

REVIEW

The risk of Rift Valley fever virus introduction and establishment in the United States and European Union

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Rift Valley fever virus (RVFV) is an arthropod-borne disease resulting in severe morbidity and mortality in both human and ruminant populations. First identified in Kenya in 1930, the geographical range of RVFV has been largely constrained to the African continent, yet has recently spread to new regions, and is identified as a priority disease with potential for geographic emergence. We present a systematic literature review assessing the potential for RVFV introduction and establishment in the United States (US) and European Union (EU). Viable pathways for the introduction of RVFV include: transport of virus-carrying vectors, importation of viremic hosts and intentional entry of RVFV as a biological weapon. It is generally assumed that the risk of RVFV introduction into the US or EU is low. We argue that the risk of sporadic introduction is likely high, though currently an insufficient proportion of such introductions coincide with optimal environmental conditions. Future global trends may increase the likelihood of risk factors for RVFV spread. *Emerging Microbes and Infections* (2013) 2, e81; doi:10.1038/emi.2013.81; published online 4 December 2013

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INTRODUCTION

Rift Valley fever virus (RVFV) is an arthropod-borne zoonotic disease responsible for widespread outbreaks in both humans and ruminants. Epizootics are characterized by mass abortions and high mortality in ruminants, resulting in high economic burden.^{1–5} High mortality rates have also been observed in humans and severe complications develop in a small proportion of people, including hemorrhagic fever, blindness and residual neurological deficits.⁶ First identified in Kenya in 1930,⁷ the geographical range of RVFV has been largely constrained to the African continent.^{2,8} However, over the past 50 years, RVFV has spread outside of its traditional endemic region and has been identified in over 30 countries, including parts of western Africa, Egypt, Madagascar and the Comoros.^{2,4,9,10} Recently, RVFV spread to the Arabian Peninsula in 2000, marking the first epidemic ever identified outside of the African continent (Figure 1).^{5,11,12}

Concerns over the potential for further spread and transmission of RVFV have been heightened by the significant spread and establishment of vector-borne diseases worldwide such as West Nile Virus (WNV), Crimean-Congo hemorrhagic fever and Japanese encephalitis. Crimean-Congo hemorrhagic fever has spread to over 30 countries in a range of ecological conditions.⁴ Similarly, Japanese encephalitis unexpectedly emerged in Australia, extending 3000 km from the previous known outbreak in Indonesia.⁴ Most notably, the widespread establishment of WNV demonstrated the vulnerability of western nations to the introduction of arboviruses.^{15–18} Similar to WNV, RVFV can be spread by a range of mosquito vector species as well as other arthropods, many of which are currently present in North America and Europe.^{19–22} RVFV is considered to have high colonization capacity and has been identified as a potential emergent risk in western nations, both as a natural exotic pathogen and an

intentionally introduced biological weapon. RVFV is, for example, classified as a category A priority pathogen by the National Institute of Allergy and Infectious Diseases—indicating the potential to cause social disruption and requiring public health preparedness²³—a high-consequence pathogen by the World Organization for Animal Health¹⁷ and the third most dangerous animal threat by the United States Department of Agriculture Animal and Plant Health Inspection Service after avian influenza and foot-and-mouth disease.²⁴

The mobility of RVFV and its ability to survive in a range of bioclimatic environments has raised concern among both the human and animal health communities regarding the probability of its introduction into western regions, including North America and Europe. Despite this, there has been no comprehensive and systematic review of the literature to evaluate the state of knowledge regarding the risk of RVFV introduction and establishment in these regions. We present a systematic scoping review of existing literature and knowledge of RVFV to assess the feasibility of emergence and establishment of the virus in the United States (US) and European Union (EU). The objectives herein include: (i) review and characterize the epidemiological characteristics of RVFV that affect transmission potential; (ii) identify and evaluate the feasibility of potential pathways for the introduction of RVFV; and (iii) assess the viability of the establishment of RVFV into the US and EU based on current knowledge.

