reflected epidemiological and health-system factors rather than viral evolution.[17]

Transmission cycles and vectors

RVFV maintains a complex transmission cycle involving both mosquitoes and vertebrate hosts.

a) Vector-borne transmission

RVFV is transmitted by multiple mosquito genera, especially *Aedes* (notably floodwater species) and *Culex*. *Aedes* spp. can maintain RVFV through vertical (trans-ovarial) transmission in eggs that survive dry periods and hatch during heavy rain, seeding new outbreaks after long inter-epidemic periods. Multiple other mosquito species and biting flies can act as secondary or bridge vectors in different ecological settings.[18] Since 1985, the transovarial transmission hypothesis has been influential, proposing that RVFV can be maintained infected, desiccation-resistant eggs of floodwater-breeding *Aedes* spp. mosquitoes. Under this model, above-average rainfall and flooding trigger mass hatching events, and infected adult mosquitoes initiate localized transmission to ruminants—often livestock. Subsequent amplification occurs through horizontal transmission by secondary biological vectors (such as *Culex* and other culicine mosquitoes), and potentially through mechanical transmission by other blood-feeding arthropods, leading to extensive infection within herds and flocks. However, a growing body of recent work suggests that this mechanism may not fully explain RVFV persistence across all ecological settings. An alternative pathway posits that while transovarial transmission may contribute to maintenance, it is not necessarily the sole or even the primary mechanism in every ecosystem. Instead, RVFV may persist through continuous, low-level cycling among mosquitoes, livestock, humans, and possibly herbivorous wildlife. This cryptic, multi-host transmission could sustain the virus in the absence of major flooding events, resulting in ongoing low-level RVF activity across broad geographic regions of Africa.[19]

b) Animal-animal and animal-human transmission

Livestock can be infected primarily through mosquito bites. High viremia in ruminants amplifies virus circulation. Humans are infected through bites of infected mosquitoes, and direct contact with blood, organs, aborted fetuses or aerosols from infected animals (e.g. at slaughterhouses, in veterinary care, and through the handling of carcasses). High-risk groups include adults—particularly males—who have close contact with animals or animal products, especially in occupational or behavioral contexts such as farming, hunting, and livestock handling.[20]

c) Human-to-human transmission

To date, there is no documented evidence that supports the occurrence of human-to-human transmission of RVFV, and the risk of nosocomial transmission of RVFV is considered lower than other viruses causing haemorragic fevers such as Ebola disease.[21]

The mechanisms that allow Rift Valley fever virus to persist between outbreaks remain incompletely understood.

Genomic characteristics and lineages

RVFV has a tripartite, negative-sense RNA genome consisting of the L segment, which encodes the viral RNA-dependent RNA polymerase; the M segment, which encodes the Gn and Gc glycoproteins and the NSm protein and is the basis for lineage classification; and the S segment, which encodes the nucleoprotein (N) and the NSs virulence factor. [22] Genes involved in replication (RdRp), envelope formation (Gn/Gc), and immune evasion (NSs) have elevated substitution rates, potentially driving phenotypic diversification, although antigenic diversity among RVFV lineages remains limited.[23]

Molecular clock analysis estimated the time to the most recent common ancestor (tMRCA) of RVFV to be around 1918 (95% highest posterior density [HPD] 1902–30).[24] Although RVFV has 15 RVFV lineages (A–O), only lineages A, C, and H have been associated with human outbreaks since 1985.[24–26] The tMRCA for lineages A, C, and H were 1971 (1968–73), 1970 (1966–75), and 2007 (2006–08), respectively.[24] Lineage A has been reported in Northern Africa, particularly during the 1977 outbreak in Egypt, while Lineage H is more commonly found in Southern Africa (e.g. 2010-2011 outbreak in South Africa),[24] and was the lineage associated with the most recent outbreak in Senegal.[27] Lineage C, probably originated in Zimbabwe in the mid-1970, with Kenya playing a major role in subsequent eastern African dissemination. Currently it is predominantly found in eastern Africa, with South African outbreaks appearing to result from distinct introduction events originating independently from both Zimbabwe and Kenya.[24] Lineage C is characterized by continued diversification into new sub lineages occurring even during interepidemic periods via cryptic transmission. [24,28]

Reassortment between RVFV strains is possible; however, there is no clear evidence that specific reassortants are driving major changes in virulence or transmissibility at a global scale.[29] RVF viruses showed low genetic diversity (≤5.4% nucleotide; ≤2.8% amino acid), indicating high conservation and likely cross-protective antigenicity.[30]

Vector distribution and ecological drivers

RVFV circulation is shaped by a combination of ecological conditions, vector dynamics, climatic variability, and human-driven land-use and livestock systems.[31,32] These factors influence both (i) the establishment of new primary foci, where the virus can persist through vertical transmission in floodwater Aedes *spp.*, and (ii) the development of secondary foci, where other mosquito species amplify transmission once livestock viremia increases.

Ecological and climatic drivers shaping primary foci (establishment and maintenance)

Ecological conditions determine where RVFV can become established. [33] Floodwater *Aedes* spp. lay desiccation-resistant eggs in temporary depressions (dambos), and their capacity for vertical transmission enables long-term viral maintenance during inter-epidemic periods. Climatic variability—such as above-average rainfall, El Niño events, and sustained flooding—creates extensive breeding sites that trigger synchronous hatching of infected *Aedes* cohorts, initiating new primary foci. [19] Increasing reports of inter-epidemic clusters, often during or shortly after rainy seasons, highlight the sensitivity of these systems to rainfall anomalies.

