Rift Valley Fever: An Emerging Mosquito-Borne Disease*

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Abstract

Rift Valley fever (RVF), an emerging mosquito-borne zoonotic infectious viral disease caused by the RVF virus (RVFV) (Bunyaviridae*: Phlebovirus*), presents significant threats to global public health and agriculture in Africa and the Middle East. RVFV is listed as a select agent with significant potential for international spread and use in bioterrorism. RVFV has caused large, devastating periodic epizootics and epidemics in Africa over the past ~60 years, with severe economic and nutritional impacts on humans from illness and livestock loss. In the past 15 years alone, RVFV caused tens of thousands of human cases, hundreds of human deaths, and more than 100,000 domestic animal deaths. Cattle, sheep, goats, and camels are particularly susceptible to RVF and serve as amplifying hosts for the virus. This review highlights recent research on RVF, focusing on vectors and their ecology, transmission dynamics, and use of environmental and climate data to predict disease outbreaks. Important directions for future research are also discussed.

INTRODUCTION

RVF: Rift Valley fever

RVFV: Rift Valley fever virus

ENSO: El Niño Southern Oscillation

Rift Valley fever (RVF) is an emerging mosquito-borne zoonotic viral disease of livestock and humans in Africa and the Middle East that is closely associated with high-rainfall conditions (24, 66). First described in the early 1930s, it was quickly determined that the Rift Valley fever virus (RVFV) was transmitted primarily by mosquitoes but could also be transmitted by direct contact with infected tissues and fluids (18). The earliest published studies of RVF and RVFV indicated that one effective way to interrupt RVFV transmission to livestock was to move animals to higher elevations away from flooded areas harboring mosquito populations (18). Later studies further indicated that ecological variables could be monitored for conditions likely to favor the growth of RVFV mosquito vectors, thus providing a basis for planning measures to reduce virus transmission during outbreaks (24). Nearly 90 years later, in the continued absence of approved, stable, and effective RVFV vaccines for livestock and humans, reducing contact with infected mosquitoes may still be the best way to prevent RVFV transmission. This comprehensive review is a primary reference for expanding scientific, public health, agriculture, and government stakeholders. In addition to this review, which focuses on entomological, ecological, and predictive aspects of RVF, a number of other reviews emphasize other aspects of this important emerging disease (10, 26, 37, 44, 81, 90, 91, 99, 102, 121).

HISTORICAL BACKGROUND, EPIDEMIOLOGY, AND ECOLOGICAL DYNAMICS

Discovery of Agent and Insect Vectors

RVF was first discovered in 1930 after mortality and abortions occurred in sheep along the north shore of Lake Naivasha, near Gilgil, Kenya (17, 19). Reports of death and abortions in exotic sheep kept for wool production as early as 1913 in the annual reports of the Veterinary Research Laboratory in Kabete, Kenya, may well have been due to RVF (20). Mosquitoes primarily of the genus *Aedes* but also of the genus *Eretmapodites* were suspected and confirmed as the vector of the virus in Uganda in 1948 (100), and virus isolations were subsequently made from mosquitoes collected during epizootics in other countries, including Kenya and Egypt (22, 69, 72, 75, 82, 100).

History, Geography, and Climatic Associations of Epizootics and Epidemics

Following the initial description in Kenya in 1930 and a subsequent outbreak in 1936–1937, RVFV caused a large epizootic in 1950–1951 in South Africa when an estimated 100,000 sheep died and 500,000 aborted (121). Repeated epizootics and epidemics occurred in eastern and southern Africa (Namibia, Zimbabwe, Kenya, Nigeria, South Africa, Malawi, Mozambique, Sudan, and Zambia) during years with exceptional rainfall.

Essentially all RVF outbreaks in sub-Saharan Africa have occurred after prolonged excessive rainfall in bushed and wooded savanna grasslands (24). RVF epizootics in the Horn of Africa typically occur late in the year during El Niño Southern Oscillation (ENSO) events. ENSO events occur when the eastern equatorial Pacific Ocean and western equatorial Indian Ocean sea surface temperatures are concurrently anomalously elevated, resulting in above-normal rainfall that extends for several months into the following year (62).

RVFV was first isolated in West Africa in Nigeria in 1967 and was associated with imported cattle (81). Limited to sub-Saharan Africa until 1977, RVFV expanded into Egypt, where it caused a significant epizootic/epidemic from 1977 to 1979 associated with irrigation schemes after the virus was apparently introduced from Sudan during an epizootic there in 1976 (80). Epidemic RVF disease was first described in West Africa in 1987, occurring for the first time in the Sahel during a

major outbreak in southern Mauritania and northern Senegal that was associated with the closing of the Diama Dam on the Senegal River but not associated with an excess of rainfall (28, 65).

The virus first left the continent of Africa in 1979, when it was detected in Madagascar, later causing an epizootic in Madagascar in 1990–1991 (85). RVF emerged in the Arabian Peninsula in 2000 and caused a significant epizootic/epidemic in the Tihamah coastal plains of Yemen and Saudi Arabia (9). Most recently, RVFV antibodies were detected in the Comoros Islands (95) and Mayotte (60). To date, RVF has been found in over 30 countries. RVF outbreaks have occurred in areas of Africa ranging from the dry, low-rainfall climate of Egypt, to the wet forest areas of Uganda and the Central African Republic, to the dry savanna lands of East and southern Africa. Temperatures associated with RVF outbreaks are characteristically cooler than dry-season temperatures.

The mechanisms by which RVF has moved to new geographic areas such as Egypt and the Arabian Peninsula are unknown. Some possibilities include migrating or windblown vectors, movements or importations of viremic mammals such as camels, phoresy of RVFV-infected ticks on migratory birds, or movement of infected humans or vectors by aircraft. Birds have been suggested as possible sources of the virus (3); however, various studies have demonstrated that birds do not become infected or develop antibodies (23). Mosquitoes involved in RVFV transmission do not appear to migrate or move appreciable distances (63).

Incidence and Risk Factors

Supplemental Material

During epizootics, RVFV infection in sheep, cattle, and goats approaches 100% (**Supplemental Figure 1**; follow the **Supplemental Material link** from the Annual Reviews home page at **http://www.annualreviews.org**). Exotic breeds of cattle, sheep, and goats, particularly from Europe, appear to be at higher risk of severe disease (21). Although it remains difficult to estimate morbidity and mortality of RVFV infection in humans, seven of the last nine major outbreaks over the past 15 years produced human cases outside of East Africa (16). Approximately 339,000 human cases occurred in five of these outbreaks, for which human case data were published (16). The proximity of humans to animals is a key factor determining whether humans become involved in the RVF disease cycle. This proximity varies extensively depending on the type of animal husbandry and other agricultural practices established in a particular area. At highest risk of infection are persons who are in close contact with RVFV-infected animals and the mosquitoes feeding on these animals (pastoralists, farmers, shepherds) or who work with meat products (slaughterhouse workers, butchers). Laboratory workers and veterinarians working with RVFV are also at high risk (81). We provide additional information on the historical background of RVF (e.g., geographic groupings, role of land-use change, climate and weather links to RVF epizootic periods, social and economic impact, and movement and trade restrictions) in the **Supplemental Material**.

TRANSMISSION CYCLES AND VECTOR ECOLOGY

RVFV Transmission in Natural Environments

Multiple studies have been conducted on the epidemiology of RVFV among vectors, hosts, and reservoirs in its endemic range, in particular to understand environmental links with vector life cycles that may underlie the episodic nature of large epizootics and epidemics.