METHODOLOGY

We review the epidemiological factors that affect the transmission and establishment potential of RVFV in the US and EU using a systematic approach to document selection.^{26–28} A systematic review approach uses pre-specified eligibility criteria to ensure that the procedure is transparent and repeatable and to minimize bias.^{25–27} US risk is used

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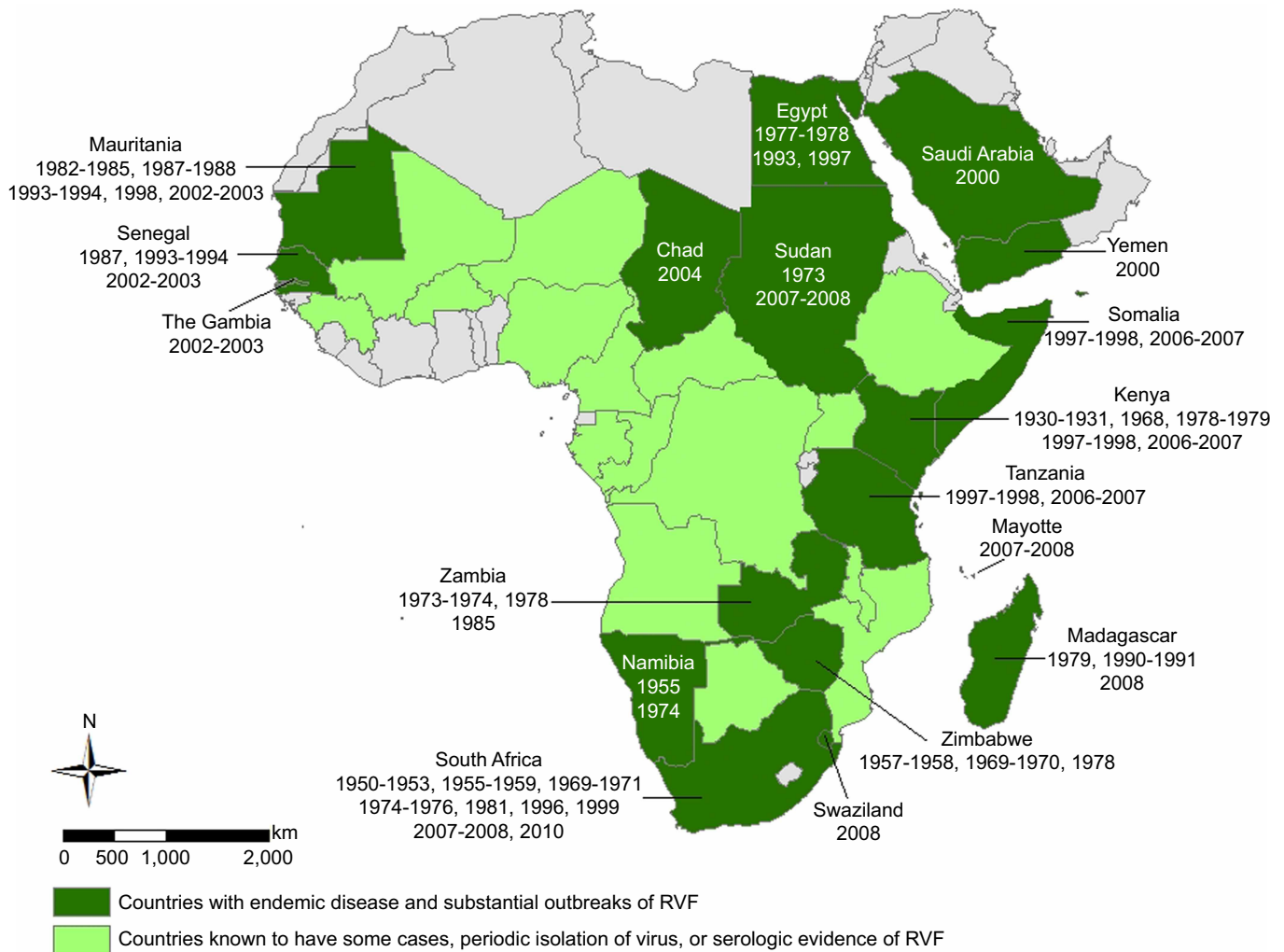


Figure 1 Geographical distribution of Rift Valley fever virus.^{2,4,13} Adapted from the Centers for Disease Control and Prevention.¹⁴

as a proxy for North America more broadly; relevant literature and data are negligible for risk in Canada and Mexico. Additionally, since RVFV has a low genetic diversity and reassortment and recombinations are thought to occur infrequently, our paper does not distinguish between different strains of RVFV.²⁸ A keyword search was performed in the search engine ISI Web of Knowledge using the topic search term 'Rift Valley fever'. The search yielded 610 documents. Titles and abstracts were reviewed and subjected to screening using the inclusion and exclusion criteria outlined in Table 1. Full article text was reviewed where necessary to confirm inclusion or exclusion. Forward and backward reference tracking approaches were applied to key documents meeting the inclusion criteria to identify additional relevant articles. Forty-six

documents met inclusion criteria and were retained for full review. The English abstracts of an additional 45 articles were also reviewed; however, the documents did not undergo full review as they were not available in English. Document review was guided by a systematic extraction of information relevant to each of the research objectives.