Human and livestock system drivers shaping secondary foci (amplification and spread)

Once primary foci are established, human-driven land-use patterns and livestock movements strongly influence where secondary amplification occurs. Irrigation schemes, dams, and expanding agricultural zones create permanent or semi-permanent water bodies that support large populations of *Culex* spp. and other secondary vectors.[34] Livestock production systems—particularly transhumance, grazing practices, and formal and informal trade routes—facilitate the movement of susceptible or viremic animals, enabling RVFV to spread into new ecological settings or re-seed previously affected areas. [35] These environments support explosive amplification cycles, especially when high livestock densities coincide with abundant secondary vectors.

Example from Senegal: shifting vector dynamics

Historically, *Aedes vexans* and *Culex poicilipes* were considered the main vectors in Senegal. However, entomological investigations during the 2025 outbreak showed that *Culex antennatus* was by far the most abundant species in the affected area. This aligns with recent studies indicating that northern regions—particularly in September—provide favorable conditions for RVFV emergence and identifying *Culex antennatus* as an increasingly important vector. .[36] The rising prevalence of this species may reflect underlying ecological and climatic changes, underscoring the need to continually reassess entomological risk as environmental variability increases.

In Senegal, a 2020 national serosurvey reported the highest seroprevalence in the Saint-Louis region (14%),[37] and a subsequent study (2022–2024) documented human seroprevalence of 11–23% in Matam, indicating sustained exposure. [38] In 2025, flooding along the river likely created temporary breeding sites that favored vector proliferation. Combined with insufficient entomological surveillance, these conditions may have increased the risk of both primary establishment and secondary amplification.

The Rift Valley Fever Early Warning Decision Support Tool

Risk mapping and modelling are essential for preventing RVF outbreaks and understanding disease dynamics across ecosystems. While traditional models provide only static snapshots, dynamic approaches incorporating climate variability enable continuous risk tracking by linking environmental changes to vector activity. FAO, in collaboration with the WHO and the WOAH, has long monitored RVF in sub-Saharan Africa.[39]

Building on this experience, FAO launched the RVF Early Warning Decision Support Tool (RVF DST) in 2019—a web-based platform for near real-time monitoring and forecasting. Based on a dynamic model developed by NASA, [40]and refined by FAO,[41] the tool integrates climate data, with expert input on RVF eco-epidemiology to identify near real-time high-risk areas, issue warnings up to two to three months in advance and build early warning capacity at national level.

First piloted in Kenya, Uganda, and Tanzania, it now provides regularly updated risk maps across Africa and is expanding to other diseases like avian influenza. Supported by the RVF Action Framework,[42]and monthly expert assessments, the system enables timely alerts, targeted interventions, and coordinated response. Cost-effective and user-friendly, the RVF DST facilitates continuous risk monitoring, real-time hotspots validation via data sharing and consultation at global, regional, and country levels.

A public version has been made freely available since November 2025, alongside tailored national versions for authorized users. By combining real-time data, expert analysis, and scalable design, the RVF DST strengthens preparedness, enhances risk communication, and enables faster, more effective outbreak prevention and control.

Currently, the model for vector amplification primarily focuses on dambo ecosystems; therefore, further refinement is needed to improve the tool's sensitivity and specificity across other ecological settings. Ongoing enhancements aim to address these gaps by incorporating additional factors such as livestock and human immunity, land-use changes, animal mobility, and more granular climatic variations. The RVF DST also supports capacity building at national and regional levels by strengthening training, improving risk interpretation skills, and enhancing the use of early warning information for decision-making. By doing so, it helps countries shift from reactive outbreak response to more proactive preparedness and prevention. Through continuous improvement, the tool strengthens preparedness, enhances risk communication, and supports more accurate and effective outbreak prevention and control.

Vertical transmission, blood safety and other transmission routes

Vertical transmission in vectors is well documented for RVFV (e.g. *Aedes* eggs). Vertical transmission in animals (e.g. transplacental infection in ruminants) is common, leading to abortion storms. In humans, congenital infection and adverse pregnancy outcomes have been reported.[43,44] A cross-sectional study recruited 130 pregnant Sudanese women and 15 (54%) of 28 women with an acute RVFV infection had miscarriages compared with 12 (12%) of 102 women negative for RVFV ($p<0·0001$).[45] Many epidemiological questions remain unaddressed regarding the impact of RVFV on pregnant women, and active surveillance of both early- and late-term pregnancy loss in areas where RVFV is endemic is needed, both during and in between outbreaks of disease in livestock and humans.[46]

Viremia can reach high levels during acute Rift Valley fever infection in humans, theoretically allowing transfusion-related transmission. No transfusion-derived cases have been documented to date; however, the risk remains biologically plausible given experience with other arboviruses and evidence that RVFV viremia can reach levels sufficient to infect competent vectors.[47]

Immune response, immune escape and vaccination

The main neutralizing epitopes are located on the viral glycoproteins (Gn/Gc) encoded on the M segment.[48] Current data do not suggest rapid antigenic drift or the emergence of immune-escape variants and are comparable to some other arboviruses; immunity after infection appears to be long-lasting, and cross-protection among RVFV strains is generally expected. [23]

Several live-attenuated and inactivated vaccines (Table 1) are in use for livestock in endemic countries, though each has limitations (e.g. potential teratogenicity, need for cold chain, dosing requirements, associated costs).[49] Smithburn strain-based vaccines for livestock are widely used in Africa, and have good safety records; however, they can cause abortions and congenital mutations when administered to pregnant livestock.[50] The Clone 13 vaccine, developed by NICD (South Africa) from a naturally attenuated isolate (natural NSs-deleted variant), has excellent safety data but requires further efficacy studies.[51]

Preventive vaccination in animals

Preventive vaccination in livestock can significantly reduce RVF transmission in both animals and humans, while safeguarding the livelihoods and food security of communities that depend on these herds and flocks. It remains the cornerstone of RVF control, and reducing infection in animals is consistently shown to be the most effective way to prevent human disease.