Vectors. Since the discovery of RVFV in Kenya, numerous blood-feeding arthropods have been incriminated as vectors of the virus. RVFV has been isolated from more than 53 species in 8 genera within the family Culicidae in regions where epizootics have occurred. To understand and model environment-driven RVF outbreaks and mosquito vector population dynamics and develop effective intervention strategies, the mosquito vector species must be identified. We list in **Table 1**

Table 1 Species from RVF-endemic or RVF-emerging regions and their RVFV status[a,b]

Group	Species	Location, year	RVFV status	Reference(s)
Mosquitoes	*Aedes aegypti*	Kenya 1956, Sudan 2007, USAMRIID colony	FI, LI, LT, MT	32, 47, 84, 98
	Aedes aegypti formosus	Sub-Saharan Africa 1983	MT	56
	Aedes africanus	Uganda 1954, 1956	FI	120
	Aedes caballus	South Africa 1953	FI, LI, LT	43, 77, 78
	Aedes cinereus	South Africa 1974, 1975	FI	78
	Aedes circumluteolus	Kenya 1985, 1989; South Africa 1955, 1956, 1981; Uganda 1955	FI, LI	55, 57, 74, 78, 79, 113, 120
	Aedes cumminsii	Burkina-Faso 1983	FI	38, 81
	Aedes dalzieli	Senegal 1974, 1975, 1983; Kenya 1982	FI	38, 81
	Aedes dendrophilus (*Aedes deboeri*)	Uganda (Semliki Forest) 1944	FI	100
	Aedes dentatus	South Africa 1970, 1971, 1980s; Zimbabwe 1969	FI, LI	53, 73, 81
	Aedes durbanensis	Kenya 1937, South Africa 1937	FI, LT	31, 81
	Aedes fowleri	Senegal 2002, 2003	FI	8
	Aedes furcifer	Burkina-Faso 1983	FI	38, 81
	Aedes juppi	South Africa 1974, 1975, 1978, 1984, 1987	FI, LI, LT	42, 53, 77, 78
	Aedes mcintoshi (also called *Aedes lineatopennis*)	Zimbabwe 1969; South Africa 1974, 1975; Kenya 1978, 1979, 1981–1984, 2006, 2007	FI, LI, LT	22, 69, 73, 74, 77, 78, 97, 113
	Aedes ochraceus	Senegal 1991–1996, 1993; Kenya 2006, 2007	FI	38, 72, 97
	Aedes palpalis	Central African Republic 1969	FI, LI	27, 38, 113
	Aedes pembaensis	Kenya 2006, 2007	FI	72, 97
	Aedes tarsalis	Uganda 1944, 1955	FI	100
	Aedes triseriatus	USAMRIID colony	LI, LT	32
	Aedes vexans	Senegal 1991–1996, 1993, 2002, 2003, 2011, 2012; Saudi Arabia 2000	FI, LI	1, 8, 33, 38, 54, 83
	Anopheles christyi	Kenya 1982	FI	81
	Anopheles cinereus	South Africa 1974, 1975	FI	78
	Anopheles coustani	Zimbabwe 1969; Kenya 1978; Sudan 2007; Madagascar 2008, 2009	FI, LI	22, 73, 76, 94, 98
	Anopheles coustani, *Anopheles fusicolor*	Madagascar 1979	FI	15
	Anopheles gambiae arabiensis	Sudan 2007	FI	98
	Anopheles pauliani, *Anopheles squamosus*	Madagascar 1979	FI	81
	Anopheles pharoensis	Kenya 1982	FI	81
	Anopheles squamosus	South Africa 1975; Madagascar 2008, 2009; Kenya 2006, 2007	FI, LT	19, 72, 78, 94, 97

(*Continued*)

Table 1 (*Continued*)

Group	Species	Location, year	RVFV status	Reference(s)
	Coquillettidia fuscopennata (*Mansonia fuscopennata*, *Taeniorhynchus fuscopennatus*, *Taeniorhynchus brevipalpis*)	Kenya 1930, Uganda 1960	FI, LI, LT	18, 19, 122
	Coquillettidia grandidieri	Madagascar 1979	FI	81
	Coquillettidia microannulata (*Mansonia microannulata*)	Kenya 1932	LI	18, 31, 82
	Coquillettidia versicolor (*Mansonia versicolor*)	Kenya 1932	LI	18, 31, 82
	Culex antennatus	Nigeria 1967–1970; Egypt 1977, 2003; Kenya 1981–1984, 1989; Madagascar 2008, 2009	FI, LI	39, 45, 58, 59, 69, 71, 82, 94, 113
	Culex antennatus, *annuloris* group, *simpsoni*, *vansomereni*	Madagascar 1979, Kenya 1982	FI	81
	Culex bitaeniorhynchus	Kenya 2006, 2007	FI	72, 97
	Culex fatigans	Kenya 1930	LT	19, 31
	Culex neavei	South Africa 1981	FI, LI, LT	74, 79
	Culex pipiens	South Africa 1970, 1981; Egypt 1977, 1978; Kenya 1989	FI, LI, LT, MT	19, 35, 47, 49, 69, 73, 79, 82, 113
	Culex pipiens complex	Sudan 2007	FI	98
	Culex poicilipes	Senegal 1993, 1998, 1999, 2002, 2003; Mauritania 1998, 1999; Sudan 2007; Kenya 2006, 2007; Egypt 1993; South Africa 1988	FI, LI, LT	8, 25, 53, 72, 97, 98, 115
	Culex quinquefasciatus	Kenya 2006, 2007; Sudan 2011, 2012	FI, LT	1, 33, 72, 78, 97
	Culex theileri	South Africa 1953, 1956, 1970, 1974, 1975; Zimbabwe 1969; Kenya 1982	FI, LI, LT	43, 73, 74, 75, 77, 78
	Culex tritaeniorhynchus	Saudi Arabia 2000	FI, LI	54, 90
	Culex univittatus	Kenya 2006, 2007	FI, LT	72, 77, 78, 97
	Culex zombaensis	Zimbabwe 1978; South Africa 1981; Kenya 1981–1984, 1989	FI, LI, LT	69, 74, 79
	Eretmapodites chrysogaster/Eretmapodites intermedius	Uganda 1944, 1948	FI, LI, LT	100
	Eretmapodites spp. (*E. chrysogaster*, *E. inornatus* Newst. group, *E. ferox*, *E. leucopus* sbsp. *productus* Edw.)	Uganda 1948	FI	31, 100
	Eretmapodites quinquevittatus	South Africa 1971; Kenya 1981–1984	FI, LI, LT	69, 73, 74, 78
	Eumelanomyia rubinotus	Kenya 1982	FI	81

(*Continued*)

Table 1 (*Continued*)

Group	Species	Location, year	RVFV status	Reference(s)
	Mansonia africana	Uganda 1959, 1968; Central African Republic 1969; Senegal 2002, 2003; Kenya 2006, 2007	FI	8, 46, 72, 97, 122
	Mansonia uniformis	Uganda 1960; Madagascar 1979; Senegal 2002, 2003; Kenya 2006, 2007	FI, LI	8, 72, 81, 97, 100, 123
Ticks	*Amblyomma variegatum*	Central African Republic 1983	FI	38
	Hyalomma truncatum	Colony from National Institute of Virology, South Africa	LI, LT	70
	Rhipicephalus appendiculatus	Kenya 1932	LI, LT (nymphs)	18
Biting midges	*Culicoides* spp. [*C. austeni*, *C. krameri*, *C. imicola* (*C. pallidipennis*), *C. shulzei*]	Uganda 1968; Nigeria 1967–1970; Kenya 1978, 1979	FI (limited)	22, 34, 38, 46, 58, 59
Tapeworm	*Taenia crassicollis*	Kenya late 1940s or early 1950s	LI, LT	31, 36
Sand flies	*Phlebotomus duboscqi*	Kenya strain	LI, LT	29, 114
	Phlebotomus sergenti	Egypt strain	LI	29
	Sergentomyia schwetzi	Kenya strain	LI	29
Other flies	*Simulium* spp.	South Africa 1953	FI	118
	Glossina morsitans	Zimbabwe strain	MT	47

[a]Species from which RVFV has been isolated from field-collected specimens (field isolations, FI), species that have been infected with RVFV under experimental conditions (lab infections, LI), species that have transmitted RVFV under experimental conditions (lab transmissions, LT), and species that are capable of mechanical transmission of RVFV under experimental conditions (mechanical transmission, MT).
[b]Data for this table are from the primary literature and several previous reviews (27, 31, 81, 82, 90, 99).
Abbreviation: USAMRIID, United States Army Medical Research Institute of Infectious Diseases.

mosquito species implicated in RVFV epidemiology as well as other possible arthropod vectors of RVFV from endemic regions, taking into account field infection rates and laboratory infection and transmission studies. In **Table 2** we list potential arthropod vectors of RVFV based on vector competence studies of potential vectors in regions where the virus is a potentially emerging threat, such as the United States, Australia, and Europe.