INTRODUCTION OF RVFV TO PREVIOUSLY UNAFFECTED REGIONS

Movement of viremic hosts

One of the most viable methods of dissemination of RVFV is the movement of viremic hosts, both intentionally (e.g., trade) and naturally (e.g., migration).^{1,16,29} Infected hosts, particularly ruminants

Table 1 Inclusion and exclusion criteria for document selection

Inclusion criteria	Exclusion criteria
<p>Phase 1: Key word search</p> <p>English only</p> <p>Published from 1 January 2000 to 15 August 2012</p> <p>Available in Web of Knowledge</p> <p>Peer-reviewed articles and reviews</p> <p>Phase 2: Title and abstract review</p> <p>Substantive discussion of environmental determinants of RVFV transmission, introduction or establishment</p>	<p>Non-English</p> <p>Pre-2000</p> <p>Not available in Web of Knowledge</p> <p>Documents other than article or review or not peer-reviewed</p> <p>No substantive discussion of the environmental determinants of RVFV transmission, introduction or establishment</p>

such as domestic livestock including camels, can function as the initial amplifying host, propagating the virus to domestic vectors, further spreading RVFV to other vertebrates (Figure 2).^{15,20,30} The recent incursion of RVFV into the Arabian Peninsula, for example, is largely attributed to the importation of viremic livestock from East Africa.^{31–33} Due to the high genetic similarity to the 1997–1998 outbreak in East Africa, research has posited that the virus was introduced to the Arabian Peninsula during the previous epidemic and had been circulating at low levels until favorable conditions facilitated its amplification and the emergence of sustained transmission cycles.^{2,6} Similar mechanisms have been suggested for the recent outbreaks in Madagascar^{9,10,34} and the Comoros.^{10,35}

The risk of importing RVFV into the US or EU via the importation of ruminants is generally assumed to be low.^{16,31,36} In the US, while there are no established federal regulations regarding the importation of ruminants indigenous to RVFV-endemic countries, importation is inadvertently prohibited as trade bans are established against countries endemic with foot-and-mouth disease, which currently coincide with all RVFV-endemic countries.¹⁶ Additionally, the illegal importation of ruminants is believed to occur infrequently due to the lack of demand and the size of the animals.¹⁶

In the EU, numerous trade restrictions have been established to prevent the importation of RVFV. All countries exporting livestock to the EU must be official RVFV-free countries, monitored by the National Veterinary Services, and trans-shipment through infected

regions is prohibited.^{31,36} Additionally, a system of veterinary checkpoints has been established to identify potential viremic animals.^{31,36} Trade should not occur in regions where appropriate surveillance is not established.^{1,33} However, while these measures should preclude trade of livestock from potentially endemic regions, cattle are traded between Egypt—a country with possible emerging endemicity—and the EU, representing a possible entry point.³⁶ The state of RVFV transmission in the Middle East is unknown, non-existent or currently undetected, with the possible exception of Saudi Arabia.^{37,38} Despite this, transmission is ecologically feasible, as illustrated by circulating WNV in Iran.³⁹ Introduction to the EU from this part of the world via legal or illegal trade may thus be possible. The probability of RVFV introduction into the EU via imported ruminants would be elevated during an epizootic.^{36,40}

The risk of incursion via the illegal importation of ruminants or other animals remains a viable pathway, though the magnitude of such risk remains unclear and has not been quantified.^{31,36} Additionally, introduction could occur via the importation of viremic wildlife species or zoo animals. Quarantine measures are established in both the US and EU, however, largely eliminating the risk of introducing RVFV into these continents.^{16,31,36}

Humans are generally considered to be dead-end hosts for RVFV, with negligible capacity to transmit the virus, amplify transmission or propagate dispersal.³¹ Some recent literature has, however, suggested that humans and international air travel may play a role in disease