Organizations such as WOAH, FAO and WHO, support preventive animal vaccination for Rift Valley fever. Routine nationwide annual vaccination is generally not recommended, as outbreaks are intermittent, costs and logistical demands are substantial, and risk varies across regions and climatic conditions. Instead, vaccination strategies are selective and risk-based.[52] Campaigns should be triggered by early warning systems incorporating indicators such as abnormal rainfall, flooding, and satellite-derived vegetation indices, allowing pre-emptive vaccination one to three months before expected vector amplification.[53] Importantly, cost-effectiveness evidence should also be generated when feasible. Targeting is equally important: rather than vaccinating all livestock, efforts should prioritize high-risk species—particularly sheep and goats—and focus on flood-prone areas, irrigation schemes, historically affected regions, and peri-urban settings where human exposure risk is elevated. Vaccine selection must be context-specific, with live attenuated vaccines preferred for rapid, large-scale pre-outbreak campaigns (while avoiding use in pregnant animals), and inactivated vaccines reserved for valuable breeding stock, pregnancy-sensitive herds, or more controlled environments. Crucially, vaccination cannot operate in isolation and must be integrated with robust surveillance systems, including monitoring of animal health indicators such as abortion events and mortality spikes, vector density surveillance, and reporting mechanisms that enable timely response. Reactive vaccination during outbreaks is generally discouraged due to delayed onset of immunity and operational constraints, shifting the focus instead to movement control, vector management, and human protection measures.

At the same time, operational challenges such as vaccine cost, limited cold-chain capacity in many high-risk regions, practical considerations such as vaccine hesitancy, challenges in efficient distribution in remote rural areas, the availability of trained personnel for vaccine administration, the achievement of sufficient coverage and the timing are critical determinants of campaign success, requiring strong collaboration with local and international institutions as well as sustained community engagement. Although vaccination remains the primary preventive option for RVF in endemic settings, its effectiveness is often constrained by uncertainties around when and where outbreaks will occur, as well as delays in vaccine production and deployment. Consequently, complementary measures—including public education, livestock quarantine, and slaughter bans—are often among the most effective interventions for limiting disease spread during both pre-outbreak and outbreak phases.

Table 1. Veterinary vaccines against Rift Valley fever.

Vaccine Type	Examples	Safety	Efficacy	Status
Live attenuated (classic)	Smithburn	Moderate (not recommended for use in pregnant animals)	High	Licenced
Live attenuated (natural NSs-deleted variant)	Clone 13	Excellent	High in small ruminants; moderate in cattle	Licenced
Inactivated	OBP (SA), VSVRI (Egypt)	Excellent	Good (requires boosters)	Licenced

Vaccine Type	Examples	Safety	Efficacy	Status
Live attenuated (engineered)	MP-12	Very good	High	Limited licencing
Recombinant / vectored	MVA-RVFV, ΔNSs mutants	Excellent	Promising	Preclinical

Human vaccines

An inactivated human vaccine has been developed but is not licenced or commercially available, but has been used experimentally for high-risk occupational groups [54,55]Multiple candidate human vaccines are in early to mid-stage clinical development, including a candidate entering Phase II trials in Kenya under CEPI support. [56]

CEPI is currently supporting the development of four RVF vaccine candidates for humans across three platforms or modalities: two live-attenuated candidates (co-funded with the European Commission), one non-replicating viral vector (ChAdOx platform) candidate and one mRNA vaccine candidate. CEPI candidates are at various stages of development from early preclinical to nearing completion of Phase 2a studies in RVF-endemic countries. All candidates are being assessed as both single and two-dose regimens in clinical trials, and while targeting use primarily for outbreaks, potential for preventative use will be explored in line with the WHO RVF TPP and use cases identified for human RVF vaccines. Details of individual candidates are summarized in Table X.

Table. Human candidate vaccines against Rift Valley fever.

R&D lead organisation	Wageningen University & Research	UC Davis / Colorado State University	University of Oxford/ Serum Institute India	Afrigen
Development phase	Ready to enter Phase 2a	Ready to enter Phase 1	Phase IIa (in progress) Phase I/II	Pre-clinical
Platform	Live attenuated	Live attenuated	Non-replicating viral vector (ChAdOx)	mRNA
Antigen	As per wild-type RVF Δ NSs (virulence factor) 4 segment genome (wt RVF has three)	As per wild-type RVF Double Δ (NSs & NSm) virulence factors	Glycoproteins (Gn, Gc)	Glycoproteins (Gn, Gc)
Dose schedule (TBC)	1-2			
Route of administration	IM	IM	IM	IM

Target indication (TBC)	RVF outbreak response and prevention			
Clinical trial sites	Kenya & Uganda	Tanzania	Kenya & Senegal	South Africa

A recent Phase I trial in southwestern Uganda found that a single intramuscular dose of the ChAdOx1 RVF vaccine was safe and well tolerated in healthy adults, with mostly mild to moderate, self-limiting adverse events and no serious adverse events reported. The vaccine induced robust immune responses, with dose-dependent neutralizing antibodies and RVF-specific IgG and T-cell responses detectable as early as day 14, and the highest dose ($5{\cdot}0 \times 10^{10}$ virus particles) showed the most durable antibody responses up to day 84. Overall, the findings support further evaluation of the highest dose in larger and more diverse populations in RVF outbreak-prone regions.[57]

Importantly, there is no evidence of any major antigenic difference that may impact virulence or animal vaccine or human candidate vaccine efficacy. For example, no mutations were found in the glycoprotein of lineage H strains detected in 2025, as compared to those detected prior to 2020 or in 2022.[58]

