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Review

At the Intersection of Vector Competence and Thermal Eco-Physiology: Molecular Determinants of Arboviral Adaptation and Transmission in Rift Valley Fever Vectors

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Abstract: Rift Valley Fever Virus (RVFV; family *Phenuiviridae*, genus *Phlebovirus*) presents a severe global biosecurity threat due to its high zoonotic burden, capacity for multi-species livestock epizootics, and potential for rapid transboundary dissemination driven by climate anomalies. Traditional epidemiological paradigms have categorized RVFV dynamics into distinct floodwater *Aedes* reservoir cycles and secondary *Culex* amplification cascades. However, emerging evidence highlights a far more complex, multi-vector landscape encompassing diverse *Culicidae* guilds (*Anopheles*, *Mansonia*, *Eretmapodites*, and *Coquillettidia*) across expanding non-endemic boundaries.

This review synthesizes recent advances at the convergence of molecular vector biology and thermal eco-physiology to evaluate how shifting environmental baselines alter host-pathogen interactions within the mosquito vector. We examine the molecular architecture of intra-vector anatomical barriers—specifically the Midgut Infection Barrier (MIB), Midgut Escape Barrier (MEB), Salivary Gland Infection Barrier (SGIB), and Salivary Gland Escape Barrier (SGEB)—and detail how host innate immune cascades, including exogenous small interfering RNA (*si*RNA) and PIWI-interacting RNA (*pi*RNA) pathways, restrict viral dissemination. Crucially, we evaluate how microclimatic temperature elevations modulate these molecular checkpoints. Thermal performance curves demonstrate that ambient warming accelerates viral replication kinetics and shortens the Extrinsic Incubation Period (*EIP*), while acute thermal stress compromises midgut basal lamina integrity and dampens antiviral immune signaling. By bridging molecular tissue tropism, vector innate immunity, and thermal performance kinetics with high-throughput field xenomonitoring, this synthesis provides an integrated framework for predicting, mapping, and mitigating climate-driven RVFV spillover into vulnerable Mediterranean, Middle Eastern, and European basins.

Keywords: Rift Valley Fever Virus, Vector Competence, Thermal Eco-Physiology, Extrinsic Incubation Period, RNA Interference, Tissue Tropism, Climate Change, One Health.

1. Introduction

1.1 Zoonotic Impact and Global Biosecurity Concerns

Rift Valley fever virus (RVFV) is an enveloped, single-stranded, negative-sense/ambisense tripartite RNA virus belonging to the family *Phenuiviridae*, order *Bunyavirales* [1]. RVFV causes severe epizootic outbreaks in domestic ruminants (sheep, goats, cattle, and camels) characterized by mass neonatal mortality and devastating miscarriages known as "abortion storms" [2, 3]. In humans, RVFV infection manifests primarily as an acute, self-limiting febrile illness; however, a subset of patients progresses to severe complications including retinitis, encephalitis, or fatal hemorrhagic fever [4, 5]. Due to its high potential for rapid transboundary movement, severe economic disruption of pastoral agricultural systems, and potential for aerosol transmission, the World Health Organization (WHO) classifies RVFV as a prioritized pathogen on the R&D Blueprint, while the World Organisation for Animal Health (WOAH) mandates strict global notification protocols [6, 7].

Table 1. Genome organization, structural components, and pathogenic roles of RVFV segments.

Genomic Segment	Polarity & Size (kb)	Encoded Polyprotein / Proteins	Functional & Pathogenic Role in Host/Vector Systems
Large (L)	Negative-sense (~6.4 kb)	L-protein (~240 kDa)	RNA-dependent RNA polymerase (RdRp); drives viral transcription & replication within mammalian/vector cells [8].
Medium (M)	Negative-sense (~3.8 kb)	Polyprotein (~110 kDa) precursor yielding: Gn & Gc NSm 78 kDa & 14 kDa	Cleaved into envelope glycoproteins Gn and Gc (mediating host/vector cell receptor binding & membrane fusion) [9]; NSm suppresses vector tissue barriers and mammalian apoptosis [10].
Small (S)	Ambisense (~1.7 kb)	Nucleoprotein (N) Non-structural protein S (NSs)	N encapsidates viral genomic RNA [11]; NSs acts as primary mammalian virulence factor, dampening IFN-α/ β signaling [12].

1.2 Paradigm Shift: Beyond the Classic Dual-Vector Model

For decades, RVFV epidemiology was framed around a rigid dual-vector paradigm:

Primary Floodwater Vectors: Floodwater *Aedes* species (e.g., *Ae. vexans*, *Ae. mcintoshi*, *Ae. ochraceus*) serve as primary reservoir vectors through transovarial (vertical) transmission [13]. Female *Aedes* deposit virus-infected eggs in low-lying ephemeral depressions (*dambos*), where dormant eggs survive desiccation for years [14].

Secondary Amplification Vectors: Following heavy rainfall, infected *Aedes* emerge and transmit RVFV to nearby livestock. Epizootic amplification ensues as secondary vectors—primarily *Culex* species (e.g., *Cx. pipiens*, *Cx. antennatus*, *Cx. poicilipes*, *Cx. tritaeniorhynchus*)—breed in stagnant surface waters and rapidly amplify transmission between ruminants and human populations [15, 16].

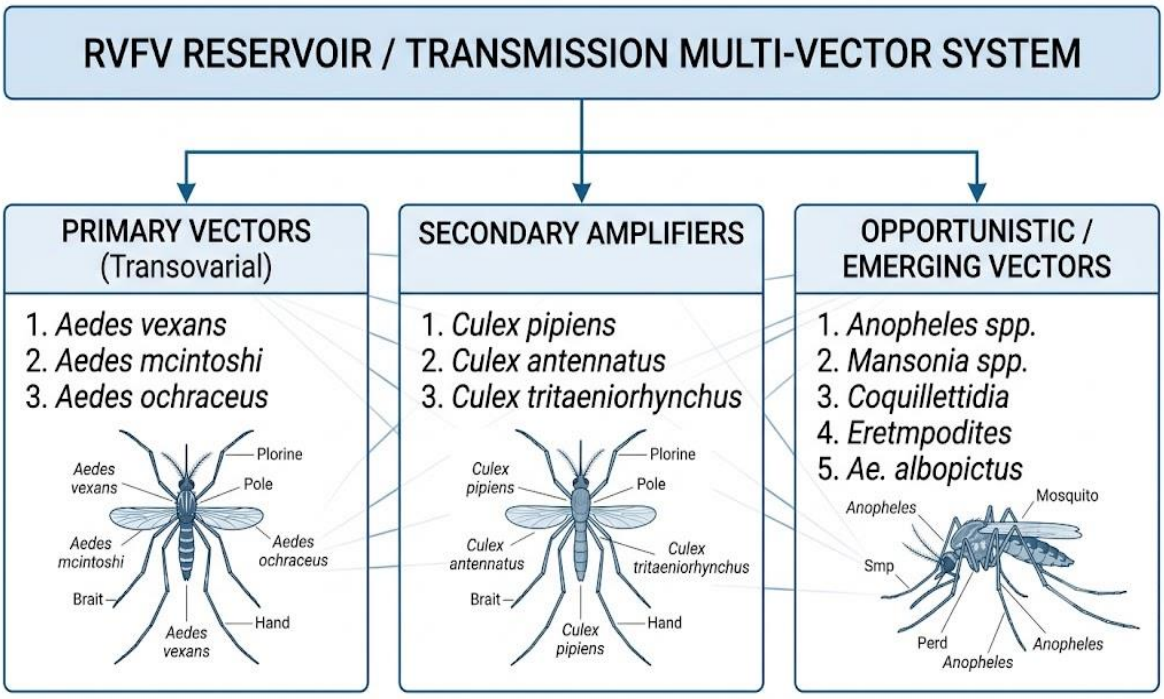

Figure 1. Hierarchical classification of mosquito vector roles in the Rift Valley Fever Virus (RVFV) transmission system. The multi-vector system is categorized into primary reservoirs capable of transovarial transmission (*Aedes* spp.), secondary amplifiers (*Culex* spp.), and opportunistic or emerging vectors (*Anopheles*, *Mansonia*, *Coquillettidia*, *Eretmapodites*, and *Aedes albopictus*).

Recent molecular field surveillance and laboratory vector competence evaluations demonstrate that this binary classification oversimplifies transmission dynamics [17]. A broader vector network actively contributes to inter-epizootic maintenance, local amplification, and transboundary spread:

Anopheline Vectors: Field isolates and vector competence assays confirm systemic infection and transmission potential in several *Anopheles* species (e.g., *An. coustani*, *An. squamosus*, *An. gambiae* s.l.), pointing to an underappreciated role in semi-arid and irrigated agricultural landscapes [18, 19].

Mansonia, Coquillettidia, and Eretmapodites Guilds: Broad surveillance across Sub-Saharan Africa and Indian Ocean islands demonstrates high viral loads in *Mansonia uniformis*, *Coquillettidia metallica*, and *Eretmapodites chrysogaster*, implicating them as vital bridge vectors between sylvatic reservoirs and domestic cycles [20, 21].