Primary mosquito vectors, endemic cycles, and the environment. Serosurveys conducted in a number of areas in the absence of epizootic circulation detected RVFV-neutralizing antibodies, indicating an endemic maintenance cycle independent of epizootics (92). Linthicum et al. (69) isolated RVFV during interepizootic periods from male and female *Aedes mcintoshi* reared to adult from larvae collected in the field after artificial flooding. These findings led to the hypothesis that RVFV is maintained during interepizootic periods in an endemic cycle that depends on intermittent periods of heavy rainfall and periodic short-term flooding of low-lying habitats, known as *dambos* in East Africa (**Supplemental Figure 2**) and *pans* and *vleis* in South Africa (2), and on the vertical transmission of the virus (i.e., transovarial inheritance of the virus from female mosquitoes to offspring) by floodwater *Aedes* mosquitoes (**Figure 1**).

Supplemental Material

In the endemic cycle (**Figure 1**, *left*) periodic short-term flooding replenishes RVFV-infected *Aedes mcintoshi* or other *Aedes* species eggs in dambo-type habitats, which may involve a limited transmission cycle between *Aedes* and local vertebrate hosts. RVFV-infected *Aedes* eggs in dambos

Table 2 Species from potentially RVF-emerging regions and their RVFV status[a]

Species	Origin	Vector potential	RVFV status	Reference(s)
United States				
Aedes abserratus	Michigan	Low	LI, LT	41
Aedes aegypti	USAMRIID colony	Moderate	LI, LT	30
Aedes albopictus	Texas	Moderate	LI, LT	105
Aedes atlanticus	Florida	Moderate	LI, LT	107
Aedes canadensis	Maryland	High	LI, LT	41
Aedes cantator	Maryland	Moderate	LI, LT	41
Aedes dorsalis	California, Colorado	Low	LI, LT	116
Aedes infirmatus	Florida	Low	LI, LT	107
Aedes japonicus japonicus	North Carolina	High	LI, LT	109
Aedes sollicitans	Virginia	Moderate	LI, LT	41
Aedes taeniorhynchus	Virginia, Florida	High (VA), moderate (FL)	LI, LT, MT	41, 47, 106, 111
Aedes triseriatus	USAMRIID colony; Maryland	Moderate (MD)	LI, LT	30, 41
Aedes vexans	Florida, California, Colorado	Moderate (FL), low (CA, CO)	LI, LT	107, 111, 116
Anopheles bradleyi-crucians	Maryland	Low	LI, LT	41
Anopheles crucians	Florida	Very low	LI, LT	107
Coquillettidia perturbans	Florida	Very high	LI, LT	107
Culex erraticus	Louisiana	Moderate/low	LI, LT	111, 116
Culex erythrothorax	California	Low	LI, LT	116
Culex nigripalpus	Florida, Louisiana	Low (FL), moderate (LA)	LI, LT	107, 111, 116
Culex pipiens	California, Colorado	High	LI, LT	116
Culex quinquefasciatus	Louisiana	–	LI	111
Culex salinarius	Maryland, Louisiana	Moderate (MD)	LI, LT	41, 111
Culex tarsalis	California, Colorado	High	LI, LT	41, 116
Culex territans	Maryland	Moderate	LI, LT	41
Mansonia dyari	Florida	Moderate	LI, LT	107
Psorophora ciliata	Florida	–	LI	108
Psorophora columbiae	Florida	Low	LI, LT	108
Psorophora ferox	Florida	High	LI, LT	107
Culicoides variipennis	Colorado	Moderate (MT), limited (LI)	MT, LI	47, 51
Stomoxys calcitrans	Texas, Florida	Moderate	MT	47, 110
Australia				
Aedes notoscriptus	Brisbane	High	LI, LT	112
Aedes vigilax	North Queensland	High	LI, LT	112
Culex annulirostris	Brisbane	High	LI, LT	112
Culex quinquefasciatus	Brisbane	Low	LI, LT	112
Canada				
Aedes communis	Manitoba	–	LI	50
Aedes dorsalis	Manitoba	–	LI	50
Aedes fitchii	Manitoba	–	LI	50
Aedes implicatus	Manitoba	–	LI	50
Aedes sticticus	Manitoba	Low	LI, LT	50

(*Continued*)

Table 2 (*Continued*)

Species	Origin	Vector potential	RVFV status	Reference(s)
Aedes stimulans	Manitoba	–	LI	50
Aedes vexans	Manitoba	–	LI	50
Coquillettidia perturbans	Manitoba	Low	LI, LT	50
Culex tarsalis	Manitoba	Low	LI, LT	50
Culiseta inornata	Manitoba	–	LI	50
Europe				
Aedes caspius	France	–	LI	86
Aedes detritus	France	–	LI	86
Culex pipiens	Southern France and Tunisia	–	LI	86
South America				
Lutzomyia longipalpis	Brazil strain	Moderate	LI, MT	47, 48
Israel				
Phlebotomus papatasi	Israel strain	Moderate	LI, LT	29

[a]Species that have been infected with RVFV under experimental conditions (lab infections, LI), species that have transmitted RVFV under experimental conditions (lab transmissions, LT), and species that have been found capable of mechanical transmission of RVFV under experimental conditions (mechanical transmission, MT).
Abbreviation: USAMRIID, United States Army Medical Research Institute of Infectious Diseases.

may survive and lie dormant through a single dry year or successive dry years, and are replaced or augmented at low or moderate levels with new cohorts in normal or moderately rainy years when dambos flood for short periods of time. Dambos vary from a few meters to many kilometers in length. Typically, emergent vegetation is a key component of habitats that enable survival of immature mosquitoes (81). Field experiments conducted in flooded dambo habitats have estimated that nearly one million mosquitoes can emerge from a single flooding of a 1,800 m^2 habitat (63). On average, the emerging mosquitoes dispersed a relatively short distance (0.15 km) and had a high daily survival rate of approximately 0.84. The maintenance cycle of RVFV shown in **Figure 1** has two different components, each of which is dependent on the amount and duration of rainfall; currently, this maintenance cycle is believed to occur only in sub-Saharan Africa (6). The endemic component of the maintenance cycle likely goes unrecognized but produces the seroconversions observed during interepizootic periods (61, 81).

Climate-driven epizootics. RVF epizootics and epidemics have occurred during exceptional years of above-normal and prolonged rainfall (**Figure 1**, *right*). Anomalously widespread and prolonged heavy rainfall raises the water table in certain areas, flooding dambo-type grassland depressions at greater depths and for extended periods compared with interepizootic (or endemic) periods. Within the first 1–2 days following flooding, *Aedes* eggs dormant in the soil are induced to hatch, causing the subsequent emergence and survival of at least one very large generation of virus-infected *Aedes* mosquitoes (67, 68). These mosquitoes then transmit RVFV to amplifying vertebrate hosts that may begin the epizootic cycle.

Vertebrate amplification and secondary mosquito vectors in epizootics. The most important known vertebrate species in RVF epizootics and epidemics are ungulate livestock, specifically sheep, cattle, and goats. Infection of domestic animals is initiated by probing or blood-feeding by female *Aedes* mosquitoes (the primary mosquito vectors) that possess a disseminated salivary gland RVFV infection. The length of time needed for *Aedes* mosquitoes to mature and develop disseminated RVFV infections after sustained flooding, followed by the length of time needed for