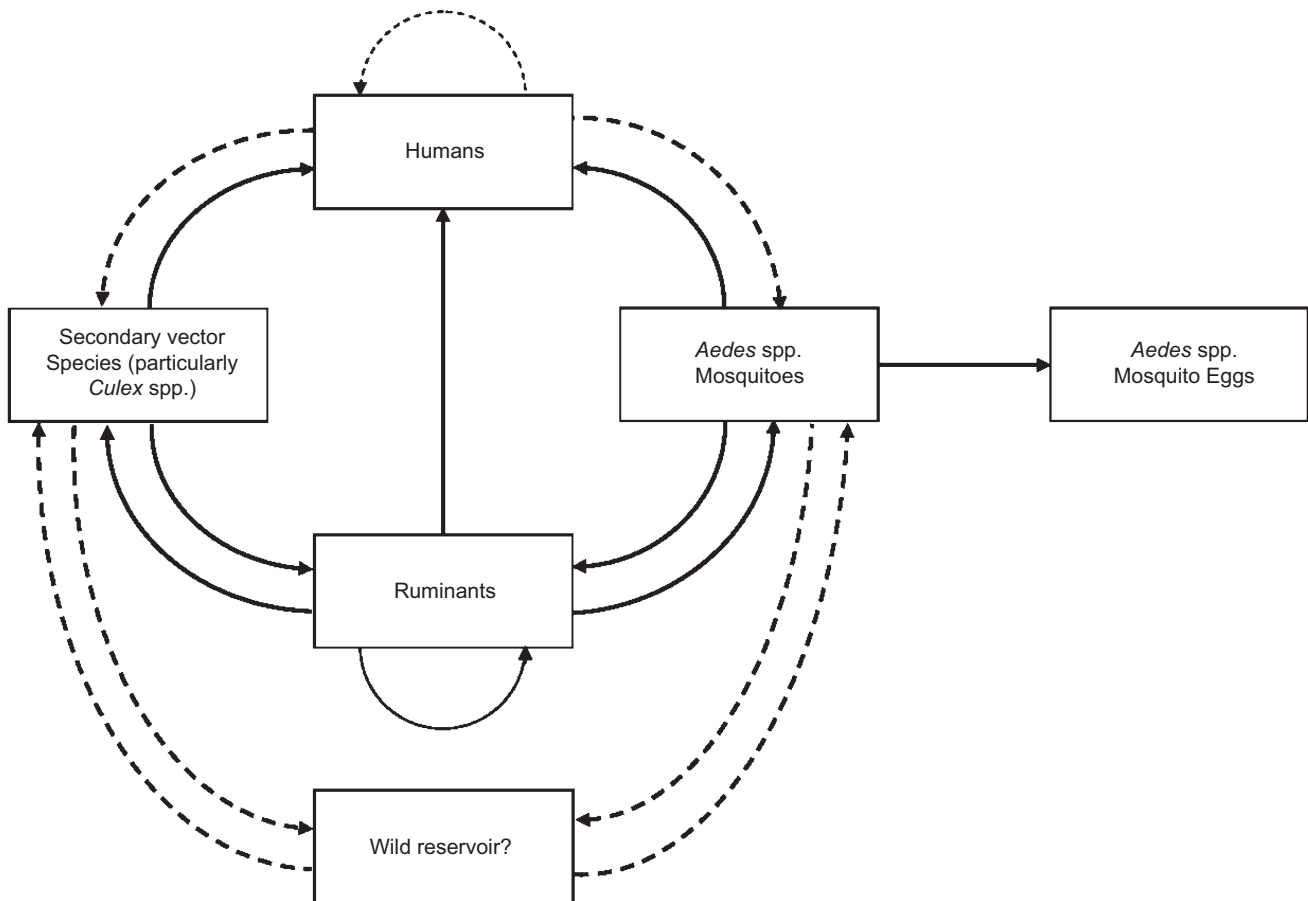


Figure 2 Transmission cycle of Rift Valley fever virus. Solid lines reflect known and established pathways of transmission; dashed lines represent pathways of potential transmission. Adapted from Chevalier *et al.*³¹

dispersal.^{16,29,40} This could have an interesting repercussion because though rare, there have been several reported cases of international travelers acquiring RVFV abroad, including a 41-year-old Canadian woman and several members of the French military.^{41,42} The epidemiological role of human hosts in RVFV transmission though presumed, probably accurately, to be negligible thus remains unresolved.