Invasive Aedines in Non-Endemic Zones: The continuous expansion of *Aedes albopictus* across Southern Europe and the Mediterranean basin presents major biosecurity concerns, as experimental studies confirm high vector competence for RVFV strains under variable thermal profiles [22, 23].

1.3 Environmental Drivers, Spatial Shifts, and Planetary Warming

The spatial and temporal distribution of RVFV vector networks is tightly linked to hydro-climatic anomalies [24]. Macro-scale climatic drivers—such as the El Niño Southern Oscillation (ENSO) and positive Indian Ocean Dipole (IOD) phases—trigger persistent heavy rainfall in arid and semi-arid regions [25, 26]. Satellite remote sensing metrics, including Normalized Difference Vegetation Index (NDVI) and Sea Surface Temperature (SST) anomalies, show strong predictive correlations with the mass emergence of floodwater *Aedes* vectors and subsequent secondary *Culex* blooms [27, 28].

Under global climate change scenarios, rising ambient temperatures non-linearly accelerate viral replication rates—shortening the Extrinsic Incubation Period (EIP)—while shifting the geographic and altitudinal envelopes of both primary and opportunistic vectors [29].

Global warming introduces two critical shifts into this ecological cascade:

Latitudinal and Altitudinal Range Expansion: Warming microclimates allow vector species (*Ae. albopictus*, *Cx. pipiens*, *Cx. tritaeniorhynchus*) to establish in previously unfavorable thermal zones, such as North Africa, Southern Europe, and high-altitude East African highlands [29, 30].

Thermal Performance Curve (TPC) Optimization: Elevated temperatures directly alter mosquito physiology, immune integrity, and viral replication kinetics [31]. Understanding the molecular mechanisms underlying these temperature-dependent processes is essential for evaluating vector competence in non-endemic regions under future climate scenarios.

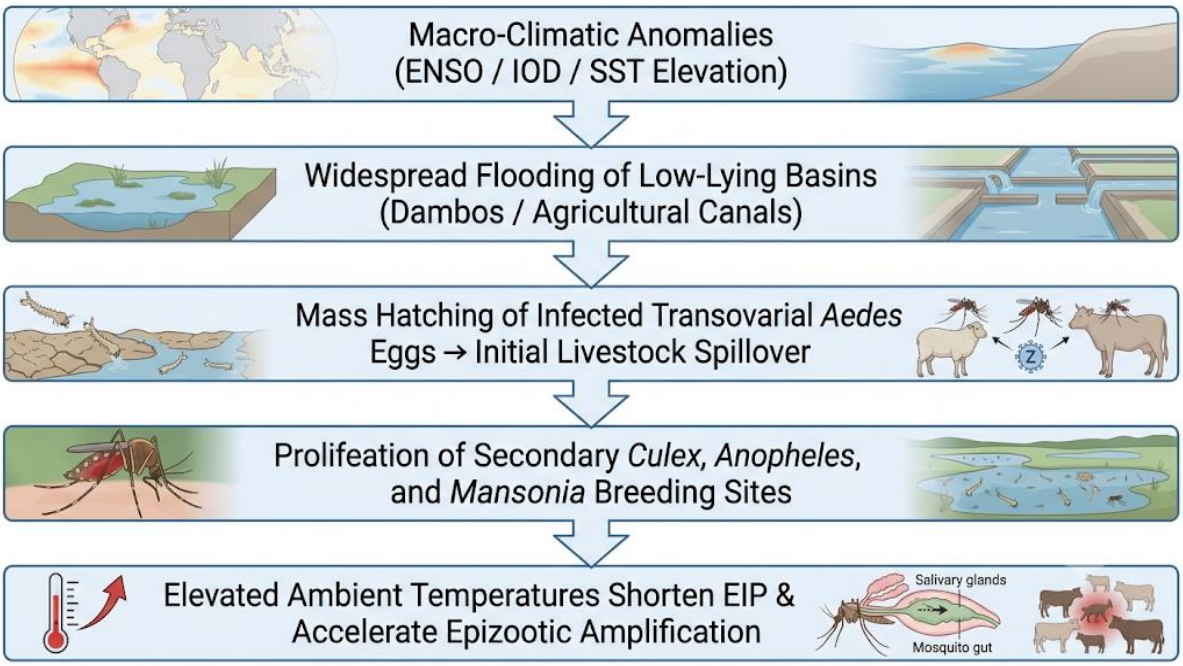

Figure 2. Environmental and biological cascade of Rift Valley Fever Virus (RVFV) epizootic amplification. Macro-climatic anomalies trigger widespread flooding, initiating a sequential transition from primary transovarial *Aedes* vector emergence to secondary vector proliferation (*Culex*, *Anopheles*, *Mansonia*). Elevated temperatures shorten the extrinsic incubation period (EIP), accelerating transmission dynamics across livestock.

1.4 Aims, Scope, and Novelty of this Review

Despite substantial progress in understanding RVFV genetics and climate correlations, existing literature remains fragmented—often treating viral molecular biology, vector tissue barriers, thermal eco-physiology, and control strategies as isolated domains. This review synthesizes empirical findings and field surveillance data to establish an integrated molecular-to-landscape framework for RVFV transmission. Specifically, it bridges the gap between viral non-structural protein function (e.g., NSm-mediated tissue barrier traversal), mosquito innate immunity cascades (RNAi, Toll, IMD, JAK-STAT), non-linear thermal performance curves ($R_0(T)$), and multi-species vector network dynamics. **The main objectives** of the present wok are to evaluate the expanded, multi-guild vector transmission network beyond the traditional *Aedes*–*Culex* binary model, to map the cellular mechanisms and viral factors governing midgut and salivary gland barrier traversal, to analyze how climate change, diurnal temperature ranges, and hydro-climatic anomalies reshape vector traits and transmission thresholds, and to assess the emerging vector control tools (*Wolbachia*, gene drives) and next-generation DIVA-compliant vaccines to inform transboundary biosecurity.

2. **Molecular Mechanics Of Intra-Vector Barriers & Tissue Tropism**

The establishment of systemic infection and subsequent transmission of Rift Valley Fever Virus (RVFV) by mosquito vectors is governed by a series of precise molecular and anatomical checkpoints within the insect host [32]. Once a female mosquito ingests an infectious blood meal from a viremic host, the virus must navigate complex anatomical tissue barriers, withstand host antiviral innate immune responses, and preferentially infect target tissues [33]. The efficiency with which RVFV traverses these intra-vector checkpoints defines overall vector competence and dictates the length of the Extrinsic Incubation Period (EIP) [34].

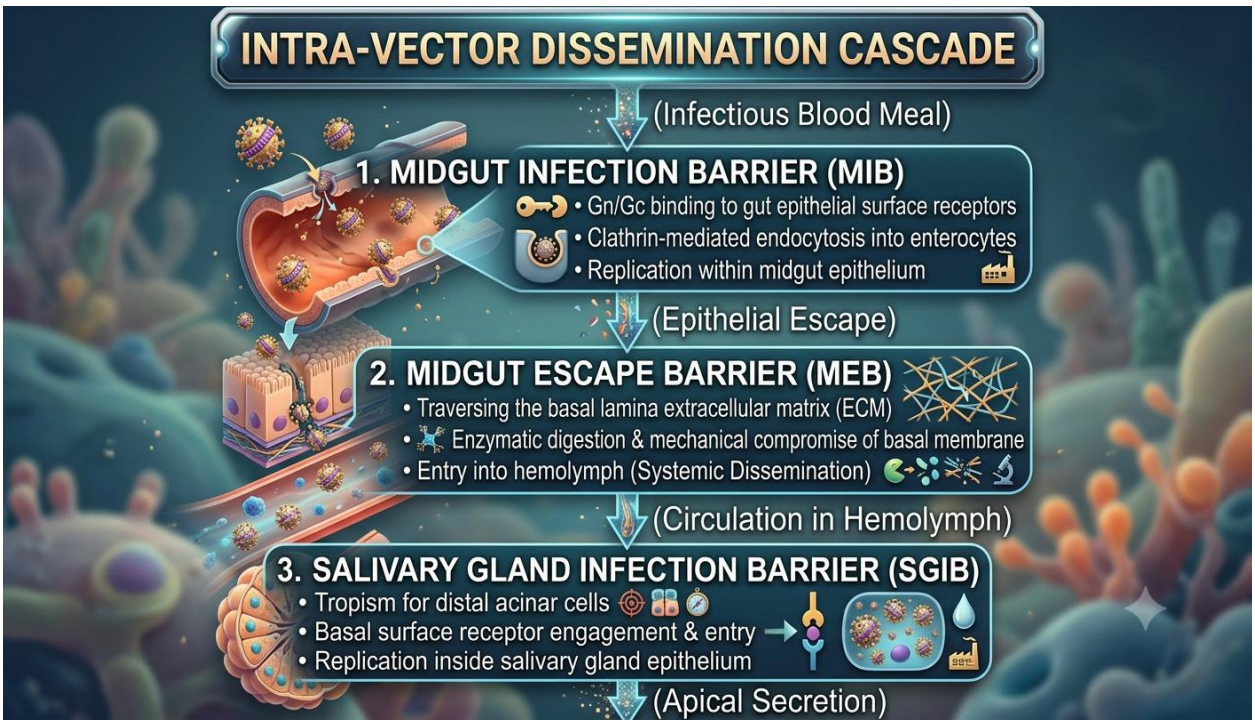

Figure 3. A detailed illustration of the Intra-Vector Dissemination Cascade. This infographic maps the multi-stage journey of a virus through its insect vector, highlighting the precise biological mechanisms and molecular interactions required to breach three critical physical and physiological checkpoints: (1) the Midgut Infection Barrier (MIB), (2) the Midgut Escape Barrier (MEB), and (3) the Salivary Gland Infection Barrier (SGIB).