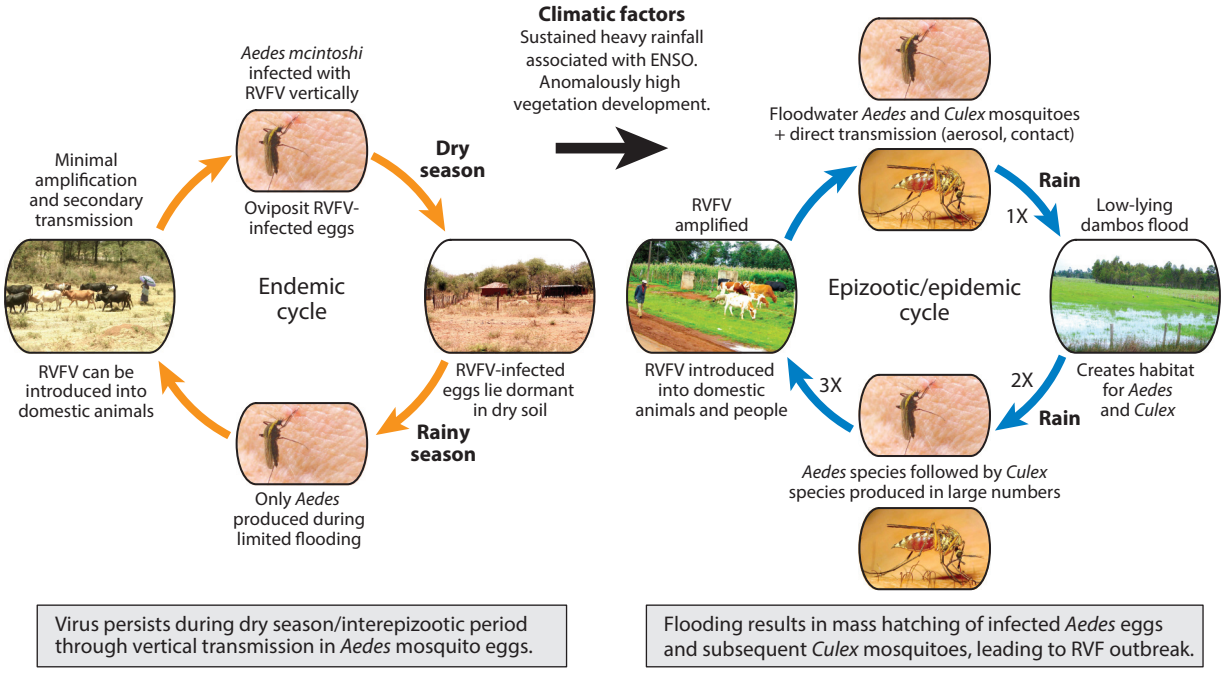

Figure 1

Schematic of the Rift Valley fever virus (RVFV) life cycle depicting the endemic (*left*) and epizootic/epidemic (*right*) phases, depending on the temporal and spatial extent of excessive rainfall. The 1X, 2X, and 3X labels in the epizootic/epidemic phase indicate key pathways that may be targeted by mosquito control measures, for instance, larviciding at 1X or 2X or adulticiding at 3X, for substantial reduction of RVFV transmission. Abbreviation: ENSO, El Niño Southern Oscillation.

RVFV to replicate to produce very high viremias in the amplifying vertebrate hosts, may coincide favorably later in the epizootic period with the stagnation of floodwaters. Stagnant floodwaters become colonized by ovipositing *Culex* and *Mansonia* species and subsequently produce vast populations of secondary RVFV vector mosquitoes, which become the dominant mosquito populations 30–40 days after flooding (67, 68).

After infection from primary *Aedes* vectors, livestock species, especially sheep populations, suffer significant morbidity, mortality, and up to 100% abortion; and their rapid, high viremias are sufficient to infect the abundant secondary mosquito vector species (61, 72). *Culex* and *Mansonia* species then serve as the primary horizontal vectors of RVFV between highly viremic domestic animals and humans (73). The pace of the epizootic is compounded because moribund ungulates infected with RVFV display little mosquito avoidance behavior and are preferentially fed upon by secondary vectors (106). Unfortunately, this period of secondary transmission occurs typically when clinical signs of the disease are first seen and the opportunity for effective control or reduction of primary transmission has passed.

The epidemic component of the maintenance cycle occurs infrequently and involves exceptionally heavy rainfall over an extended period up to many months, resulting in epizootics and epidemics that occur approximately every 5 to 15 years, depending on the ENSO phenomenon, described below, and the immunological status of host species (6). Cattle, sheep, and goats are the most important amplifying hosts for RVFV, and the numbers of each in a particular region play a key role in determining which host(s) is the most important. The immunological status of these

vertebrates critically determines if and when the next epizootic will occur. The high attack rates and subsequent high RVFV antibody prevalence rates will likely prevent another RVF outbreak for several years until new immunologically naïve animals replace older animals, even if rainfall conditions create ideal breeding conditions for virus-infected *Aedes* mosquitoes.

Vector host preference. Relatively few studies have examined host preference and biting activity of potential RVFV vectors in endemic areas. *Aedes ochraceus*, *Aedes mcintoshi*, and *Mansonia uniformis* infected with RVFV during the epizootic in Kenya in 2006–2007 fed mostly on goats, followed by cattle, donkeys, sheep, and humans (72). These mosquito species tend to be active during crepuscular periods.

Wild vertebrate hosts. The role of wild vertebrates (vertebrate hosts other than domestic animals) in the maintenance of RVFV during interepizootic periods or as amplifying hosts during epizootics has been studied since the first recognition of RVF disease. Mammalian wildlife species do become infected with RVFV, especially during epizootics, but their possible roles in interepizootic and epizootic periods are not known (12). Evidence suggests that wild rodents are not amplification hosts of the virus (21, 101, 119), yet evidence exists of serologically positive rodents in South Africa (93). Various surveys of serum collected from wild ungulates have failed to detect many positive specimens (81); however, a relatively high antibody prevalence was found in several species of wild ungulates, including African buffalo (*Syncerus caffer*), common warthog (*Phacochoerus africanus*), and giraffe (*Giraffa camelopardalis*), as well as domesticated camel (*Camelus dromedarius*), during and immediately following the East African epizootic of 2006–2007 (12). Bird and nonhuman primates are not believed to be involved in the life cycle of RVFV (81).

RVFV Transmission in Experimental Infection Studies

Given the recent movement of RVFV out of its endemic range and the potential for the virus to further expand its range, experimental infection studies have been conducted to identify possible vectors and hosts in emerging regions.

Vectors. Laboratory studies have identified many European, African, and North American mosquito species as potential competent vectors of RVFV (61, 69). The ability of mosquitoes to become infected with RVFV and transmit the virus varies widely. More extensive laboratory investigations are needed to quantify these differences, particularly in different geographic populations of the same species (107), and in other vectors that might transmit RVF in different geographical regions of sub-Saharan Africa.

The results of vector competence studies conducted in controlled laboratory settings are shown in **Tables 1** and **2**. Relatively few mosquito species from Africa and other places that may be affected by potential globalization of RVF have been evaluated (107, 113). Even fewer sand flies (e.g., *Phlebotomus* or *Lutzomyia* spp.), filth or biting flies (e.g., *Musca* or *Stomoxys* spp.), or biting midges (*Culicoides* spp.) have been evaluated (110). Mosquito species have demonstrated a wide range of vector competencies, and many species can transmit RVFV under laboratory settings. Some species of *Phlebotomus* and *Lutzomyia* sand flies also can transmit the virus in the laboratory (48, 114). Vertical transmission has not been demonstrated in the laboratory for suspected vectors, including mosquitoes, sand flies, or ticks; however, transstadial transmission of RVFV was demonstrated from virus-inoculated *Hyalomma truncatum* nymphs to adult ticks (70). (*H. truncatum* is a common tick on African cattle and a vector of Crimean-Congo hemorrhagic fever.) *Stomoxys calcitrans* stable flies, but not the house fly, *Musca domestica*, can mechanically transmit RVFV (47, 110).

Vertebrate hosts. RVFV circulates in the blood of experimentally infected domestic livestock for 1 to 4 days and has been found in the liver and spleen of sheep for up to several weeks (81, 106). The persistence of RVFV in sheep could contribute to the transmission cycle by permitting the virus to be introduced into new geographic areas through the movement of infected animals. Additional information on the historical background of RVFV (geographic groupings, role of land-use change, climate and weather links to RVF epizootic periods, social and economic impact, and movement and trade restrictions) and on the virus in general (strain variation, antigenic relationships, evolution, reassortment, and geography; host range; diagnostic procedures; disease association; and effects of virus on vectors) can be found in the **Supplemental Material**.

Supplemental Material

ENVIRONMENTAL SURVEILLANCE AND RVF DISEASE PREDICTION

Directly observing RVFV activity such as abortion in sheep and cattle, or monitoring herds for seroconversions, has historically guided how control measures such as use of vaccines or vector control in RVF-endemic areas are implemented, but these phenomena occur long after the environment has triggered RVF epizootics and do not provide an early-warning system. Alternatively, prolonged periods of heavy rainfall that can lead to flooding and emergence of RVFV-infected mosquitoes that initiate RVF epizootics also cause distinct patterns of vegetation development that can be monitored using satellite remote sensing technology (64). An early-warning system, the *Rift Valley Fever Monitor* (117), based on satellite vegetation data, has been developed for Africa, the Middle East, and the Arabian Peninsula; it is maintained by the USDA and NASA and is updated monthly (4–6, 62).