Clinical disease, severity and therapeutics

Animals

Outbreaks of RVF have profound socio-economic consequences for communities that depend on livestock, where animal health is tightly linked to household income, food security, and social stability. Livestock losses—often the earliest and most visible impact—include high mortality among young animals (especially lambs and kids) and widespread abortion events (abortion storms) leading to immediate reductions in herd size and long-term declines in productivity. [59] These losses translate into diminished income from the sale of animals and animal products such as milk, meat, and hides, while also eroding savings and financial resilience, as livestock frequently function as a primary asset in pastoral and smallholder systems. Secondary economic effects can be equally severe: trade restrictions, market closures, and movement bans imposed during outbreaks disrupt value chains and reduce market access, further depressing prices and household earnings. At the same time, households may face increased expenditures related to animal health interventions or replacement of lost stock, compounding financial strain. Beyond direct economic impacts, RVF outbreaks can exacerbate food insecurity, reduce access to nutrition, and disrupt livelihoods, particularly in regions where alternative income sources are limited, thereby amplifying vulnerability and slowing post-outbreak recovery.[59]

Humans

Most human RVF infections are asymptomatic or present as a short, self-limiting febrile illness with symptoms such as fever, muscle pain, joint pain, and headache. About 0.5–2% of patients develop ocular disease (retinitis), with retinal lesions appearing 1–3 weeks after initial symptoms; these can lead to permanent vision loss. A small proportion develop meningoencephalitis 1–4 weeks after early symptoms, characterized by severe headache, memory loss, hallucinations, confusion, vertigo, convulsions, lethargy, and coma. Neurological complications may emerge months later; death is uncommon, but long-term neurological deficits are frequent.

Fewer than 1% of patients develop the haemorrhagic fever form, which begins 2–4 days after illness onset with severe liver impairment, followed by bleeding manifestations such as vomiting blood, blood in the stool, purpuric rash, nose or gum bleeding, heavy menstrual bleeding, or bleeding at injection sites. This form carries a high fatality rate of around 50%, with death typically occurring 3–6 days after symptoms begin. [60–63]

Overall, human case fatality ratios reported by WHO are typically below 1% when considering total number of infections, including asymptomatic or mild cases that are unlikely to be diagnosed.[64] Higher CFRs (5-20%) have been documented among severe hospitalized cases, reflecting selection of the severely ill patients.[65]

The 2025 Senegal outbreak initially high severity likely reflected early detection bias, delays in accessing care, and limited availability of supportive therapies. Reassuringly, no mutations were found in known virulence factors (e.g. NSs/NSm). A key mutation in the polymerase - a single nonconservative D11N mutation - unique to the 2025 outbreak strains and located in the endonuclease domain was observed, but no clear evidence that this mutation would impact viral replication or virulence was found. Further studies are ongoing to further assess the role of this mutation on the virus phenotype.[58]

There is no specific antiviral treatment for RVFV neither for animals nor for humans. Management is supportive, including haemodynamic support, management of hepatic and renal dysfunction, and treatment of neurological complications.[66]

Diagnostic considerations

Animals

RVFV diagnostics in animals play a central role in detecting outbreaks early, because livestock typically exhibit infection and clinical signs before humans do.

Laboratory confirmation relies on a combination of RT-PCR, virus isolation, and serological assays such as IgM and IgG ELISAs. PCR and virus isolation offer high specificity but are limited by the short viremic period in animals, often only a few days, which means many infected animals test negative by the time samples are collected. Serological tests are more practical for field surveillance, yet their validation across different livestock species is uneven, and cross-reactivity with other viruses can reduce accuracy.[67] Although diagnostic tools exist, few have undergone rigorous inter-laboratory validation, and commercial veterinary kits vary widely in performance.[68] These scientific limitations are compounded by practical constraints: many RVF-endemic regions lack sufficient veterinary laboratory capacity, cold-chain infrastructure, and trained personnel, making timely sample collection and testing difficult. As a result, passive surveillance—particularly the reporting of unusual abortion events—remains the most feasible early detection method in many settings. Strengthening and validating animal diagnostics, alongside improving veterinary service capacity, is essential for effective RVF early warning and outbreak control.

Humans

Diagnostics of acute infection involves the detection of RVFV RNA by RT-PCR in blood or tissues. Most assays target conserved regions of the genome (e.g. L or S segments), [69,70] and mutation-driven diagnostic escape has never been reported for RVFV to date.

The evidence from a recent systematic review indicates that rapid and near-patient diagnostics for RVF show consistently high sensitivity and specificity under laboratory conditions but remain under-validated in real-world field contexts. Strengthening context-specific validation will ensure reliable and practical deployment of these diagnostics for early outbreak detection and response. In addition, cost-effectiveness analyses are urgently needed to evaluate the economic feasibility of implementing rapid and near-patient diagnostics in low-resource and endemic settings.[71]

Because of the short duration of viral RNA (and antigen) detection (on average 5-10 days) in blood, anti-RVFV IgM antibodies serve as a key indicator of recent infections. However, there are limited validated, commercially available RVFV serological tests, particularly for IgM detection.[72]

Preparedness activities and recommended studies

The priorities below reflect areas where strengthened evidence, improved coordination, and targeted monitoring would most directly support RVFV early warning, risk assessment, and virus evolution monitoring. They draw on existing WHO/FAO/WOAH guidance, recent R&D roadmaps (e.g. Collaborative Open Research Consortium on *Phenuiviridae*), and lessons from countries where components of these activities have already been implemented. In settings with established surveillance or modelling systems, effectiveness has varied—often limited by inconsistent data flows between sectors, gaps in animal-level diagnostics, and insufficient integration of climate and vector information. The TAG-VE's perspective therefore emphasizes activities most relevant to monitoring virus evolution, improving early detection in animals, and understanding transmission dynamics across species.