2.1 Anatomical Dissemination Barriers Across Culicidae

Viral dissemination within the mosquito vector is strictly compartmentalized into four classic anatomical barriers: the Midgut Infection Barrier (MIB), Midgut Escape Barrier (MEB), Salivary Gland Infection Barrier (SGIB), and Salivary Gland Escape Barrier (SGEB) [35, 36].

2.1.1 Midgut Infection and Escape Barriers (MIB & MEB)

The midgut epithelium constitutes the primary cellular barrier to arboviral establishment [37]. Upon entering the posterior midgut lumen during blood feeding, RVFV virions must bind specific, as-yet-unidentified surface receptors on the microvillar membrane of midgut enterocytes [38]. Entry occurs predominantly via clathrin-mediated endocytosis, followed by low-pH-dependent fusion driven by structural rearrangements in the viral glycoprotein envelope [39].

Following internal replication within enterocytes, the virus faces the **Midgut Escape Barrier (MEB)** [40]. To enter the open circulatory system (hemolymph), RVFV virions must breach the midgut basal lamina—a dense, mesh-like extracellular matrix (ECM) composed of type IV collagen, laminin, nidogen, and perlecan [41]. In highly competent species like *Aedes vexans* and *Culex pipiens*, non-structural viral proteins (specifically NSm) and host matrix metalloproteinase (MMP) modulations facilitate viral passage through structural matrix pores or via tracheolar cells that innervate the midgut tissue [42, 43]. In refractory vector species, the virus remains sequestered within midgut epithelial cells or is degraded before crossing the basal membrane, halting systemic dissemination [44].

2.1.2 Salivary Gland Infection and Escape Barriers (SGIB & SGEB)

Virions that successfully cross the MEB enter the hemolymph, circulating throughout the hemocoel where they encounter host hemocytes [45]. To achieve biological transmission, RVFV must display high tissue tropism for the female salivary glands [46].

Salivary Gland Infection Barrier (SGIB): Circulating virions bind to receptors located on the basal plasma membrane of lateral and medial acinar cells [47]. Differences in acinar cell surface receptor density across *Culicidae* species account for marked variations in SGIB susceptibility [38].

Salivary Gland Escape Barrier (SGEB): Once replicated within acinar cells, virions must bud through the apical plasma membrane into the secretory canaliculi and apical salivary ducts [48]. The SGEB represents a critical final checkpoint; vectors with a permissive SGIB but non-permissive SGEB accumulate high intra-glandular viral titers yet fail to secrete infectious virus during feeding [49].

2.2 Mosquito Innate Immune Cascades vs. RVFV

The mosquito innate immune system acts as an active molecular filter restricting RVFV replication and systemic spread [50]. Mosquitoes rely entirely on potent germline-encoded innate defenses, comprising small RNA pathways, inducible signaling cascades, and cellular immune mechanisms [51].

2.2.1 Small RNA Interference Pathways (siRNA & piRNA)

RNA interference (RNAi) is the principal antiviral immune pathway in insects [52].

Exogenous small interfering RNA (exo-siRNA) Pathway: Triggered by double-stranded RNA (dsRNA) replicative intermediates generated during RVFV genome replication [53]. Dicer-2 (Dcr-2) recognizes and cleaves viral dsRNA into 21-nucleotide (nt) viral small interfering RNAs (vsiRNAs) [54]. These vsiRNAs are loaded into the RNA-Induced Silencing Complex (RISC), where Argonaute-

2 (Ago2) utilizes the single-stranded guide RNA to target and cleave complementary RVFV genomic and messenger RNAs [55].

PIWI-interacting RNA (piRNA) Pathway: Operating primarily via Dicer-independent processing, the piRNA pathway generates 25–30 nt viral piRNAs (vpiRNAs) bound to PIWI-clade proteins (Aubergine, Argonaute-3, and Piwi) [56]. In *Aedes* and *Culex* vectors, functional vpiRNAs are generated from viral negative and positive strands through a bi-directional "ping-pong" amplification loop, providing sustained transcriptional and post-transcriptional silencing of RVFV replication [57].

2.2.2 Inducible Signaling Pathways: Toll, IMD, and JAK-STAT

Complementing RNAi, three major canonical immune pathways regulate antiviral gene expression in response to RVFV infection [58]:

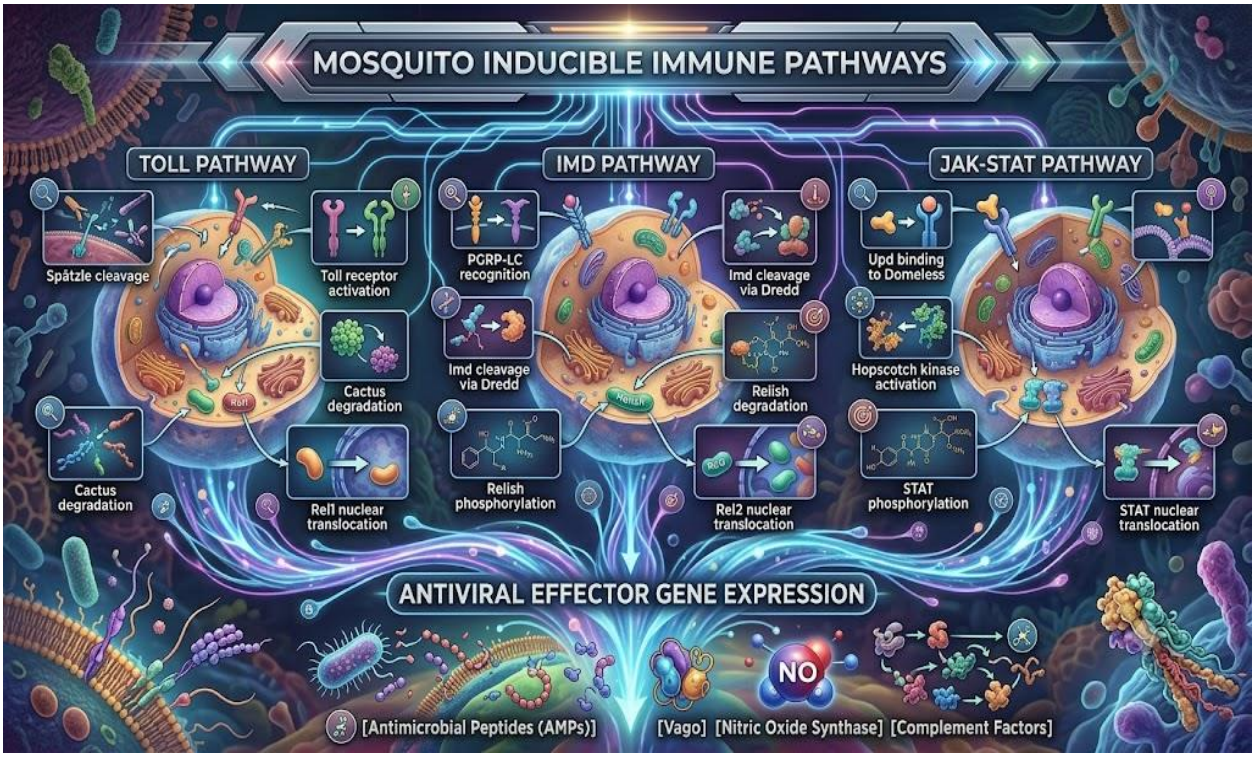

Figure 4. An intricate visual overview mapping the mosquito's multi-layered innate immune defense system. This illustration details the complex molecular signaling cascades of the Toll, IMD, and JAK-STAT pathways within a cellular context, tracking their progression from receptor activation to the nuclear translocation of transcription factors (Rel1, Rel2, STAT). The diagram culminates in the downstream activation of 'Antiviral Effector Gene Expression,' highlighting the ultimate production of key physiological responses including Antimicrobial Peptides (AMPs), Nitric Oxide Synthase, and other essential complement factors that combat pathogens.

Toll Pathway: Activated upon recognition of viral pathogen-associated molecular patterns (PAMPs) by extracellular pattern recognition receptors, triggering cleavage of the cytokine Spätzle [59]. Spätzle binding to the Toll receptor leads to degradation of the inhibitor Cactus, allowing nuclear translocation of the NF-κB-like transcription factor Rel1 and subsequent transcription of antiviral effector proteins [60].