Climate and Weather Links to RVF Epizootic Periods

RVF outbreaks follow periods of anomalously high rainfall in eastern Africa (66). Past studies indicate that periods of above-normal rainfall in equatorial eastern Africa are associated with warm ENSO events (96). Cloudy and cool conditions during periods of above-normal rainfall further enhance the survival of and rapid increase in populations of RVFV mosquito vectors in dambo-type habitats over vast regional areas (66).

The history of ENSO can be reconstructed back to the late nineteenth century and may provide evidence of RVF epizootics in the early 1900s. Since 1950, approximately 90% of RVF outbreaks have occurred during El Niño events (62). ENSO has different hemispheric impacts on precipitation and vegetation growth such that during the warm, or El Niño, phase eastern Africa receives above-normal rainfall, whereas during the cool, or La Niña, phase southern Africa receives above-normal rainfall. Therefore, RVF outbreak patterns in these two major RVF epizootic regions are synchronized to ENSO-induced climate variability cycles (7).

Fine-Scale Environmental Indicators of RVF Epizootic Activity

The development of certain ENSO conditions warns that imminent environmental changes could favor a RVF epizootic. However, additional fine-scale environmental data are monitored to determine when and where there is a risk of RVF activity. The link between ENSO and RVF outbreaks is that precipitation is directly related to vegetation development in RVF-endemic areas; the presence of vegetation significantly enhances the survival of immature and adult RVFV-infected *Aedes* populations, thereby elevating the risk of RVF outbreaks (64, 104). Vegetation development has been

routinely monitored by satellite sensors since 1981 in the form of the normalized difference vegetation index (NDVI) (103), which is widely used in various environmental applications and is the basis of the *Rift Valley Fever Monitor*, which monitors areas where RVFV mosquito vectors can emerge.

NDVI: normalized difference vegetation index

PEAM: potential epizootic area mask/map

RVF Risk Mapping and Prediction

The *Rift Valley Fever Monitor* utilizes the NDVI to map the interaction of several variables, including rainfall, temperature, and soil moisture, that are important for the emergence, survival, and propagation of RVFV mosquito vectors and the potential risk of RVF (5). The system successfully retrospectively predicted where RVF outbreaks occurred between 1981 and 1998 (5) and subsequently predicted areas of recent RVF outbreaks in East Africa (2006–2007), Sudan (2007), and southern Africa (2008–2010) (4, 6). The *Rift Valley Fever Monitor* is an up-to-date and readily accessible resource for a diversity of stakeholders throughout RVF-endemic areas who can use the risk maps to initiate surveillance and mitigation activities weeks to months before outbreaks.

To derive and map areas at risk for RVF outbreaks, the *Rift Valley Fever Monitor* model analyzes two sets of real-world data. The first dataset is the potential epizootic area mask/map (PEAM) (green-shaded areas indicated in **Figure 2**). The PEAM combines (*a*) maps of epizootic/epidemic regions derived from areas of reported historical outbreaks with (*b*) maps of areas with high interannual variability of long-term historical NDVI and rainfall. The second dataset consists of temporal dynamics of mosquito vector populations in dambo habitats, as measured empirically (67, 68). The model is then run with inputs of current and ongoing NDVI anomalies to detect areas with persistent positive NDVI anomalies (greater than +0.1 NDVI units) using a three-month moving window. The system thus accounts for periods of sustained above-normal NDVI (and, by inference, rainfall) and considers the complex life cycle of mosquitoes that maintain and transmit RVFV to domestic livestock and humans. Regions with sustained positive NDVI anomalies that are located both in the PEAM and in areas with livestock and human populations are at the greatest risk for RVF transmission (red-shaded areas indicated in **Figure 2**). Regions with sustained positive NDVI anomalies in the PEAM but not near livestock or human populations are lower risk and are shaded in purple (not shown in **Figure 2**).

Figure 2 shows an example of an aggregate risk map with locations of human and animal cases for the period of RVF outbreak episodes spanning 2006–2011. The model performed the best in East Africa, with 65% of the human case locations mapped to be in at-risk areas, followed by Sudan with 50%, Madagascar with 23%, and southern Africa with 20% of the human cases (6). Factors that influence this varied performance include the current configuration of the model, which is based largely on the evolution of vector population findings from East Africa and thus might not be applicable to other regions. The weak performance of surveillance systems and the unpredictability of the livestock trade further compound this variability. In addition, human case data are not an optimum indicator of the spatial distribution of RVF cases, and the PEAM needs refinement in some areas. In the future, models must consider the unique factors in each region.

RVF Prediction with Mathematical Modeling

Several studies have used mathematical modeling techniques to simulate the epidemiology of RVF under various scenarios of vector population dynamics, virus transmission, different livestock populations, animal movement, and establishment in new regions (14, 40, 87, 89). These efforts may be viewed as learning tools because as currently designed they include many assumptions

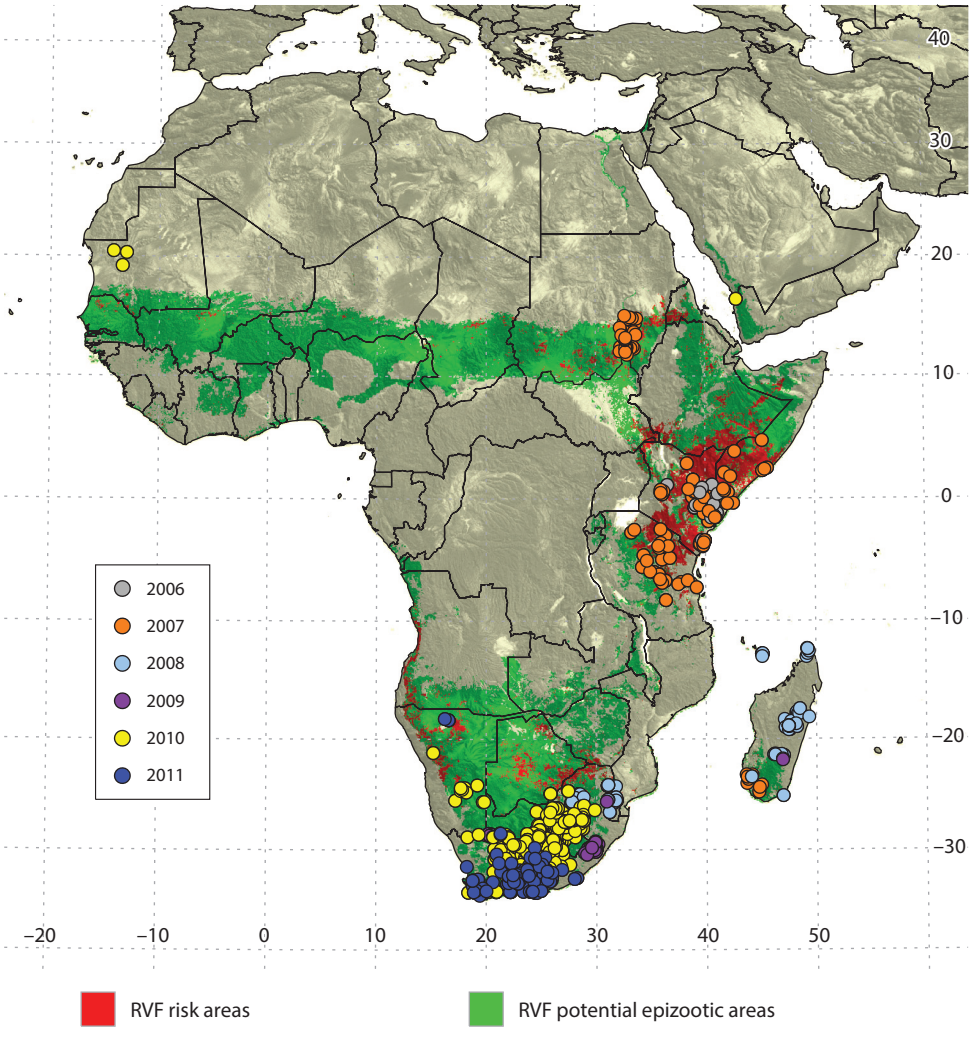

Figure 2

Composite 2006–2011 risk map produced by the *Rift Valley Fever Monitor* model based on extended heavy rainfall, ecological habitats associated with epizootics, and population densities of cattle, sheep, and goats. The potential epizootic/epidemic area mask (PEAM) is shown in green. Areas modeled to be at high risk of transmission of Rift Valley fever (RVF) in 2006–2011 are shown in red, including East Africa (September 2006–May 2007), Sudan (June–November 2007), and southern Africa, including Madagascar (October–May composite aggregated for each year from 2007 to 2011). Outbreak locations (colored circles, classified by year) are based on 2006–2011 human RVF case data from East Africa (Kenya, Somalia, Tanzania; 2006–2007), Sudan (2007), southern Africa (Madagascar, South Africa, Botswana, Namibia, South Africa; 2008–2011), and West Africa/Middle East (Yemen/Saudi Arabia; 2010).

not supported with real-world data or they lack a spatial component. Mathematical modeling can help derive factors, such as immunity decay, that can be used in spatially explicit models of risk. Additional information on RVF disease prediction (e.g., investigation of epizootics and epidemics) can be found in the **Supplemental Material**.