1. Enhanced Integrated Surveillance

- Strengthen coordinated human–animal–vector surveillance systems in endemic and high-risk regions, ensuring synchronized data collection and reporting across sectors.
- Expand syndromic and laboratory-based surveillance for febrile illness in humans and abortion events in livestock, building on existing national systems where available.
- Generate evidence on the cost-effectiveness and operational feasibility of point-of-care diagnostics in field settings.
- Prioritize field validation of rapid and point-of-care diagnostic tools in animals, particularly for early detection of abortion clusters or acute infections, to reinforce early-warning capacity.
- Where integrated surveillance has been piloted, document performance, bottlenecks, and lessons learned to guide scale-up.

2. Ecology, Vectors, and Climate Modelling

- Refine predictive models linking rainfall, vegetation indices, flooding, and RVFV risk, building on existing early-warning frameworks used in East Africa and the Arabian Peninsula.
- Update vector distribution and competence data, including potential vectors in non-endemic regions such as the Mediterranean Basin and Asia, to inform preparedness for geographic expansion.

- Identify limitations of current models—such as coarse spatial resolution or limited entomological data—and prioritize improvements that enhance operational utility.

3. Human Disease Burden and Severity

- Conduct multi-country cohort and case–control studies to quantify the incidence of severe disease, neurological and ocular complications, and adverse pregnancy outcomes.
- In settings with prior outbreaks, review existing clinical data to identify gaps in understanding disease severity and long-term sequelae.

4. Vaccine Evaluation and Deployment Strategies

- Validate One Health vaccination strategies by assessing optimal combined use of livestock and human vaccines for prevention and control.
- Assess the cost-effectiveness of preventive livestock vaccination and explore mechanisms to support at-risk countries in implementing such vaccination programs, given their critical role in mitigating the burden of RVF, including economic and social losses as well as morbidity.
- Advance human vaccine candidates and develop clear use case scenarios (e.g., occupationally exposed groups, ring vaccination during outbreaks, high-risk geographic areas).
- Align research and deployment planning with existing global R&D roadmaps to ensure consistency and avoid duplication.

5. Risk to Non-Endemic Regions

- Assess the likelihood of RVFV introduction through animal movement and trade, vector spread, or traveler importation into other WHO regions—including Europe and Asia—integrating climate projections and vector distribution data.
- Review past incursions or near-miss events to identify vulnerabilities in surveillance or border controls.

6. Occupational Risks and Blood Safety

- Update biosafety and exposure-prevention guidance for slaughterhouse workers, veterinarians, and laboratory personnel, including PPE use, safe handling procedures, and surveillance protocols.

Overall global risk evaluation

There is currently no evidence that RVFV evolution is causing major increased virulence, immune escape, vaccine failure, or diagnostic escape. The main challenges remain preparedness, surveillance, preventive vaccination, and early warning rather than viral evolution itself. The overall risk posed by RVFV varies by region and depends on vector presence, livestock density and immunity, climate conditions, and preparedness capacity.

High to moderate risk in **endemic areas of sub-Saharan Africa** during rainy seasons where competent vectors, susceptible livestock and ecological conditions favour repeated epizootics and human outbreaks.

Moderate risk in **neighboring regions** with competent vectors and intense livestock trade, including livestock importation from sub-Saharan Africa, or climate conditions conducive to RVFV, especially parts of North Africa, the Arabian Peninsula (at risk of reintroduction) and potentially the Mediterranean Basin, depending on future climate and vector shifts.

Low to currently very low risk in regions without known competent vectors or **with limited ecological suitability**, though climate change and vector range expansion may alter this assessment over time.

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References

1. Nanyingi MO, Munyua P, Kiama SG, et al. A systematic review of Rift Valley Fever epidemiology 1931–2014. *Infection Ecology & Epidemiology*. 2015;5(1):28024. doi:10.3402/iee.v5.28024

2. Linthicum KJ, Britch SC, Anyamba A. Rift Valley Fever: An Emerging Mosquito-Borne Disease. *Annu Rev Entomol*. 2016;61(1):395-415. doi:10.1146/annurev-ento-010715-023819

3. Rahman MdM, Islam MdR, Dhar PS. Recent re-emergence of Rift Valley fever: epidemiology, clinical characteristics, transmission, symptoms, diagnosis, prevention, and treatment. *International Journal of Surgery*. 2023;109(2):117-119. doi:10.1097/JS9.0000000000000043

4. Fawzy M, Helmy YA. The One Health Approach is Necessary for the Control of Rift Valley Fever Infections in Egypt: A Comprehensive Review. *Viruses*. 2019;11(2):139. doi:10.3390/v11020139

5. Nsengimana I, Juma J, Roesel K, et al. Genomic Epidemiology of Rift Valley Fever Virus Involved in the 2018 and 2022 Outbreaks in Livestock in Rwanda. *Viruses*. 2024;16(7):1148. doi:10.3390/v16071148

6. Cosseddu GM, Magwedere K, Molini U, et al. Genetic Diversity of Rift Valley Fever Strains Circulating in Namibia in 2010 and 2011. *Viruses*. 2020;12(12):1453. doi:10.3390/v12121453

7. Anyamba A, Damoah R, Kemp A, et al. Climate Conditions During a Rift Valley Fever Post-epizootic Period in Free State, South Africa, 2014-2019. *Front Vet Sci*. 2021;8:730424. doi:10.3389/fvets.2021.730424

8. Tantely LM, Andriamandimby SF, Ambinintsoa MF, et al. An Entomological Investigation during a Recent Rift Valley Fever Epizootic/Epidemic Reveals New Aspects of the Vectorial Transmission of the Virus in Madagascar. *Pathogens*. 2024;13(3):258. doi:10.3390/pathogens13030258

9. Bastard J, Durand GA, Parenton F, et al. Reconstructing Mayotte 2018–19 Rift Valley Fever outbreak in humans by combining serological and surveillance data. *Commun Med*. 2022;2(1):163. doi:10.1038/s43856-022-00230-4