Immune Deficiency (IMD) Pathway: Cleavage of the NF-κB factor Rel2 by the caspase Dredd leads to Rel2 nuclear translocation, driving the synthesis of antimicrobial peptides (AMPs) and reactive oxygen species (ROS) within gut enterocytes [61].

JAK-STAT Pathway: Secretion of the cytokine Vago following viral sensing leads to binding of the transmembrane receptor Domeless, activating the Janus kinase Hopscotch [62]. Activated Hopscotch phosphorylates STAT transcription factors, which dimerize and enter the nucleus to upregulate virucidal effector genes [63].

2.2.3 Cellular Immunity & Hemocyte Responses

Within the hemocoel, circulating hemocytes (granulocytes, oenocytoids, and plasmatocytes) engage in cellular defenses [64]. RVFV infection induces rapid hemocyte recruitment, triggering phagocytosis of viral particles and activating the prophenoloxidase (proPO) activating system [65]. ProPO activation leads to localized melanization—the deposition of melanin around infected tissues—and the production of toxic quinone intermediates that directly neutralize virions within the hemolymph [66].

2.3 Viral Molecular Intermediates and Vector Tropism

The tripartite genome of RVFV encodes key structural and non-structural proteins that interact directly with mosquito host factors to mediate cell entry, overcome tissue barriers, and suppress innate defenses [67].

Table 2. Molecular Determinants of RVFV Surface Attachment, Tissue Tropism, and Intra-Vector Dissemination.

Viral Component	Genomic Origin	Primary Structural & Functional Role	Intra-Vector Tropism & Barrier Interactions
Gn Glycoprotein	Medium (M) Segment	Outer envelope spike protein; mediates cell surface attachment and receptor binding	Interacts directly with microvillar surface receptors on midgut enterocytes and acinar cells of the salivary glands; primary determinant of tissue tropism [68].
Gc Glycoprotein	Medium (M) Segment	Class II viral fusion protein; hidden beneath Gn in static state	Undergoes low-pH conformational changes inside endosomes, exposing hydrophobic fusion loops to fuse viral and endosomal membranes [69].
NSm Protein (78 kDa & 14 kDa)	Medium (M) Segment	Non-structural polyprotein precursor product	**Critical vector determinant:** 78 kDa NSm dispensable in mammals but required for efficient midgut dissemination (MEB traversal) and viral infection of salivary glands [70].
NSs Protein	Small (S) Segment	Non-structural protein; primary virulence factor in mammals	Forms nuclear filamentous structures; suppresses mammalian IFNα/β; dampens mosquito hemocyte activity and RNAi pathway efficacy [71].
Nucleoprotein (N)	Small (S) Segment	Encapsid	

3. Thermal Eco-Physiology & Transmission Dynamics

3.1 Thermal Performance Curves (TPCs) for Mosquito Vector Traits

The biological processes governing mosquito life histories and viral interactions are inherently temperature-dependent. Trait responses across the physiological range typically follow asymmetric Thermal Performance Curves (TPCs), rising non-linearly to a optimum (T_{opt}) before declining rapidly as critical thermal limits (T_{max}) are approached [50, 51].

Key traits influencing transmission potential—including biting rate (a), extrinsic incubation period rate ($EIP_r = 1/EIP$), mosquito longevity (μ), vector competence (b, c), and fecundity/fecundity-derived population density (p)—display distinct thermal optima [52]. While larval development and adult biting rates generally peak at warmer temperatures (≈28-32 °C), adult survival drops precipitously above 28 °C, creating a complex physiological trade-off that determines net transmission efficiency [53, 54].

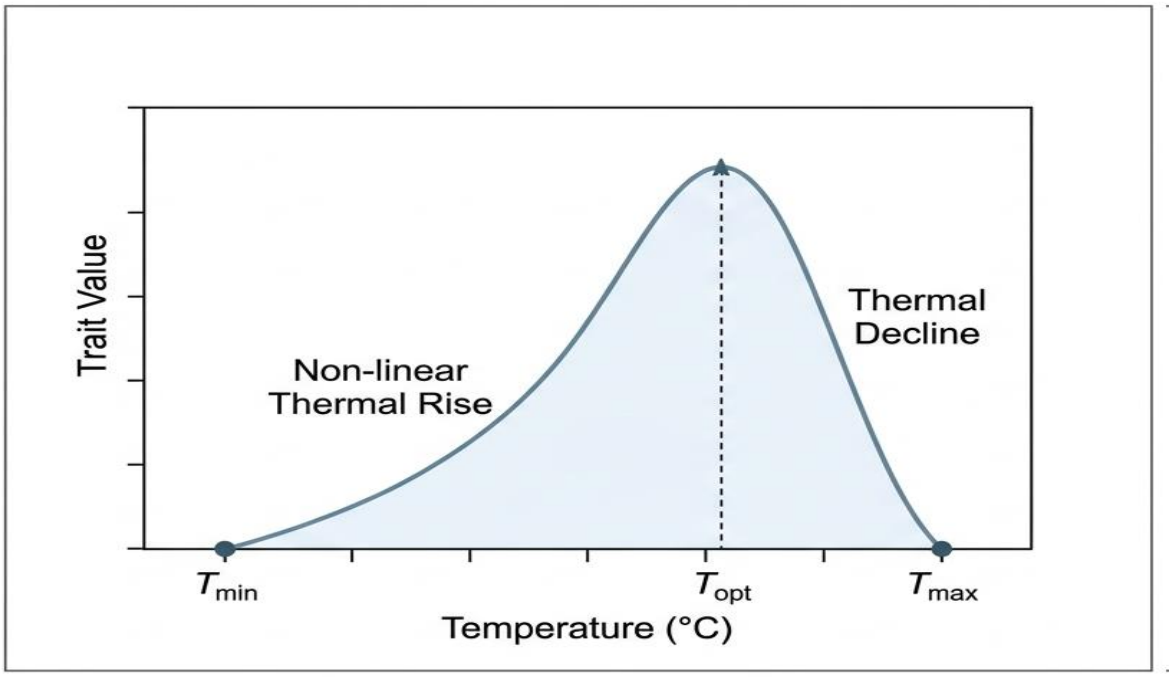

Figure 5. Generalized Asymmetric Thermal Performance Curve (TPC) for Vector Traits. This illustration depicts the characteristic, non-linear relationship between temperature and the performance of biological traits in disease vectors. Performance increases as temperature rises towards an optimum (T_{opt}), but then rapidly declines beyond T_{opt} until the maximum tolerance (T_{max}) is reached, demonstrating a pronounced thermal asymmetry. Note: T_{min} and T_{max} represent the operational thermal limits.

3.2 Extrinsic Incubation Period (EIP) Mechanics Under Temperature Fluctuations

The Extrinsic Incubation Period (EIP)—the time required for ingested virus to breach intra-vector barriers and reach the salivary glands—is inversely proportional to ambient temperature [55]. At lower temperatures (18- 20 °C), enzymatic processes, viral replication, and intracellular trafficking slow down, extending EIP for RVFV beyond 20- 30 days and often exceeding the average adult mosquito lifespan [56]. Conversely, under warmer regimes (28 -30 °C), enhanced viral replication kinetics and cell membrane fluidity significantly shorten the EIP to fewer than 5- 7 days [57].

However, constant-temperature laboratory studies often fail to capture field dynamics governed by Diurnal Temperature Range (DTR) fluctuations [58]. Moderate DTR fluctuations around cool mean temperatures can accelerate viral replication and shorten EIP compared to equivalent constant means, whereas high DTR fluctuations around high mean temperatures can induce thermal stress, damaging midgut tissue integrity and increasing mosquito mortality [59, 60].

3.3 Mathematical Formulation of Thermal R_0 Dynamics

The Basic Reproduction Number (R_0) for vector-borne transmission incorporates temperature-dependent trait parameters [61]. Integrating these thermal performance functions yields a non-linear expression for $R_0(T)$:

$$R_0(T) = \sqrt{\frac{a(T)^2 \cdot b(T) \cdot c(T) \cdot e^{-\mu(T) \cdot EIP(T)} \cdot m(T)}{\mu(T) \cdot r}}$$

Where $a(T)$ = Temperature-dependent biting rate, $b(T)$ = Vector competence (probability of infection given exposure), $c(T)$ = Transmission efficiency (probability of host infection per bite), $EIP(T)$ = Extrinsic Incubation Period duration ($1/EIP_r(T)$), $\mu(T)$ = Adult mosquito mortality rate, $m(T)$ = Vector-to-host ratio (dependent on temperature-driven larval development and survival) and r = Host recovery rate.

Because transmission requires adult mosquitoes to survive the entire duration of the *EIP*, the exponential term $e^{-\mu(T).EIP(T)}$ acts as the primary bottleneck [62]. The thermal optimum for $R_0(T)$ typically centers around 25 -27 °C, balancing accelerated viral replication against reduced adult longevity [63].

Table 3. Temperature sensitivity of key epidemiological traits governing RVFV vector dynamics.