Supplemental Material

LEVERAGING THE *RIFT VALLEY FEVER MONITOR* FOR INTERVENTION

Contact with RVFV-infected mosquitoes may be reduced by limiting the development of key vector mosquito populations with, for example, habitat modification, larvicides, or residual or aerosol adulticides, or by protecting individual hosts with repellents or physical barriers. Individual protective measures for humans are challenging but attainable; however, it is extremely difficult to keep mosquitoes from livestock and impossible to prevent mosquitoes from contacting wild ungulate reservoir and amplifying hosts (6). Given the vast areas where mosquito populations develop and where people live in close contact with millions of free-ranging wild and domestic ungulate animals in RVFV-endemic areas, we face significant challenges to focus limited mosquito control and individual protection resources to effectively reduce RVFV transmission during outbreaks (6, 117).

One solution is to use the *Rift Valley Fever Monitor* to leverage key environmental signals to map the development of ecological conditions that underlie emergence of unusually large vector populations that are a main cause of enzootic outbreaks (5, 7, 62, 64, 117). In a given year, these critical ecological conditions are not uniform across the entire endemic range of RVFV transmission, as can be seen in **Figure 2**, where the high-risk areas for RVF transmission are not continuous across the entire known endemic range (PEAM). Thus, disease interventions such as vector (and virus) surveillance and control measures, deployment of medical and veterinary providers, and distribution of educational and informational materials may be targeted in space and time weeks to months before potential RVF epizootics (6).

For example, dambos falling within high-risk areas for mosquito emergence (i.e., grid cells colored red by the *Rift Valley Fever Monitor*; **Figure 2**) may be treated with aerially applied slow-release larvicides well before sustained flooding occurs to interrupt the natural RVFV epidemiological cycle. Livestock owners in these areas could be warned in time to move their herds and flocks to lower-risk areas. Local pastoralists such as the Somali in northeastern Kenya and the Maasai of northern Tanzania are adept at recognizing RVF in their livestock and often move animals away from areas occupied by RVFV vector mosquitoes to avoid infection (52).

During periods of elevated risk, public health officials can immediately respond by conducting vector surveillance activities in at-risk areas. A decision-support tool developed for the directors of Veterinary Services in the Horn of Africa provides clear, practical guidance for appropriate responses to various states of the RVF epizootic cycle (11). Utilizing the *Rift Valley Fever Monitor* (117) to monitor the risk of an RVF epizootic, the Decision Support document lists a sequence of events, relating to increasing risk, that include surveillance of animal movement and sentinel herds, vaccination of animals, and vector control. The procedures outlined in this document are currently the state of the art for RVF control.

Such surveillance activities, after an early warning of RVF originating from the Department of Defense–Armed Forces Health Surveillance Center, Global Emerging Infections Surveillance and Response (AFHSC/GEIS)-USDA-NASA RVF Risk Monitoring website (now hosted as the *Rift Valley Fever Monitor*; see 117), implemented by the Food and Agriculture Organization and the World Health Organization led to the first detection of RVF in December 2006, the start of the 2006–2007 epizootic/epidemic in the Horn of Africa (4, 6, 117). This early detection was successfully leveraged in high-risk areas of Kenya and Tanzania to conduct enhanced surveillance for RVFV in vectors, livestock, and humans, and to initiate public education, dissemination of vaccines, and animal movement restrictions (4). Additional information on intervention (e.g., use of vaccines) can be found in the **Supplemental Material**.

Supplemental Material

POTENTIAL OF GLOBALIZATION OF RVFV

Global vigilance and surveillance in nonendemic countries are becoming more important, especially because commercial aircraft can transport RVFV-infected humans during periods of disease activity. RVFV introductions into Egypt in 1977 and the Arabian Peninsula in 2000 followed epizootics in Sudan in 1976 and the Horn of Africa in 1998, respectively. The potential for such globalization of arboviruses has been demonstrated by West Nile and chikungunya viruses over the past 15 years (13, 88).

After an extended period with low levels of RVFV activity through the 1980s to mid-1990s, a series of large-scale RVF outbreaks throughout the endemic range, including East Africa (1997–1998, 2006–2007), Sudan (2007), Madagascar (2008), southern Africa (2008–2011), Mauritania (2010, 2012), and Saudi Arabia and Yemen (2000), severely affected the health and economy of tens of thousands of humans and hundreds of thousands of livestock. RVFV can move outside the endemic sub-Saharan region and into the Comoros Islands, Madagascar, Egypt, and the Arabian Peninsula. In addition to these regions, which are geographically adjacent to RVFV-endemic countries, in other more distant areas of the world the ecological infrastructure exists to allow RVFV to potentially thrive in vector, wildlife, livestock, and human hosts. The virus could be purposefully introduced into new areas as a tactic of bioterrorism, or moved accidentally via viremic mosquitoes (including infected mosquito eggs), humans, wildlife, or livestock along the extensive networks of global transportation, expanding the range of the virus to other continents. Humans, for instance, may develop RVFV viremia sufficient to infect naïve mosquitoes, and laboratory studies have demonstrated that numerous North American mosquito species of medical or veterinary importance can become infected with and transmit RVFV (107, 108, 111, 116) (**Table 2**). Prevention might best be exercised through vigilance and monitoring RVF activity in endemic settings (117) and increased vigilance for RVFV at high-risk locations in other continents that are connected to these endemic areas (61).

SUMMARY POINTS

1. RVF is a mosquito-borne zoonotic disease that causes serious disease in cattle, sheep, goats, and humans.
2. Prior to 1977, RVF occurred only in sub-Saharan Africa but has since expanded its range to Egypt, Madagascar, the Comoros Islands, and the Arabian Peninsula.
3. The potential globalization of RVFV is facilitated by the presence of susceptible domestic animal hosts and mosquitoes in many parts of the world.
4. *Aedes* spp. are the most important mosquitoes involved in vertical transmission of RVFV, and *Culex* and *Mansonia* spp. are the most important horizontal vectors of the virus.
5. The ecology of RVF outbreaks is closely linked to periods of anomalously heavy rainfall.
6. The RVF link to climate can be exploited to monitor potential areas at risk for RVF with earth-orbiting satellite remote sensing technology, e.g., the *Rift Valley Fever Monitor*.
7. Risk prediction is now possible by using a combination of monitoring global climate indicators such as ENSO and regional and local vegetation development, rainfall, and land surface temperatures.
8. Control is enhanced through a combination of RVF monitoring, targeted ground surveillance, animal vaccination, and vector control.

FUTURE ISSUES

1. It will be critical to develop a better understanding of the interepizootic maintenance of RVFV and to determine the relative contributions of mosquito vectors, wildlife, and livestock to this maintenance.
2. The pathogenicity of RVFV in wild mammals in endemic regions should be investigated, and the variation in susceptibility of humans and other mammals to serious RVF disease should be evaluated.
3. Population dynamics and vector competence for RVFV of putative mosquito and other vectors such as *Stomoxys*, *Culicoides*, *Simulium*, and *Hyalomma* spp. in nonendemic areas need to be studied in order to develop surveillance, response, and containment plans should RVFV appear in these areas. Similarly, pathogenicity of RVFV in wild mammals, especially ungulates, and livestock strains, in particular cattle, sheep, and goats, from nonendemic regions should be evaluated.