Movement of virus-carrying vectors

RVFV can be spread to new geographic regions via the movement of virus-carrying vectors. Competent vectors of RVFV include over 30 species of mosquitoes, particularly mosquitoes from the *Aedes*, *Culex* and *Anopheles* genera.^{31,43} A RVFV-infected vector introduced into a new region does not necessarily need to become established. A single or small number of infected vector introductions, if successfully transferring the virus to local animal hosts and competent domestic vectors, can facilitate virus spread.¹⁶

Wind-borne dispersal of virus-carrying vectors has been implicated in the spread of numerous arboviruses, including bluetongue and Japanese encephalitis.⁴ Dispersal via winds has been controversially posited as one of the possible mechanisms of the spread of RVFV into Egypt in 1977.^{2,36} Transcontinental movement of virus-carrying vectors for RVFV is, however, generally considered unfeasible as most competent vectors have a bio-ecology that does not involve wind-borne dispersal.⁴ However, as vector flight capacity is influenced by species as well as topographical and environmental conditions, it is difficult to generalize for all competent vector species of RVFV.³⁶

The geographical range of RVFV has reached as far north as the southern coast of the Mediterranean Basin and has spread into Egypt, Mauritania, and Saudi Arabia,^{2,4} raising concerns over the possible dispersal of RVFV-carrying vectors into EU.³⁶ While the distance across the Mediterranean Sea likely exceeds the maximum flight capacity of all RVFV-carrying vectors, this risk remains unknown and it is unclear whether this mechanism may pose theoretical potential for introduction.^{31,36} Furthermore, short distance dissemination may be possible from the coast of northern Africa to Spain.⁴⁴ Additionally, it is possible that RVFV-carrying vectors could travel to EU via the Middle East, particularly through Turkey.⁴⁵ However, RVFV remains non-existent or undetected in Turkey at present.⁴⁵ Assuming that this is a viable pathway, the risk of dissemination to the EU would be elevated during epidemic periods.³⁶ In comparison, it is highly unlikely that a vector could traverse the Atlantic Ocean to the US via natural wind dispersal, even in hurricane winds.¹⁶

A more likely mechanism for dispersal is via mechanical transport: transport of virus-carrying vectors confined within aircrafts and ship cargo holds.^{1,16} Numerous arthropods have been discovered alive within aircrafts and luggage after international flights.¹⁶ In the US,

there are currently few established measures to control the admittance of arthropods. US custom officials are not required to inspect cargo for arthropods and no public health measures, such as disinfection, are compulsory on commercial aircrafts.¹⁶ The introduction of RVFV into the US via the transport of vectors on aircraft, combined with increasing intercontinental air travel, highlights the potential for such a scenario to occur despite presumptions of low probable risk. There are limited published data on the risk of introduction of RVFV into the EU via mechanical transport of virus-carrying vectors. However, the risk is likely similar, if not greater, in the EU due to the shorter transit times to RVFV-endemic countries.

Intentional entry of RVFV

In addition to the natural movement of RVFV, the intentional introduction of RVFV is considered a viable threat. RVFV is classified as a category A priority pathogen by the National Institute of Allergy and Infectious Diseases and ranked as the third most dangerous animal threat by the National Veterinary Stockpile. RVFV was successfully developed as a biological weapon by the US offensive biological weapons program prior to the program's elimination in 1969.⁴⁶ RVFV has been identified as a high risk due to numerous characteristics facilitating its use in biological warfare. While RVFV cannot be transmitted person to person, aerosol dissemination of the virus could enable rapid widespread transmission.^{46–48} Virus potential to become established in affected areas given suitable conditions for transmission also implies the potential for sustained public and veterinary health impacts, trade restrictions, and therein significant economic burden.^{15,20,46,49,50}

In 2004, a United States Department of Agriculture study of the potential effects and severity of a RVFV bioterrorism event⁵⁰ estimated economic impacts of 1 L of RVFV in excess of \$50 billion. The study hypothesized that within 2 years, RVFV would be considered endemic within the entire continental US. United States Department of Agriculture estimates have not been published in the peer-reviewed literature, however, and no studies analyzing the severity of potential RVFV bioterrorism events have been commissioned in EU. If accurate, similar scenarios and economics costs are likely in both regions. The feasibility of a range of risk pathways for transcontinental transmission is summarized in Table 2.