Vector Trait / Parameter	Symbol	Thermal Sensitivity & Trend	Optimal Range (∘C)	Critical Thresholds
Biting Rate	$a(T)$	Increases monotonically up to thermal optimum; drops sharply near upper limits.	28-31 °C	Inhibited below 12 °C or above 36 °C [52, 54].
Extrinsic Incubation Rate	$EIP_r(T)$	Non-linear acceleration with warming; shortens EIP duration.	28-30 °C	Minimal replication below 16 °C [55, 57].
Adult Mortality Rate	$\mu(T)$	U-shaped/exponential increase at elevated thermal regimes.	18-23 °C	Severe mortality above 32 °C [53, 59].
Basic Reproduction Number	$R_0(T)$	Unimodal curve balancing biting, replication speed, and lifespan.	24-27 °C	Transmission ceases at T < 15 °C or T > 33 °C [61, 63].

3.4 Synergistic Microclimate Effects: Temperature and Humidity Interactions

Ambient temperature operates synergistically with relative humidity (RH) to determine adult vector survival and desiccation rates [64]. High temperatures coupled with low relative humidity accelerate desiccation, triggering behavioral shifts wherein mosquitoes increase feeding frequency to replenish lost body fluids, thereby elevating host contact rates [65]. Conversely, saturation deficit microclimates within dense vegetation or breeding habitats buffer vector populations against extreme ambient temperatures, allowing localized transmission to persist even during unfavorable seasonal macroclimates [66, 67]. Understanding these microclimatic micro-refugia is essential for accurate spatial modeling of epizootic risk [68, 69].

4. Global Climate Change & Spatial Vector Shifts

4.1 Poleward and Altitudinal Range Expansions of Culicidae Guilds

Anthropogenic climate change is restructuring the global distribution of mosquito vectors by altering fundamental thermal and hydrologic envelopes [70]. As mean annual temperatures rise and winter temperature extremes soften, species historically restricted to tropical and subtropical zones are undergoing poleward and altitudinal range expansions [71, 72].

Primary RVFV vectors within the genus *Aedes* (such as *Ae. vexans*) and *Culex* (such as *Cx. pipiens* and *Cx. theileri*) demonstrate high ecological plasticity, enabling rapid establishment in temperate zones of Europe, North America, and Central Asia [73, 74]. Higher elevations that previously functioned as thermal barriers against vector survival—such as the East African Highlands and Mediterranean mountain ranges—are increasingly experiencing microclimates capable of supporting seasonal vector persistence and viral amplification [75, 76].

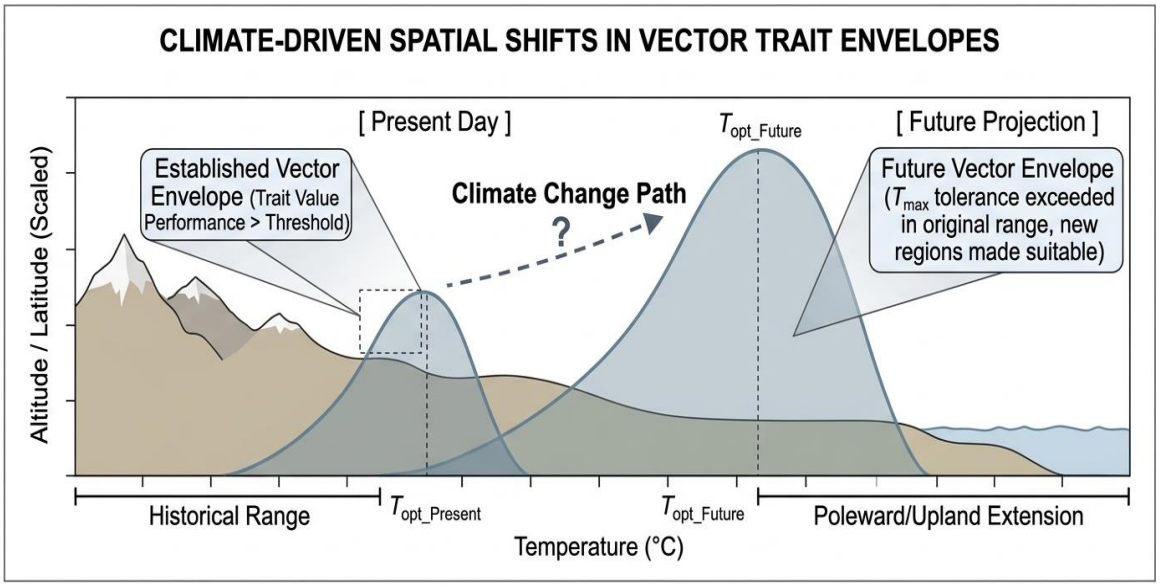

Figure 6. Climate-Driven Spatial Shifts in Vector Trait Envelopes. This diagram illustrates the expected shift and expansion of vector and pathogen transmission envelopes under a future climate scenario. On the right, the smaller, established envelope of trait performance ('Present Day') and lay') defines the current historical range. Under future, warmer conditions, a larger, poleward and upland extension creates an "Expanded Habitat & Transmission Zone'. Performance is lost in the historical range due to increased Tmax, while new areas become suitable. Note: Vertical and horizontal scales are integrated to represent cumulative climate suitable area for vector population and disease transmission.

4.2 Altered Hydro-Climatic Regimes and Flooding Dynamics

Rift Valley Fever epizootics are intimately linked to extreme hydrologic events [77]. The life cycle of primary reservoir vectors (*Aedes* species subgenera *Aedimorphus* and *Neomelaniconion*) depends on temporary floodwater pools (dambos) [78]. Desiccation-resistant *Aedes* eggs, infected transovarially with RVFV, remain dormant in dry soil for years until heavy rainfall triggers mass hatching [79].

Global climate projections indicate an increased frequency and intensity of extreme precipitation events punctuated by severe droughts [80]. Extended drought periods increase the accumulation of unhatched, infected eggs in soil, while subsequent intense rainfall events produce synchronous, high-density surges of primary vectors [18]. As surface waters persist, secondary bridge vectors (*Culex* species) proliferate in the surrounding vegetation, initiating explosive horizontal transmission cycles among domestic ruminants and humans [81, 82].

4.3 Integrated Species Distribution Modeling (SDM) Frameworks

Predicting future RVFV transmission risks relies on Ecological Niche Modeling (ENM) and Species Distribution Modeling (SDM) frameworks [83]. Modern ensemble models integrate high-resolution bioclimatic variables (e.g., WorldClim, CHELSA), remote sensing metrics (Normalized Difference Vegetation Index [NDVI], Land Surface Temperature [LST]), and land-cover dynamics [84, 85].

Table 4. Spatial Modeling Frameworks and Environmental Variables for RVFV Risk Mapping

Modeling Approach	Core Environmental Predictors	Methodological Strengths	Analytical Limitations
MaxEnt (Maximum Entropy)	Bio1 (Annual Mean Temp), Bio12 (Annual Precip), NDVI	Performs exceptionally well with presence-only vector surveillance data.	Sensitive to sampling bias; lacks physiological mechanism constraints [83, 86].
Random Forest / XGBoost	LST, Soil Moisture, Vegetation Indices, Livestock Density	Captures non-linear interactions across high-dimensional covariates.	Prone to overfitting without strict spatial cross-validation [84, 87].
Mechanistic $R_0(T)$ Maps	Thermal Performance Functions, Humidity, EIP Kinetics	Integrates physiological tipping points and biological mechanisms directly.	Requires parameterization data across non-model vector species [60, 88].

4.4 Sovereign Vulnerability and Biosecurity Implications

The geographic expansion of RVFV vectors poses severe challenges to international livestock biosecurity and global public health surveillance [89]. Non-endemic regions possessing competent vector guilds—such as Southern Europe and the Arabian Peninsula—face high vulnerability due to naïve host populations and the potential for rapid viral amplification upon introduction via infected livestock trade or migratory vector transport [90, 91].

Establishing proactive One Health surveillance networks—integrating real-time satellite climate monitoring, vector surveillance, and veterinary diagnostic capabilities—is critical to mitigating the threat of climate-driven RVFV emergence [92, 93].

5. Vector Control, Transmission-Blocking Strategies & One Health Interventions

5.1 Integrated Vector Management (IVM) and Ecological Source Reduction

Controlling the mosquito vectors of Rift Valley fever virus (RVFV) requires a dual-pronged, ecology-targeted strategy addressing both transovarial reservoir hosts (*Aedes* spp.) and secondary amplification vectors (*Culex* and *Mansonia* spp.) [94]. Integrated Vector Management (IVM) serves as a primary non-pharmaceutical defense against epizootic emergence [95].

5.1.1 Primary Vector Suppression (Aedes species)

Habitat Modification in Dambos: *Aedes mcintoshi* and *Aedes vexans* oviposit along the flood-prone margins of shallow natural depressions (dambos) across endemic regions [77, 94]. Physical modification or managed drainage of temporary water collections during dry periods significantly reduces transovarial eggbank accumulation [80].