DISCLOSURE STATEMENT

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LITERATURE CITED

1. Abdelgadir DM, Bashab HMM, El Hadi Mohamed RA, Abulmaali SA. 2010. Risk factor analysis for outbreak of Rift Valley fever in Khartoum state of Sudan. *J. Entomol. Sci.* 45:239–51
2. Ackerman E. 1936. Dambos in Nordrhodesien. *Wiss. Veroffenlichunguen Mus. Landerkunde Leipzig* 4:147–57
3. Alexander RA. 1957. *Discussion on both Rift Valley fever* (*R.V.F.*) *and Wesselsbron virus disease* (*W.V.D.*). Presented at Proceedings of the 4th Annual Meeting of the Inter African Advisory Committee on Epizootic Disease, p. 29, May 10–11, Dakar, Senegal
4. Anyamba A, Chretien J-P, Small J, Tucker CJ, Formenty PB, et al. 2009. Prediction of a Rift Valley fever outbreak. *PNAS* 106:955–59
5. Anyamba A, Linthicum KJ, Mahoney R, Tucker CJ, Kelley PW. 2002. Mapping potential risk of Rift Valley fever outbreaks in African savannas using vegetation index time series data. *Photogramm. Eng. Remote Sens.* 68:137–45
6. Anyamba A, Linthicum KJ, Small J, Britch SC, Pak E, et al. 2010. Prediction, assessment of the Rift Valley fever activity in East and Southern Africa 2006–2008 and possible vector control strategies. *Am. J. Trop. Med. Hyg.* 83(S2):43–51
7. Anyamba A, Linthicum KJ, Small JL, Collins KM, Tucker CJ, et al. 2012. Climate teleconnections and recent patterns of human and animal disease outbreaks. *PLOS Negl. Trop. Dis.* 6:e1465
8. Ba Y, Sall AA, Diallo D, Mondo M, Girault L, et al. 2012. Re-emergence of Rift Valley fever virus in Barkedji (Senegal, West Africa) in 2002–2003: identification of new vectors and epidemiological implications. *J. Am. Mosq. Control Assoc.* 28:170–78
9. Balkhy HH, Memish ZA. 2003. Rift Valley fever: an uninvited zoonosis in the Arabian Peninsula. *Int. J. Antimicrob. Agents* 21:153–57
10. Bird BH, Ksiazek TG, Nichol ST, MacLachlan NJ. 2009. Rift Valley fever virus. *J. Am. Vet. Med. Assoc.* 234:883–93
11. Breiman RF, Consultative Group for RVF Decision Support. 2010. Decision-support tool for prevention and control of Rift Valley fever epizootics in the greater horn of Africa. *Am. J. Trop. Med. Hyg.* 83(S2):75–85

12. Britch SC, Binepal YS, Ruder MG, Kariithi HM, Linthicum KJ, et al. 2013. Rift Valley fever risk map model and seroprevalence in selected wild ungulates and camels from Kenya. *PLOS ONE* 8:e66626
13. Cassadou S, Boucau S, Petit-Sinturel M, Huc P, Leparc-Goffart I, Ledrans M. 2014. Emergence of chikungunya fever on the French side of Saint Martin Island, October to December 2013. *Euro Surveill.* 19(13):24721536
14. Chamond F, Cantrell RS, Cosner C, Hassan AN, Beier JC, Ruan S. 2014. A modeling approach to investigate epizootic outbreaks and enzootic maintenance of Rift Valley fever virus. *Bull. Math. Biol.* 76:2052–72
15. Clerc Y, Coulanges P. 1981. Rapport du laboratoire des arbovirus 1980. *Arch. Inst. Pasteur Madag.* 49:65–69
16. Dar O, McIntyre S, Hogarth S, Heymann D. 2013. Rift Valley fever and a new paradigm of research and development for zoonotic disease control. *Emerg. Infect. Dis.* 19:189–93
17. Daubney R, Hudson JR. 1932. Rift Valley fever. *Lancet* 219:611–12
18. Daubney R, Hudson JR. 1933. Rift Valley fever. *East Afr. Med. J.* 10:2–19
19. Daubney R, Hudson JR, Garnham PC. 1931. Enzootic hepatitis or Rift Valley fever. An undescribed virus disease of sheep, cattle and man from East Africa. *J. Pathol. Bacteriol.* 34:545–79
20. Davies FG. 2010. The historical and recent impact of Rift Valley fever in Africa. *Am. J. Trop. Med. Hyg.* 83(S2):73–74
21. Davies FG. 1975. Observations on the epidemiology of Rift Valley fever in Kenya. *J. Hyg.* 75:219–30
22. Davies FG, Highton RB. 1980. Possible vectors of Rift Valley fever in Kenya. *Trans. R. Soc. Trop. Med. Hyg.* 74:815–16
23. Davies FG, Linthicum KJ. 1986. The Sudan dioch (*Quelea quelea aethiopica*) and Rift Valley fever. *Trans. R. Soc. Trop. Med. Hyg.* 80:171–72
24. Davies FG, Linthicum KJ, James AD. 1985. Rainfall and epizootic Rift Valley fever. *Bull. World Health Organ.* 63:941–43
25. Diallo M, Nabeth P, Ba K, Sall AA, Ba Y, et al. 2005. Mosquito vectors of the 1998–1999 outbreak of Rift Valley fever and other arboviruses (Bagaza, Sanar, Wesselsbron and West Nile) in Mauritania and Senegal. *Med. Vet. Entomol.* 19:119–26
26. Dighe NS, Pattan SR, Bhawar SB, Gaware VM, Hole MB, et al. 2010. Rift Valley fever: a review. *J. Chem. Pharm. Res.* 2:228–39
27. Digoutte JP, Cordellier R, Robin Y, Pajot FX, Geoffroy B. 1974. Le virus zinga (AR B 1976), nouveau prototype d'arbovirus isole en Republique Centreafricaine. *Ann. Microbiol.* 125B:107–18
28. Digoutte JP, Peters CJ. 1989. General aspects of the 1987 Rift Valley fever epidemic in Mauritania. *Res. Virol.* 140:27–30
29. Dohm DJ, Rowton ED, Lawyer PG, O'Guinn M, Turell MJ. 2000. Laboratory transmission of Rift Valley fever virus by *Phlebotomus duboscqi*, *Phlebotomus papatasi*, *Phlebotomus sergenti*, and *Sergentomyia schwetzi* (Diptera: Psychodidae). *J. Med. Entomol.* 37:435–38
30. Easterday BC. 1961. *Experimental Rift Valley fever*. PhD Thesis, Univ. Wisconsin, Madison
31. Easterday BC. 1965. Rift Valley fever. *Adv. Vet. Sci.* 10:65–127
32. Easterday BC, Murphy LC, Bennett DG. 1962. Experimental Rift Valley fever in lambs and sheep. *Am. J. Vet. Res.* 23:1231–40
33. El Hadi Mohamed RA, Konozy EH, El Rayah E-A. 2013. Rift Valley fever virus (RVFV) dissemination inside mosquitoes and investigation of the influence of climate on mosquitoes abundance. *J. Entomol. Zool. Stud.* 1:1–9
34. Fagbami AH, Tomori O, Fabiyi A, Isoun TT. 1975. Experimental Rift Valley fever in West African dwarf sheep. *Res. Vet. Sci.* 18:334–35
35. Faran ME, Romoser WS, Routier RG, Bailey CL. 1988. The distribution of Rift Valley fever virus in the mosquito *Culex pipiens* as revealed by viral titration of dissected organs and tissues. *Am. J. Trop. Med. Hyg.* 39:206–13
36. Findlay GM, Howard EM. 1951. Notes on Rift Valley fever. *Arch. Gesamte Virusforsch.* 4:411–23
37. Flick R, Bouloy M. 2005. Rift Valley fever virus. *Curr. Mol. Med.* 5:827–34
38. Fontenille D, Traore-Lamizana M, Diallo M, Thonnon J, Digoutte JP, Zeller HG. 1998. New vectors of Rift Valley fever in West Africa. *Emerg. Infect. Dis.* 4:289–93