10. Nyakarahuka L, Balinandi S, Mulei S, et al. Ten outbreaks of rift valley fever in Uganda 2016-2018: epidemiological and laboratory findings. *International Journal of Infectious Diseases*. 2019;79:4. doi:10.1016/j.ijid.2018.11.029

11. Ramadan OPC, Berta KK, Wamala JF, et al. Analysis of the 2017-2018 Rift valley fever outbreak in Yirol East County, South Sudan: a one health perspective. *Pan Afr Med J*. 2022;42(Suppl 1):5. doi:10.11604/pamj.supp.2022.42.1.33769

12. Nsengimana I, Juma J, Roesel K, et al. Genomic Epidemiology of Rift Valley Fever Virus Involved in the 2018 and 2022 Outbreaks in Livestock in Rwanda. *Viruses*. 2024;16(7):1148. doi:10.3390/v16071148

13. Ishema L, Colombe S, Ndayisenga F, et al. One Health investigation and response to a nationwide outbreak of Rift Valley fever in Rwanda - March to December 2022. *One Health*. 2024;19:100854. doi:10.1016/j.onehlt.2024.100854

14. Shoemaker T, Boulianne C, Vincent MJ, et al. Genetic analysis of viruses associated with emergence of Rift Valley fever in Saudi Arabia and Yemen, 2000-01. *Emerg Infect Dis*. 2002;8(12):1415-1420. doi:10.3201/eid0812.020195

15. Tucker CJ, Melocik KA, Anyamba A, Linthicum KJ, Fagbo SF, Small JL. Reanalysis of the 2000 Rift Valley fever outbreak in Southwestern Arabia. Samy AM, ed. *PLoS ONE*. 2020;15(12):e0233279. doi:10.1371/journal.pone.0233279

16. Ndishimye P, Umuhoza T, Umutoni B, et al. Rift Valley Fever outbreaks in the East African Community: insights from ProMed data (2010–2024). *Front Public Health*. 2024;12:1298594. doi:10.3389/fpubh.2024.1298594

17. World Health Organization, Food and Agriculture Organization, World Organization for Animal Health. Joint FAO/WHO/WOAH Rapid Risk Assessment of Rift Valley fever (RVF) in Senegal and Mauritania: Implications for Public Health and Animal Health. doi:https://cdn.who.int/media/docs/default-source/2021-dha-docs/web---20251020_rapid-risk-assessment_rift-valley-fever_senegal---mauritania.pdf?sfvrsn=466f832f_4&download=true

18. Hartman A. Rift Valley Fever. *Clin Lab Med*. 2017;37(2):285-301. doi:10.1016/j.cll.2017.01.004

19. Rostal MK, Thompson PN, Anyamba A, et al. Rift Valley fever epidemiology: shifting the paradigm and rethinking research priorities. *The Lancet Planetary Health*. 2025;9(9):101299. doi:10.1016/j.lanplh.2025.101299

20. Lepore L, Odinoh R, Dawa J, et al. Understanding the Rift Valley fever exposure risk: A comparative perspective from a multi-country study in East and Central Africa, 2021-24. *PLoS Negl Trop Dis*. 2026;20(3):e0014082. doi:10.1371/journal.pntd.0014082

21. Al-Hamdan NA, Panackal AA, Al Bassam TH, et al. The Risk of Nosocomial Transmission of Rift Valley Fever. Bird B, ed. *PLoS Negl Trop Dis*. 2015;9(12):e0004314. doi:10.1371/journal.pntd.0004314

22. Eifan S, Schnettler E, Dietrich I, Kohl A, Blomström AL. Non-Structural Proteins of Arthropod-Borne Bunyaviruses: Roles and Functions. *Viruses*. 2013;5(10):2447-2468. doi:10.3390/v5102447

23. Nair N, Osterhaus ADME, Rimmelzwaan GF, Prajeeth CK. Rift Valley Fever Virus—Infection, Pathogenesis and Host Immune Responses. *Pathogens*. 2023;12(9):1174. doi:10.3390/pathogens12091174

24. Juma J, Tegally H, Konongoi SL, et al. Spatiotemporal patterns of Rift Valley fever virus in Africa: a retrospective genomic epidemiology and phylodynamic modelling study. *The Lancet Microbe*. 2026;7(3):101251. doi:10.1016/j.lanmic.2025.101251

25. Bird BH, Githinji JWK, Macharia JM, et al. Multiple Virus Lineages Sharing Recent Common Ancestry Were Associated with a Large Rift Valley Fever Outbreak among Livestock in Kenya during 2006-2007. *J Virol*. 2008;82(22):11152-11166. doi:10.1128/JVI.01519-08

26. Grobbelaar AA, Weyer J, Leman PA, Kemp A, Paweska JT, Swanepoel R. Molecular Epidemiology of Rift Valley Fever Virus. *Emerg Infect Dis*. 2011;17(12):2270-2276. doi:10.3201/eid1712.111035

27. Sene O, Sagne SN, Bob NS, et al. Re-Emergence of Rift Valley Fever Virus Lineage H in Senegal in 2022: In Vitro Characterization and Impact on Its Global Emergence in West Africa. *Viruses*. 2024;16(7):1018. doi:10.3390/v16071018

28. Bakamutumaho B, Juma J, Clancey E, et al. Hyperendemicity of Rift Valley Fever in Southwestern Uganda Associated With the Rapidly Evolving Lineage C Viruses. *J Infect Dis*. 2026;233(1):e77-e88. doi:10.1093/infdis/jiaf417

29. Gaudreault NN, Indran SV, Balaraman V, Wilson WC, Richt JA. Molecular aspects of Rift Valley fever virus and the emergence of reassortants. *Virus Genes*. 2019;55(1):1-11. doi:10.1007/s11262-018-1611-y