ESTABLISHMENT POTENTIAL OF RVFV

Abundance and competence of domestic vectors

Similar to Crimean-Congo hemorrhagic fever,⁴ RVFV has demonstrated low vector specificity: the ability to be transmitted by numerous different vectors. Thus far, it has been identified in over 30 species of mosquitoes from seven different genera (*Aedes*, *Anopheles*,

Table 2 Feasibility and risk of pathways for transcontinental transmission

	The United States	Europe
Entry via viremic hosts		
Ruminants (legal)	Negligible	Viable (low)
Ruminants (illegal)	Negligible	Viable (unknown—likely low)
Wildlife species, zoo and circus animals	Viable (almost negligible)	Viable (almost negligible)
Humans	Unknown	Unknown
Entry via virus-carrying vectors		
Wind-borne dispersal	Negligible	Epidemic periods: unknown; inter-epidemic periods: negligible
Mechanical transport	Epidemic periods: viable (low); inter-epidemic periods: negligible	Epidemic periods: viable (low); inter-epidemic periods: negligible
Intentional entry (bioterrorism)		
	Viable (risk unknown)	Viable (risk unknown)

Coquillettidia, *Culex*, *Eretmapoites*, *Mansonia* and *Ochlerotatus*) and has the potential to be spread by other arthropods such as sand flies.^{31,51} Laboratory testing has identified several species of capable vectors throughout North America and EU.^{19,21,22,52,53} Vector competence in transmitting the virus, however, may differ among locations, even for the same mosquito species.⁵² Additionally, factors such as seasonal vector and host density, feeding preferences and foraging behaviors could have an effect on the efficiency of RVFV transmission.^{22,52} Vector competence for RVFV is poorly understood, though it is generally presumed that there exist competent domestic vectors in most regions of the US and EU.^{19,21,22,52}

The environmental conditions supporting and constraining vector transmission are poorly understood, due predominantly to a dearth of knowledge regarding mosquito ecologies in North America and the EU. Studies have demonstrated that environmental temperature may affect vector competence, but the effect varies by species.⁵⁴ In the two species that have been the focus of research, Egyptian *Culex pipens* and North American *Aedes taeniorhynchus*, viral dissemination and transmission occurred more rapidly at high temperatures.⁵⁴ More research is needed in order to understand the transmission cycle of RVFV in the US and EU, including identifying which species are likely to maintain the virus in nature and in what regions and environmental conditions.

Adequate environmental conditions and virus persistence

Research has not identified the specific temperature constraints for RVFV; however, the virus has been documented in a wide diversity of bioclimatic environments. The presence of RVFV has most commonly been described in (i) dambos (shallow depressions which provide an ideal mosquito habitat when flooded); (ii) semi-arid regions; and (iii) irrigated areas with a distinct epidemiological cycle in each different environment.^{2,4,34}

In the dambos regions traditionally found in eastern and southern Africa, the transmission cycle of RVFV is rainfall-dependent, and the beginning of the epidemic period is strongly correlated with heavy precipitation, often linked with the El Niño Southern Oscillation.^{4,30,55,56} Heavy rainfall floods the dambos and lead to a mass hatching of RVFV vectors, particularly from the *Aedes* genera.^{4,30,55} Linthicum and Davies⁵⁷ demonstrated that the *Aedes mcintoshi* species are capable of transmitting the virus to their offspring via transovarial transmission; quiescent, infected eggs may survive up to several years of conditions unsuitable for active transmission including cold and dry periods.^{55,57} RVFV in these regions typically re-emerges every 5–15 years, reinitiated by a suitable rainfall event.^{16,34,55}

In contrast to the dambos, RVFV outbreaks in semi-arid regions, seen in West Africa, have not been correlated with rainfall surplus, and were in fact often observed during years of rainfall deficit.^{58,59} The mechanism for virus persistence in these regions remains unclear. During the wet season, RVFV has been shown to circulate at low levels without clinical signs.^{4,31,60} In dry seasons, the maintenance of the virus may be linked to transovarial transmission of the *Aedes mcintoshi* mosquito as demonstrated in East Africa or through an unknown wildlife reservoir.^{4,34} Additionally, it is possible that the multiple outbreaks are a result of multiple introductions—likely through nomadic herds from neighboring endemic areas—rather than through viral persistence.³⁴

In irrigation regions, permanent bodies of water enable year-long transmission of RVFV through predominantly *Culex* species mosquitoes.^{4,34} Dam construction is believed to have facilitated outbreaks in semi-arid regions by the creation of new vector habitat due to subsequent irrigation and flooding.^{5,49,61}