Targeted Larviciding: Application of insect growth regulators (IGRs) like methoprene or biological larvicides (*Bacillus thuringiensis israelensis* [Bti] and *Lysinibacillus sphaericus*) to flooded dambo margins prior to primary adult emergence interrupts the initial viral amplification cycle [94].

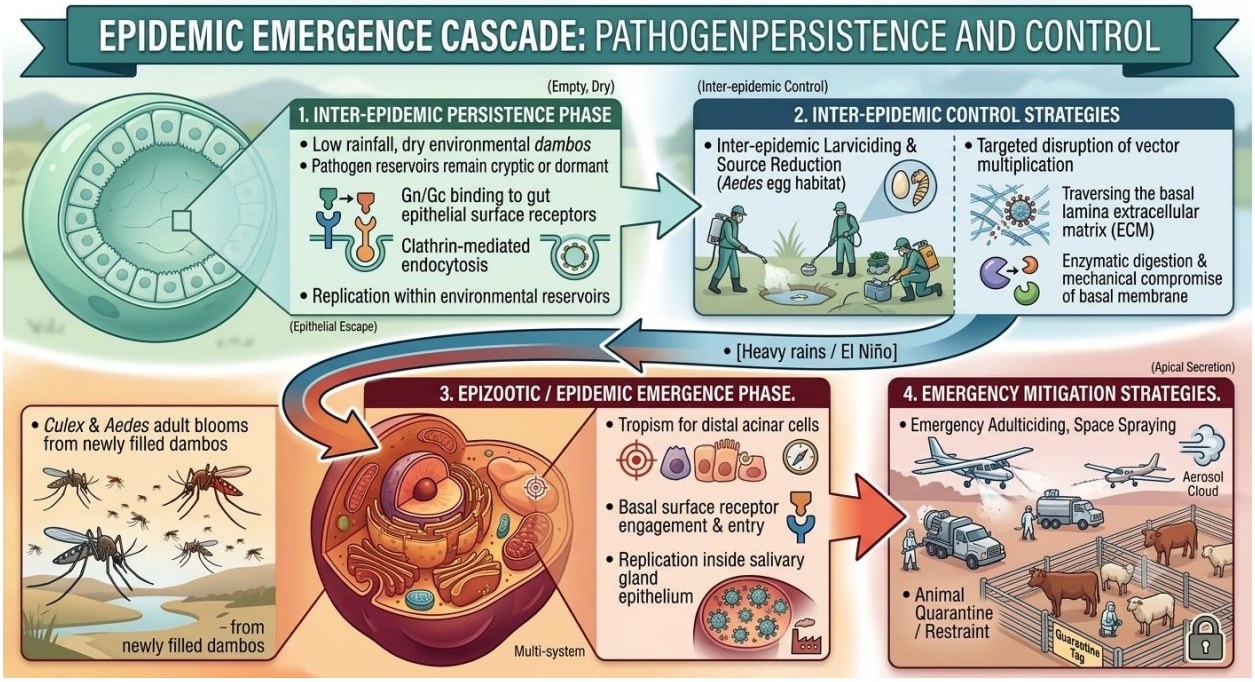

Figure 7. Integrated Strategies for Inter-Epidemic Control and Emergency Outbreak Mitigation.

5.1.2 Secondary Vector Interruption (Culex species)

Space Spraying & Chemical Control: Ultra-low volume (ULV) thermal fogging with pyrethroids (e.g., deltamethrin, permethrin) or organophosphates (e.g., malathion) is deployed during active epizootics to suppress high-density adult *Culex pipiens* and *Culex quinquefasciatus* populations [15, 83].

Insecticide Resistance Management: Widespread agricultural pesticide use has driven selection for resistant vector populations. Regular surveillance of knockdown resistance (*kdr*) mutations and acetylcholinesterase alterations is mandatory to preserve field chemical efficacy [95].

5.2 Biological Control Technologies and Genetic Approaches

To mitigate environmental toxicity and chemical resistance liabilities, novel biological vector control platforms are increasingly being evaluated within arboviral management frameworks [95, 96].

5.2.1 Symbiotic Wolbachia Endosymbionts

Cytoplasmic Incompatibility (CI): Transinfection of *Culex* or *Aedes* vectors with specific *Wolbachia* strains (e.g., *w*Mel, *w*Pip) induces CI, causing embryonic lethality when infected males mate with wild uninfected females [96].

Pathogen Blocking Mechanisms: *Wolbachia* infection triggers host innate immune priming (upregulating Toll and IMD cascades) and induces intracellular competition for host lipids, directly impeding RVFV replication within vector tissues [95].

5.2.2 Sterile Insect Technique (SIT) & Genetic Suppression

Sterile Insect Technique (SIT): Mass-rearing male mosquitoes exposed to ionizing radiation induces dominant lethal mutations in sperm. Sequential field releases of sterile males suppress target populations over successive generations [96].

Self-Limiting & Gene Drive Technologies: Conditional lethal genetic systems (e.g., RIDL) and CRISPR-Cas9 homing gene drives targeting female fertility genes (e.g., *doublesex*) offer high-specificity population suppression tools for primary vectors [96].

5.3 Veterinary & Human Vaccination Paradigms

Vaccination remains the cornerstone of preventing epizootic abortion storms in livestock and halting zoonotic spillover to human populations [97, 98].

Table 5. Comparative Performance of Clinical and Veterinary RVFV Vaccine Platforms

Vaccine Candidate / Platform	Strain Type	Target Host	Advantages	Limitations / Regulatory Status
Smithburn Strain	Neurotropic Live Attenuated	Ruminants	Low cost; rapid single-dose immunity.	Teratogenic in pregnant livestock; risk of reversion to virulence [94, 97].
MP-12	Chemical Mutagen-derived Live	Ruminants & Humans	Broad immunogenicity; Phase II human trial data.	Mild teratogenicity at high doses in first trimester [98, 99].
Clone 13	Naturally Attenuated (*δNSs*)	Ruminants	DIVA compliant; safe in pregnant livestock.	Requires high manufacturing doses; not widely licensed for humans [99, 100].

ChAdOx1 RVF	Viral Vector (Chimpanzee Adenovirus)	Humans & Ruminants	Non-replicating; high antibody titers; multi-species safety.	Requires cold-chain infrastructure; Phase I/II clinical trials ongoing [101, 102].
Ad5-vectored RVF	Recombinant Adenovirus	Ruminants & Primates	Sterilizing protection in single dose.	Pre-existing anti-Ad5 vector immunity in human populations [100].

5.3.1 DIVA Compatibility

Vaccine constructs carrying deletions in non-structural protein genes (δ NSs, such as Clone 13 and ChAdOx1 RVF) permit Differentiation of Infected from Vaccinated Animals (DIVA) [101, 102]. Companion diagnostic ELISAs detecting anti-NSs antibodies distinguish natural field exposure (anti-NSs positive) from vaccination (anti-NSs negative), preserving international livestock trade status during routine surveillance [99, 103].

5.4 One Health Framework and Early Warning Systems (EWS)

Rift Valley fever represents a classic One Health paradigm requiring synchronized action across veterinary, public health, environmental, and meteorological disciplines [92, 97].