30. Bird BH, Khristova ML, Rollin PE, Ksiazek TG, Nichol ST. Complete genome analysis of 33 ecologically and biologically diverse Rift Valley fever virus strains reveals widespread virus movement and low genetic diversity due to recent common ancestry. *J Virol*. 2007;81(6):2805-2816. doi:10.1128/JVI.02095-06

31. Sang R, Lutomiah J, Said M, et al. Effects of Irrigation and Rainfall on the Population Dynamics of Rift Valley Fever and Other Arbovirus Mosquito Vectors in the Epidemic-Prone Tana River County, Kenya. *J Med Entomol*. Published online December 22, 2016:tjw206. doi:10.1093/jme/tjw206

32. Anyamba A, Linthicum KJ, Small JL, et al. Climate Teleconnections and Recent Patterns of Human and Animal Disease Outbreaks. Zhou XN, ed. *PLoS Negl Trop Dis*. 2012;6(1):e1465. doi:10.1371/journal.pntd.0001465

33. Anyamba A, Damoah R, Kemp A, et al. Climate Conditions During a Rift Valley Fever Post-epizootic Period in Free State, South Africa, 2014-2019. *Front Vet Sci*. 2021;8:730424. doi:10.3389/fvets.2021.730424

34. Wichgers Schreur PJ, Vloet RPM, Kant J, et al. Reproducing the Rift Valley fever virus mosquito-lamb-mosquito transmission cycle. *Sci Rep*. 2021;11(1):1477. doi:10.1038/s41598-020-79267-1

35. Danzetta ML, Bruno R, Sauro F, Savini L, Calistri P. Rift Valley fever transmission dynamics described by compartmental models. *Preventive Veterinary Medicine*. 2016;134:197-210. doi:10.1016/j.prevetmed.2016.09.007

36. Cecilia H, Métras R, Fall AG, Lo MM, Lancelot R, Ezanno P. It’s risky to wander in September: Modelling the epidemic potential of Rift Valley fever in a Sahelian setting. *Epidemics*. 2020;33:100409. doi:10.1016/j.epidem.2020.100409

37. Sankhe S, Talla C, Thiam MS, et al. Seroprevalence of Crimean-Congo Hemorrhagic Fever Virus and Rift Valley Fever Virus in human population in Senegal from October to November 2020. *IJID Regions*. 2023;7:216-221. doi:10.1016/j.ijregi.2023.03.016

38. Diarra M, Sankhe S, Barry MA, et al. Prevalence and associated factors of human haemorrhagic fevers in Senegal: a comprehensive analysis of Hantaan, Crimean-Congo haemorrhagic fever and Rift Valley fever. *Front Public Health*. 2026;13:1745257. doi:10.3389/fpubh.2025.1745257

39. Food and Agriculture Organization. Africa El Niño and increased risk of Rift Valley fever – Warning to countries. *EMPRES WATCH*. 2015;Vol. 35. http://www.fao.org/3/a-i5282e.pdf

40. Anyamba A, Chretien JP, Small J, et al. Prediction of a Rift Valley fever outbreak. *Proc Natl Acad Sci U S A*. 2009;106(3):955-959. doi:10.1073/pnas.0806490106

41. Pittiglio C, Kivaria F, Morteo K, et al. Real-time disease risk monitoring and forecasting for early action. In: *2024 12th International Conference on Agro-Geoinformatics (Agro-Geoinformatics)*. IEEE; 2024:1-5. doi:10.1109/Agro-Geoinformatics262780.2024.10661081

42. *Rift Valley Fever Action Framework*. FAO; 2022. doi:10.4060/cb8653en

43. Arishi HM, Aqeel AY, Al Hazmi MM. Vertical transmission of fatal Rift Valley fever in a newborn. *Annals of Tropical Paediatrics*. 2006;26(3):251-253. doi:10.1179/146532806X120363

44. Adam I, Karsany MS. Case report: Rift Valley Fever with vertical transmission in a pregnant Sudanese woman. *Journal of Medical Virology*. 2008;80(5):929-929. doi:10.1002/jmv.21132

45. Baudin M, Jumaa AM, Jomma HJE, et al. Association of Rift Valley fever virus infection with miscarriage in Sudanese women: a cross-sectional study. *The Lancet Global Health*. 2016;4(11):e864-e871. doi:10.1016/S2214-109X(16)30176-0

46. McMillen CM, Hartman AL. Rift Valley Fever: a Threat to Pregnant Women Hiding in Plain Sight? Klein S, ed. *J Virol*. 2021;95(9):e01394-19. doi:10.1128/JVI.01394-19

47. Gibson S, Linthicum KJ, Turell MJ, Anyamba A. Rift Valley fever virus: Movement of infected humans threatens global public health and agriculture. *CABI Reviews*. Published online August 13, 2022:cabireviews202217029. doi:10.1079/cabireviews202217029

48. Gerken KN, LaBeaud AD, Mandi H, L'Azou Jackson M, Breugelmans JG, King CH. Paving the way for human vaccination against Rift Valley fever virus: A systematic literature review of RVFV epidemiology from 1999 to 2021. *PLoS Negl Trop Dis*. 2022;16(1):e0009852. doi:10.1371/journal.pntd.0009852

49. Ikegami T. Rift Valley fever vaccines: an overview of the safety and efficacy of the live-attenuated MP-12 vaccine candidate. *Expert Review of Vaccines*. 2017;16(6):601-611. doi:10.1080/14760584.2017.1321482

50. Matsiela MS, Naicker L, Khoza T, Mokoena N. Safety and immunogenicity of inactivated Rift Valley Fever Smithburn viral vaccine in sheep. *Virol J*. 2023;20(1):221. doi:10.1186/s12985-023-02180-2