Despite the adaptation of the virus to various bioclimatic environments, it is unknown whether and to what extent RVFV could persist in the US and EU. Bioclimatic suitability for RVFV based on vector species from current transmission zones may differ from climatic requirements of competent vectors in the US and EU. Hongoh *et al.*,⁶² for example, note that climatic and biological determinants of arbovirus vectors in North America are not generalizable and public health risk assessments thus necessitate species-specific ecological research. Inferring the suitability of environments for vectors of RVFV is constrained by limited—and dated—research on the topic in both the US and EU. Recent studies have attempted to identify the competence of local vectors in the laboratory; however, our understanding of vector competence for RVFV in the US and EU remains limited and largely based on presumption or a small number of studies. Temperature is often found to be a limiting factor for many arboviruses at broad regional scales, and the occurrence of RVFV in primarily warm climates suggests the likelihood of some temperature dependence on transmission.^{63,64} It is unclear, however, whether it is the vector or the virus life cycle that is subject to temperature dependence. If the virus exhibits temperature constraints, spread into temperate conditions is unlikely. If the current vectors in endemic regions are temperature-dependent species, however, then it is possible that competent vectors exist in temperate regions. This distinction is critical to inferring the potential for transmission into cooler climates in the US and EU. Notably, recent emergence of RVFV in Madagascar illustrates that RVFV is able to circulate under more temperate conditions.^{9,10,34}

Despite the uncertainty, Konrad and Miller⁶⁴ identified high-risk regions and time periods for RVFV entry and establishment in the US; however, the model uses the temperature/transmission relationship of WNV as a proxy for RVFV due to the lack of knowledge. Posited high-risk regions include southern California and Texas, the Baltimore and New York City metro areas, and Florida where parts of the state are at risk up to 325 days per year. Additionally, almost the entire continental US is posited as having potential transmission in August.⁶⁴

Projections of increased temperature due to climate change may alter the suitability of habitats in the US and EU,^{65–67} especially as temperature is posited to be the primary limiting factor of RVFV transmission in the US.^{63,64} For example, the Intergovernmental Panel on Climate Change⁶⁸ concluded that the projected increase in rainfall would likely increase the risk of RVFV in livestock and humans. However, climatic impacts on mosquito survival and distribution vary substantially by species, and impacts are thus difficult to infer.⁶²

Wildlife reservoirs likely play an important role in maintaining the virus, particularly during interepizootic periods. While currently no reservoir has been established, antibodies to RVFV have been detected in a large number of species including rodents,^{69–71} bats^{72,73} and African buffaloes.^{2,70}

DISCUSSION

It is generally posited in the literature that low risk of pathogen introduction is the dominant constraint to RVFV spread onto new continents. Similarly, it is widely assumed that establishment of the virus in native mosquito populations, once introduced, is probable, and that environmental conditions in North America and Europe are suitable for transmission. Our review, particularly through the comparison of RVFV with WNV, indicates that this presumption of pathogen introduction as the key limiting factor of RVFV spread may be largely oversimplified. As is now generally accepted for WNV, the RVFV

pathogen may have been introduced many times into the US and EU, most plausibly via entry of viremic animals or airborne transport of virus-carrying vectors. Introduction of the pathogen via viremic ruminants into the US and the EU is limited by trade barriers. It is notable that trade barriers to RVFV in the US are to some extent serendipitous; if RVFV were to spread to regions not endemic for foot-and-mouth disease, then the effective barrier to RVFV introduction would be reduced. Livestock continue to be traded between Egypt, a potentially endemic country, the Middle East and the EU, representing a possible entry point for pathogen introduction. Mechanical transport of virus-carrying vectors on airlines is also a feasible mechanism for introduction. Increasing international travel has meant that vector transport via this route may expand. This method was possibly the source of WNV introduction into North America and contributes to numerous cases of 'airport' malaria annually.^{74,75}

Additionally, while ecological conditions and local vectors are presumed to be suitable for transmission, temperature constraints to vector, and possibly pathogen, survival and replication may reduce both the probability and the temporal duration of transmission. For example, the conditions for malaria transmission (suitable vector, adequate environmental conditions, regular pathogen introduction via infected travelers) exist in both the US and EU, yet transmission parameters and risk factors are sufficiently low that autochthonous malaria transmission is considered negligible in most countries.⁷⁶ While environmental conditions may be suitable for RVFV establishment in the US and EU, conditions may be suboptimal, thus constraining transmission in sporadic cases of pathogen introduction. Delayed transmission of RVFV on the Arabian Peninsula suggests that it was necessary for pathogen introduction to coincide with suitable seasonal conditions to allow establishment. This is also supported by the presence of established transmission in predominantly warm or hot climates; evidence of transmission and establishment in temperate regions is less prevalent or convincing. If indeed suboptimal temperatures do restrict the probability of establishment, projections of a warmer climates may have important—and non-linear—implications for RVFV transmission potential.