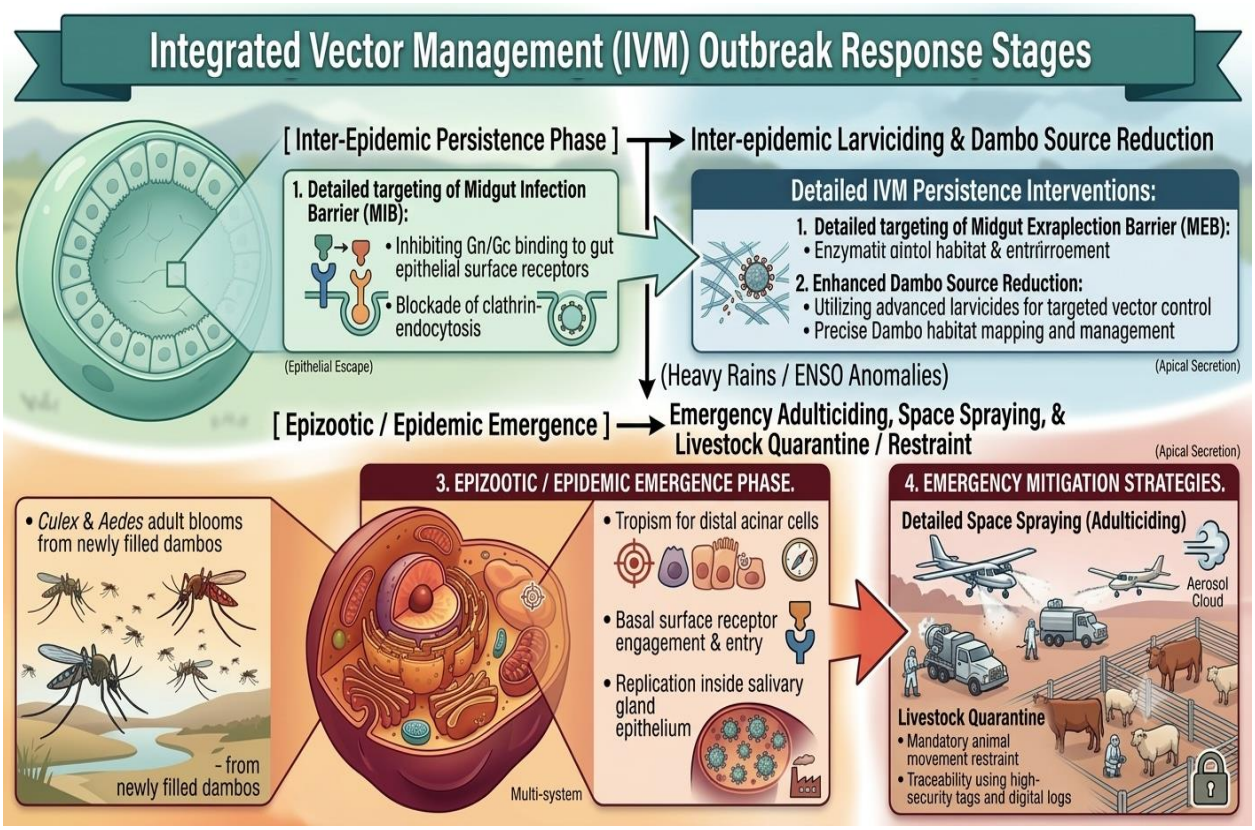

Figure 8. An infographic detailing an integrated vector and pathogen mitigation framework. Using satellite remote sensing (ENSO, SST, NDVI metrics), early warning alerts are generated to trigger proactive veterinary actions and public health abatement, preventing epidemic emergence.

5.4.1 Satellite Remote Sensing & Predictive Modeling

ENSO & Sea Surface Temperature (SST) Anomalies: Warm phases of the El Niño-Southern Oscillation (ENSO) coupled with Indian Ocean Dipole anomalies predict heavy, persistent rainfall in East Africa 2 to 5 months prior to vector emergence [24, 104].

Normalized Difference Vegetation Index (NDVI): Real-time monitoring of vegetation greenness and surface wetness via satellite sensors identifies active dambo flooding, triggering automated Early Warning System (EWS) alerts [28, 104].

5.4.2 Inter-Sectoral Epidemic Response Protocols

Targeted Pre-Epizootic Vaccination: Proactive livestock vaccination during the early warning window, avoiding vaccination *during* an active epizootic to prevent iatrogenic transmission via contaminated needles [99, 103].

Occupational Health Protections: Mandatory personal protective equipment (PPE)—including respirators, face shields, and heavy gloves—for abattoir personnel, veterinarians, and animal handlers during epizootics [99, 105].

Animal Movement Restrictions: Enforcing strict quarantines and banning livestock transit from high-risk zones to urban markets reduces viral dissemination along regional trade corridors [92, 97].

6. Critical Knowledge Gaps, Emerging Threats & Future Research Directions

6.1 Epizootic Dynamics, Ecological Reservoirs, and Spillover Triggers

Despite major advancements in predictive modeling, key ecological aspects of Rift Valley fever virus (RVFV) persistence during inter-epidemic periods (IEP) remain unresolved [104]. Addressing these gaps is critical for anticipating outbreak frequency and spatial expansion under changing climatic conditions [105].

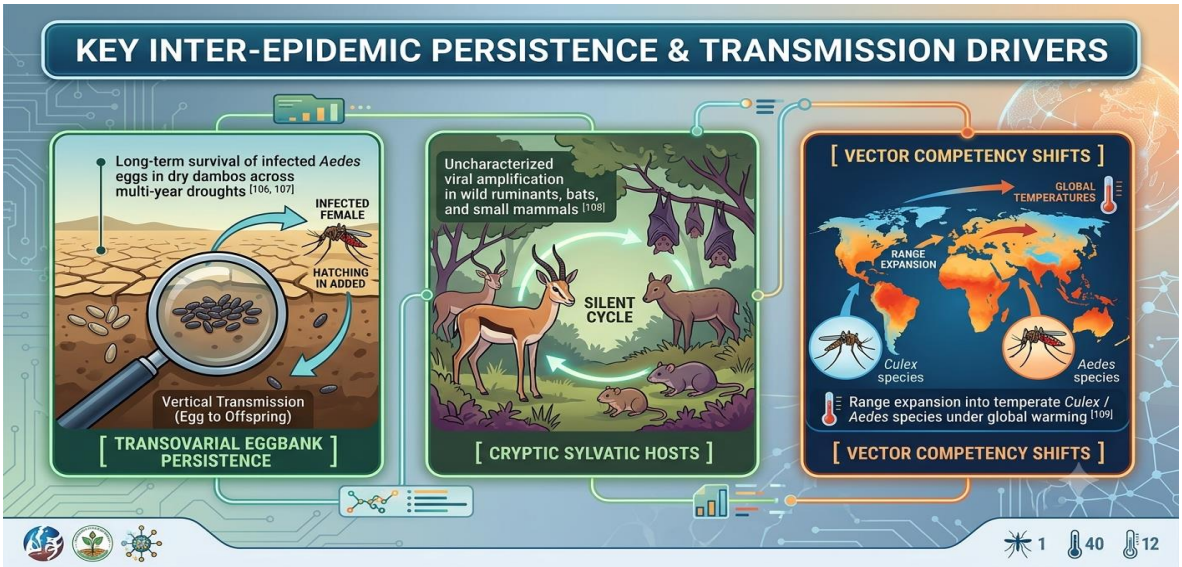

Figure 9: Key Inter-Epidemic Persistence & Transmission Drivers**.** This infographic details three primary mechanisms that allow the Rift Valley Fever virus to persist between outbreaks: **Transovarial Eggbank Persistence** in dry dambos, **Cryptic Sylvatic Hosts** maintaining a "silent cycle" among wild animals and **Vector Competency Shifts** leading to range expansion under global warming.

6.1.1 Transovarial Eggbank Survival Mechanisms

Desiccation Tolerance: The molecular mechanisms allowing RVFV-infected *Aedes mcintoshi* and *Aedes vexans* eggs to remain viable in dry soil for years during prolonged droughts remain poorly understood [104].

Transovarial Transmission (TOT) Rates: Precise natural rates of vertical transmission in field populations are difficult to quantify during IEPs due to low baseline viral loads, limiting baseline risk assessments [105].

6.1.2 Cryptic Hosts and Non-Vector Transmission

Wildlife Reservoirs: While domestic ruminants suffer high mortality and abortion storms, the role of indigenous wildlife (e.g., buffalo, wildlife ungulates, rodents, and bats) in maintaining low-level viral cycles without overt clinical signs requires broader surveillance [106].

Direct Transmission Dynamics: Transmission via mucosal exposure to infected fluids, aerosolization during slaughter, and unpasteurized milk consumption requires clearer quantitative risk profiling for occupational groups [97, 106].

6.2 Pathogenesis, Neuroinvasion, and Host Immunity Gaps

While structural viral proteins (Gn/Gc, N) and non-structural virulence factors (NSs, NSm) have been characterized, critical cellular interactions determining severe clinical outcomes—such as encephalitis and retinitis—remain active areas of investigation [108, 109].

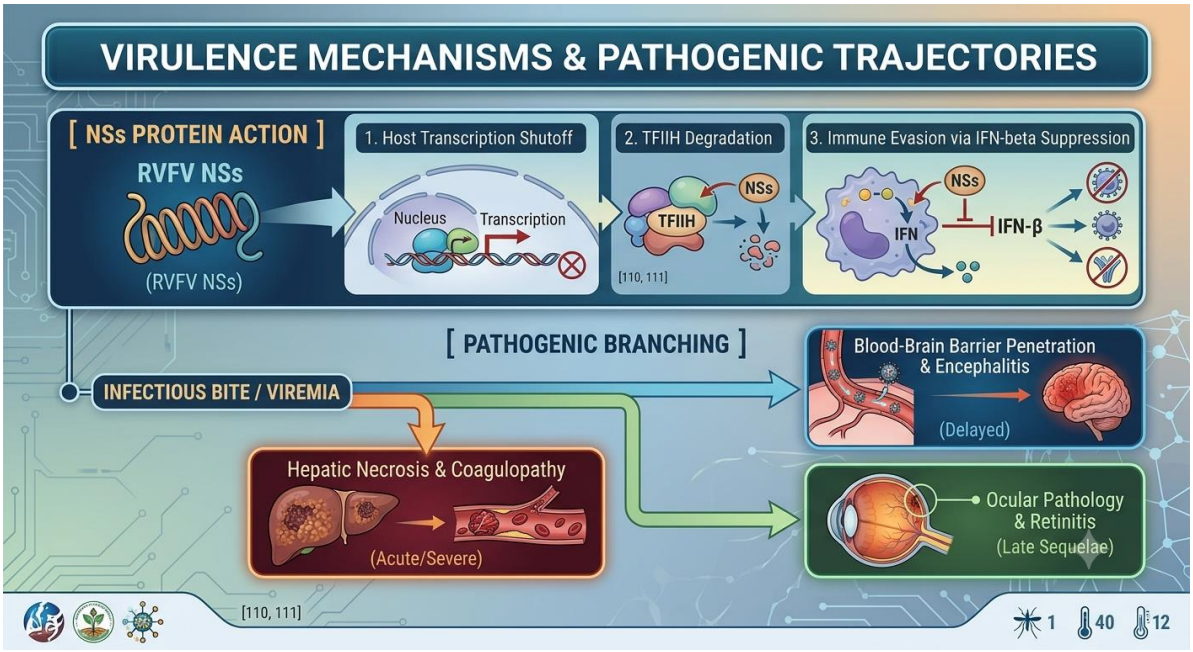

Figure 10: Virulence Mechanisms and Pathogenic Trajectories. This infographic details the cellular and systemic strategies employed by the Rift Valley Fever virus. The top panel illustrates how the NSs protein targets host cell functions by shutting off transcription, degrading TFIIH, and blocking IFN-beta production. The bottom panel traces the multiple pathogenic branching outcomes that can follow initial infection (viremia), ranging from severe, acute conditions like hepatic necrosis and coagulopathy to delayed-onset complications such as encephalitis and late-stage sequelae like retinitis.