51. Njenga MK, Njagi L, Thumbi SM, et al. Randomized Controlled Field Trial to Assess the Immunogenicity and Safety of Rift Valley Fever Clone 13 Vaccine in Livestock. Marques ETA, ed. *PLoS Negl Trop Dis*. 2015;9(3):e0003550. doi:10.1371/journal.pntd.0003550

52. World Health Organization for Animal Health. Rift Valley fever. https://www.woah.org/en/disease/rift-valley-fever/?utm_source=chatgpt.com

53. Pittiglio C, Kivaria F, Morteo K, et al. Real-time disease risk monitoring and forecasting for early action. In: *2024 12th International Conference on Agro-Geoinformatics (Agro-Geoinformatics)*. IEEE; 2024:1-5. doi:10.1109/Agro-Geoinformatics262780.2024.10661081

54. Kark JD, Aynor Y, Peters CJ. A Rift Valley fever vaccine trial: 2. Serological response to booster doses with a comparison of intradermal versus subcutaneous injection. *Vaccine*. 1985;3(2):117-122. doi:10.1016/0264-410X(85)90060-X

55. Pittman PR, Liu CT, Cannon TL, et al. Immunogenicity of an inactivated Rift Valley fever vaccine in humans: a 12-year experience. *Vaccine*. 1999;18(1-2):181-189. doi:10.1016/S0264-410X(99)00218-2

56. Alkan C, Jurado-Cobena E, Ikegami T. Advancements in Rift Valley fever vaccines: a historical overview and prospects for next generation candidates. *npj Vaccines*. 2023;8(1):171. doi:10.1038/s41541-023-00769-w

57. Anywaine Z, Serwanga J, Ggayi ABM, et al. Safety, tolerability, and immunogenicity of the ChAdOx1 RVF vaccine against Rift Valley fever among healthy adults in Uganda: a single-centre, single-blind, randomised, placebo-controlled, dose-escalation, phase 1 trial. *The Lancet Infectious Diseases*. 2026;26(3):302-313. doi:10.1016/S1473-3099(25)00565-1

58. Diagne MM, Fall G, Sall A, et al. Molecular Characterization of Rift Valley Fever Virus From the 2025 Outbreak in Northern Senegal Reveals Lineage H Persistence and Key Polymerase Mutations. *J Med Virol*. 2025;97(12):e70734. doi:10.1002/jmv.70734

59. Food and Agriculture Organization. Rift valley fever. https://www.fao.org/animal-health/animal-diseases/rift-valley-fever/en

60. Laughlin LW, Meegan JM, Strausbaugh LJ, Morens DM, Watten RH. Epidemic Rift Valley fever in Egypt: observations of the spectrum of human illness. *Transactions of the Royal Society of Tropical Medicine and Hygiene*. 1979;73(6):630-633. doi:10.1016/0035-9203(79)90006-3

61. Siam AL, Meegan JM, Gharbawi KF. Rift Valley fever ocular manifestations: observations during the 1977 epidemic in Egypt. *British Journal of Ophthalmology*. 1980;64(5):366-374. doi:10.1136/bjo.64.5.366

62. Madani TA, Al-Mazrou YY, Al-Jeffri MH, et al. Rift Valley fever epidemic in Saudi Arabia: epidemiological, clinical, and laboratory characteristics. *Clin Infect Dis*. 2003;37(8):1084-1092. doi:10.1086/378747

63. Al-Hazmi A, Al-Rajhi AA, Abboud EB, et al. Ocular complications of Rift Valley fever outbreak in Saudi Arabia. *Ophthalmology*. 2005;112(2):313-318. doi:10.1016/j.ophtha.2004.09.018

64. World Health Organisation. Rift Valley fever fact sheet. Published online December 20, 2024. https://www.who.int/news-room/fact-sheets/detail/rift-valley-fever

65. Woods CW, Karpati AM, Grein T, et al. An Outbreak of Rift Valley Fever in Northeastern Kenya, 1997-98. *Emerg Infect Dis*. 2002;8(2):138-144. doi:10.3201/eid0802.010023

66. Afshar Moghaddam N, Yekanipour Z, Akbarzadeh S, et al. Recent advances in treatment and detection of Rift Valley fever virus: a comprehensive overview. *Virus Genes*. 2025;61(4):400-411. doi:10.1007/s11262-025-02164-0

67. Hartman A. Rift Valley Fever. *Clin Lab Med*. 2017;37(2):285-301. doi:10.1016/j.cll.2017.01.004

68. Bird BH, Ksiazek TG, Nichol ST, MacLachlan NJ. Rift Valley fever virus. *javma*. 2009;234(7):883-893. doi:10.2460/javma.234.7.883

69. Drosten C, Göttig S, Schilling S, et al. Rapid detection and quantification of RNA of Ebola and Marburg viruses, Lassa virus, Crimean-Congo hemorrhagic fever virus, Rift Valley fever virus, dengue virus, and yellow fever virus by real-time reverse transcription-PCR. *J Clin Microbiol*. 2002;40(7):2323-2330. doi:10.1128/JCM.40.7.2323-2330.2002

70. Bird BH, Bawiec DA, Ksiazek TG, Shoemaker TR, Nichol ST. Highly Sensitive and Broadly Reactive Quantitative Reverse Transcription-PCR Assay for High-Throughput Detection of Rift Valley Fever Virus. *J Clin Microbiol*. 2007;45(11):3506-3513. doi:10.1128/JCM.00936-07

71. Tusiime M, Nyakarahuka L, Baluka SA, et al. Accuracy of rapid and near-patient diagnostics for detection of rift valley fever and Crimean-Congo haemorrhagic Fever infections: A systematic review and meta-analysis. *Diagn Microbiol Infect Dis*. 2026;115(3):117377. doi:10.1016/j.diagmicrobio.2026.117377

72. FIND. DxConnect Test Directory. https://finddx.shinyapps.io/testdirexplorer_beta/