A combination of pathogen introduction and optimal environmental conditions for establishment, and chance coincidence of both sets of factors in both space and time, would be required for RVFV to establish in a new region. Currently, these conditions have not yet coincided sufficiently to allow for disease spread to the US and EU. It seems likely that the risk of sporadic introduction of the pathogen into the US and the EU is relatively high, though quantification of such risk remains unfeasible and elusive. Sustained transmission may in contrast be constrained predominantly by a lack of coincidence in a sufficient number of such introductions with conditions sufficiently optimal to allow sustained transmission, sufficient and adjacent densities of competent vector and host populations, and ideal temperature and water conditions for vector and pathogen survival. Such probabilities, however, are stochastic rather than deterministic, meaning that coincidence of suitable conditions is subject to a degree of chance. The introduction of WNV to North America provides a prescient example reflective of such chance introduction. The introduction and rapid spread of WNV into the US and Canada was not predicted in the literature, and the probability of WNV emergence may not have been substantively different than for RVFV currently. Sporadic introduction of the WNV pathogen may have occurred more frequently than for RVFV given existing trade barriers to RVFV introduction. In contrast, there are many more

vector species believed to be competent for RVFV transmission than exist for WNV. There are no published, peer-reviewed risk assessments or reviews prior to the establishment of WNV in North America, suggesting that the risk of its introduction and establishment was presumably deemed relatively low. Herein, the spread of WNV highlights the need to consider emergent pathogen introductions as stochastic processes, requiring the use of stochastic, process-based models to simulate scenarios of emergence and identify parameters to which transmission probabilities are highly sensitive. A recent review of the probability of (autochthonous) malaria reemergence in Canada⁷⁶ similarly underscores the need for stochastic approaches to emergent disease risk modeling, noting that transmission determinants interact, are generally probabilistic rather than deterministic and are temporally and spatially scale-dependent.

Based on existing knowledge and qualitative assessment, if RVFV were to be introduced, the most plausible conditions for such introduction would be via vector transport on aircraft or trade of ruminants or wild animals during an epizootic in endemic countries, when levels of viremia are high. The probability of establishment would be higher during the warmer summer season and vary within season based on local vector ecologies. Risks are likely higher in the EU, where proximity to endemic regions is higher. The ecology of RVFV in the US and EU, particularly related to vector ecologies, is very poorly understood. Concern for the potential emergence and spread of arboviruses to US and EU has not been met with equivalent development of entomological experience or funding to investigate vectors, pathogens and environmental conditions for transmission. To this end, evaluations of risk for potentially emergent pathogens such as RVFV—including the one presented here—are necessarily qualitative and speculative; scenario models, while useful even when data are lacking, are difficult to parameterize given poor input data.

Future global trends may increase the likelihood of risk factors for RVFV spread. Temperature dependence of vectors and some pathogens indicates that projected climate changes will affect and possibly extend or enhance areas and seasons suitable for transmission. Growth in international travel and trade will also increase the frequency of sporadic pathogen introduction between continents. Bioterrorism and intentional introduction of viruses remains an unknown risk requiring separate risk assessment and alternate scenarios from unintentional pathways of spread, though even here entomological research and knowledge of vector ecologies would facilitate risk assessment once a hypothetical pathogen was introduced.

The spread and introduction of WNV in North America highlights the potential for unpredictable and seemingly low-risk scenarios to occur, with significant implications for public health. The introduction of RVFV has the potential to be more severe than has been seen for WNV from both a public health and economic perspective. Despite this, there is insufficient research and literature available to evaluate RVFV risk beyond qualitative presumption. Such research should focus on improved understanding of entomological and environmental parameters for transmission, as well as development of stochastic scenario models. In parallel, consideration of prevention measures may be prudent given the potential for increasing risks under climate change and global travel and trade projections. Evaluation of trade barriers and aircraft disinfection regulations, for example, may become increasingly warranted. The current probability of RVFV spreading to the US and the EU is presumed to be low, and such presumptions are probably correct. However, the probability that a

related arbovirus or similar vector-borne disease may spread to these continents is higher, and these probabilities are likely to increase in the future.

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