6.2.1 Determinants of Neuroinvasion and Ocular Tropism

Blood-Brain Barrier (BBB) Crossing: The precise viral entry routes across the BBB—whether via infected monocytes (Trojan horse mechanism), direct endothelial cell infection, or olfactory nerve pathways—are not fully delineated [108].

Retinopathy Pathogenesis: Delayed ocular manifestations leading to permanent visual loss occur in a subset of human cases. The relative roles of direct viral cytopathology versus immune-complex-mediated inflammatory damage remain unclear [109].

6.2.2 Correlates of Protection

Cell-Mediated Immunity: While neutralizing antibodies against Gn/Gc are known to confer protection, the specific roles of CD^{4+} and CD^{8+} T-cell responses in clearing infection and preventing long-term sequelae require standardized immunological markers [108].

6.3 Translational Challenges: Antivirals, Diagnostics, and One Health Integration

Translating experimental interventions into deployable products faces logistical, economic, and regulatory hurdles, particularly in low- and middle-income countries (LMICs) [111, 112].

Table 6. Priority Gaps and Emerging Solutions Across Intervention Pillars.

Pillar	Key Knowledge / Operational Gap	Proposed Future Strategy
Diagnostics	Lack of affordable, low-cold-chain point-of-care (POC) tests capable of multiplexing RVFV with co-endemic febrile pathogens (e.g., Malaria, Dengue).	Deployment of lyophilized CRISPR-Cas isothermal amplification assays and microfluidic paper strips [111, 112].
Therapeutics	No approved human antivirals; treatment relies on supportive care during severe disease.	Broad-spectrum viral polymerase inhibitors (e.g., favipiravir analogs) and human monoclonal antibody cocktails [110, 113].
Vaccines	Balancing rapid single-dose immunity with safety in pregnant livestock and human safety profiles.	Phase II/III clinical evaluation of DIVA-compliant subunit and viral-vectored platforms (e.g., ChAdOx1 RVF) [99, 114].
One Health EWS	Siloed communication between meteorological, veterinary, and public health infrastructure.	Integrated real-time dashboards linking satellite remote sensing (NDVI/SST) directly to local response protocols [102, 115].

6.4 Global Expansion Risk and Climate Change Scenario Modeling

Rising global temperatures, shifting precipitation patterns, and international trade expansion increase the risk of RVFV introduction into non-endemic regions, such as Southern Europe, North America, and parts of Asia [107, 116].

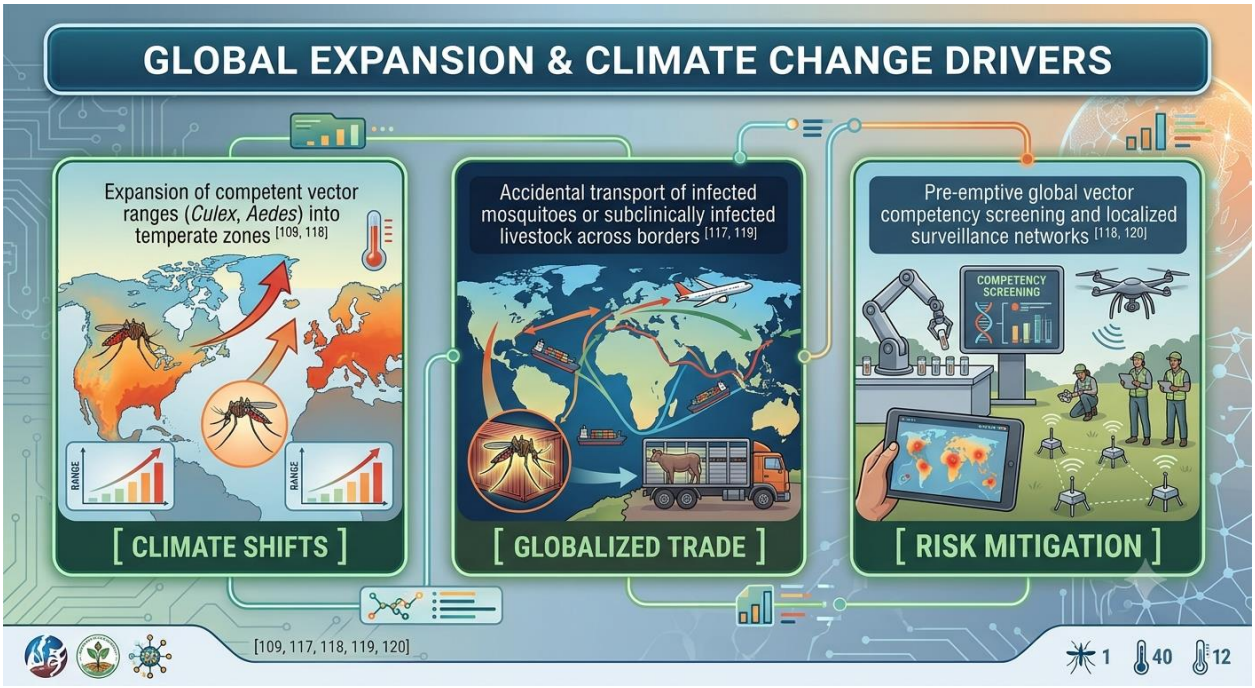

Figure 11: Global Expansion and Climate Change Drivers of Rift Valley Fever. This infographic illustrates the factors contributing to the geographical spread of Rift Valley Fever. It is divided into three sections: **[Climate Shifts]**, detailing the expansion of *Culex* and *Aedes* vector ranges into temperate zones; **[Globalized Trade]**, showing the accidental cross-border transport of infected mosquitoes and livestock; and **[Risk Mitigation]**, outlining preventive strategies such as global vector competency screening and localized surveillance networks.

6.4.1 Vector Competency in Temperate Species

Indigenous Vector Susceptibility: Studies demonstrate that North American and European mosquito species (e.g., *Aedes albopictus*, *Culex pipiens*) are competent vectors for RVFV in laboratory settings [107, 116]. However, field transmission dynamics under temperate microclimates require further study [116].

Preparedness and Global Security: Establishing standardized surveillance networks and stockpiling human and veterinary vaccines remain essential measures to mitigate potential global health threats [117, 118].

7. Conclusions

The epidemiology of Rift Valley fever virus (RVFV) extends beyond the traditional binary vector model, relying on a diverse, multi-guild vector ecosystem where intra-vector anatomical barriers (MIB, MEB, SGIB, and SGEB) and dual-layered innate immunity (RNAi and proPO melanization cascades) govern viral transmission and vector competence. Thermal eco-physiology demonstrates non-linear impacts on vector traits, where ambient warming accelerates replication kinetics and drastically shortens the extrinsic incubation period, though net transmission capacity $R_0(T)$ remains constrained by adult vector survival at elevated temperature extremes. Driven by climate change,

shifting rainfall patterns, and rising global temperatures, invasive vector species such as Aedes albopictus and Culex pipiens pose an expanding risk of introducing and establishing RVFV in previously non-endemic regions across Europe and the Mediterranean basin.

8. Recommendations and Future Perspectives

Future control strategies must adopt a comprehensive One Health framework that pairs pre-emergence vector suppression—such as habitat management, targeted larviciding, and advanced Wolbachia or gene-drive technologies—with satellite-driven early warning systems (tracking NDVI and SST anomalies) to trigger proactive interventions months prior to potential outbreaks. Epizootic preparedness relies on replacing older, teratogenic livestock vaccines with DIVA-compliant platforms (e.g., Clone 13, ChAdOx1 RVF) integrated with companion anti-NSs diagnostic assays, strict livestock movement controls, and enforced personal protective equipment for high-risk workers. Furthermore, future research priorities must focus on parameterizing thermal performance curves for invasive non-model vectors under diurnal temperature fluctuations, elucidating the molecular mechanisms of Aedes eggbank desiccation tolerance, and clarifying the viral pathways driving neuroinvasion and delayed ocular pathology.

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