39. Gad AM, Hassan MM, el Said S, Moussa MI, Wood OL. 1987. Rift Valley fever virus transmission by different Egyptian mosquito species. *Trans. R. Soc. Trop. Med. Hyg.* 81:694–98
40. Gaff HD, Hartley DM, Leahy NP. 2007. An epidemiological model of Rift Valley fever. *Electron. J. Differ. Equ.* 115:1–12
41. Gargan TP 2nd, Clark GG, Dohm DJ, Turell MJ, Bailey CL. 1988. Vector potential of selected North American mosquito species for Rift Valley fever virus. *Am. J. Trop. Med. Hyg.* 38:440–46
42. Gargan TP II, Jupp PG, Novak RJ. 1988. Panveld oviposition sites of floodwater *Aedes* mosquitoes and attempts to detect transovarial transmission of Rift Valley fever virus in South Africa. *Med. Vet. Entomol.* 2:231–36
43. Gear J, de Meillon B, Le Roux AF, Kofsky R, Innes RR, et al. 1955. Rift Valley fever in South Africa; a study of the 1953 outbreak in the Orange Free State, with special reference to the vectors and possible reservoir hosts. *S. Afr. Med. J.* 29:514–18
44. Gerdes GH. 2004. Rift Valley fever. *Rev. Sci. Tech. Off. Int. Epiz.* 23:613–23
45. Hanafi HA, Fryauff DJ, Saad MD, Soliman AK, Mohareb EW, et al. 2011. Virus isolations and high population density implicate *Culex antennatus* (Becker) (Diptera: Culicidae) as a vector of Rift Valley fever virus during an outbreak in the Nile Delta of Egypt. *Acta Trop.* 119:119–24
46. Henderson BE, McCrae AW, Kirya BG, Ssenkubuge Y, Sempala SD. 1972. Arbovirus epizootics involving man, mosquitoes and vertebrates at Lunyo, Uganda 1968. *Ann. Trop. Med. Parasitol.* 66:343–55
47. Hoch AL, Gargan TP II, Bailey CL. 1985. Mechanical transmission of Rift Valley fever virus by hematophagous Diptera. *Am. J. Trop. Med. Hyg.* 34:188–93
48. Hoch AL, Turell MJ, Bailey CL. 1984. Replication of Rift Valley fever virus in the sand fly *Lutzomyia longipalpis*. *Am. J. Trop. Med. Hyg.* 33:295–99
49. Hoogstraal H, Meegan JM, Khalil GM, Adham FK. 1979. The Rift Valley fever epizootic in Egypt 1977–78. 2. Ecological and entomological studies. *Trans. R. Soc. Trop. Med. Hyg.* 73:624–29
50. Iranpour M, Turell MJ, Lindsay LR. 2011. Potential for Canadian mosquitoes to transmit Rift Valley fever virus. *J. Am. Mosq. Control Assoc.* 27:363–69
51. Jennings M, Platt GS, Bowen ET. 1982. The susceptibility of *Culicoides variipennis* coq. (Diptera: Ceratopogonidae) to laboratory infection with Rift Valley fever virus. *Trans. R. Soc. Trop. Med. Hyg.* 76:587–89
52. Jost CC, Nzietchueng S, Kihu S, Bett B, Njogu G, et al. 2010. Epidemiological assessment of the Rift Valley fever outbreak in Kenya and Tanzania in 2006 and 2007. *Am. J. Trop. Med. Hyg.* 83(S2):65–72
53. Jupp PG, Cornel AJ. 1988. Vector competence tests with Rift Valley fever virus and five South African species of mosquito. *J. Am. Mosq. Control Assoc.* 4:4–8
54. Jupp PG, Kemp A, Grobbelaar A, Lema P, Burt FJ, et al. 2002. The 2000 epidemic of Rift Valley fever in Saudi Arabia: mosquito vector studies. *Med. Vet. Entomol.* 16:245–52
55. Jupp PG, McIntosh BM, Thompson DL. 1983. Isolation of Rift Valley fever virus from *Aedes* (*Neomelaniconion*) *circumluteolus* and/or *luteolateralis* collected during an outbreak in cattle in the coastal region of Natal, South Africa. *S. Afr. J. Sci.* 79:377
56. Jupp PG, McIntosh BM, Thompson DL. 1984. Mechanical transmission of Rift Valley fever virus by mosquitos. *S. Afr. J. Sci.* 80:276
57. Kokernot RH, Heymann CS, Muspratt J, Wolstenholme B. 1957. Studies on arthropod-borne viruses of Tongaland. V. Isolation of Bunyamwera and Rift Valley fever viruses from mosquitoes. *S. Afr. J. Med. Sci.* 22:71–80
58. Lee VH. 1970. *University of Ibadan Arbovirus Research Project*, *1970 Annual Report*, pp. 29–30. Ibadan, Nigeria: Univ. Ibadan
59. Lee VH. 1979. Isolation of viruses from field populations of *Culicoides* (Diptera, Ceratopogonidae) in Nigeria. *J. Med. Entomol.* 16:76–79
60. Lernout T, Cardinale E, Jego M, Desprès P, Collet L, et al. 2013. Rift Valley fever in humans and animals in Mayotte, an endemic situation? *PLOS ONE* 8:e74192
61. Linthicum KJ, Anyamba A, Britch SC, Chretien J-P, Erickson RL, et al. 2007. A Rift Valley fever risk surveillance system for Africa using remotely sensed data: potential for use on other continents. *Vet. Ital.* 43:663–74
62. Linthicum KJ, Anyamba A, Tucker CJ, Kelley PW, Myers MF, Peters CJ. 1999. Climate and satellite indicators to forecast Rift Valley fever epidemics in Kenya. *Science* 285:397–400

63. Linthicum KJ, Bailey CL, Davies FG, Kairo A. 1985. Observations on the dispersal and survival of a population of *Aedes lineatopennis* (Ludlow) (Diptera, Culicidae) in Kenya. *Bull. Entomol. Res.* 75:661–70
64. Linthicum KJ, Bailey CL, Davies FG, Tucker CJ. 1987. Detection of Rift Valley fever viral activity in Kenya by satellite remote sensing imagery. *Science* 235:1656–59
65. Linthicum KJ, Bailey CL, Tucker CJ, Gordon SW, Logan TM, Peters CJ. 1994. Observations with NOAA and SPOT satellites on the effect of man-made alterations in the ecology of the Senegal River basin in Mauritania on Rift Valley fever virus transmission. *Sist. Terra* 3:44–47
66. Linthicum KJ, Bailey CL, Tucker CJ, Mitchell KD, Logan TM, et al. 1990. Application of polar-orbiting, meteorological satellite data to detect flooding of Rift Valley fever virus vector mosquito habitats in Kenya. *Med. Vet. Entomol.* 4:433–38
67. Linthicum KJ, Davies FG, Bailey CL, Kairo A. 1983. Mosquito species succession in a dambo in an East African forest. *Mosq. News* 43:464–70
68. Linthicum KJ, Davies FG, Bailey CL, Kairo A. 1984. Mosquito species encountered in a flooded grassland dambo in Kenya. *Mosq. News* 44:228–32
69. Linthicum KJ, Davies FG, Kairo A, Bailey CL. 1985. Rift Valley fever virus (family Bunyaviridae, genus *Phlebovirus*). Isolations from Diptera collected during an inter-epizootic period in Kenya. *J. Hyg.* 95:197–209
70. Linthicum KJ, Logan TM, Bailey CL, Dohm DJ, Moulton JR. 1989. Transstadial and horizontal transmission of Rift Valley fever virus in *Hyalomma truncatum*. *Am. J. Trop. Med. Hyg.* 41:491–96
71. Logan TM, Linthicum KJ, Davies FG, Binepal YS, Roberts CR. 1991. Isolation of Rift Valley fever virus from mosquitoes (Diptera, Culicidae) collected during an outbreak in domestic animals in Kenya. *J. Med. Entomol.* 28:293–95
72. Lutomiah J, Omondi D, Masiga D, Mutai C, Mireji PO, et al. 2014. Blood meal analysis and virus detection in blood-fed mosquitoes collected during the 2006–2007 Rift Valley fever outbreak in Kenya. *Vector Borne Zoonotic Dis.* 14:656–64
73. McIntosh BM. 1972. Rift Valley fever. 1. Vector studies in the field. *J. S. Afr. Vet. Med. Assoc.* 43:391–95
74. McIntosh BM, Dickinson DB, Dos Santos I. 1973. Rift Valley fever. 3. Viraemia in cattle and sheep. 4. The susceptibility of mice and hamsters in relation to transmission of virus by mosquitoes. *J. S. Afr. Vet. Assoc.* 44:167–69
75. McIntosh BM, Jupp PG. 1981. *Epidemiological aspects of Rift Valley fever in South Africa with reference to vectors*. Presented at Contributions to Epidemiology and Biostatistics: Rift Valley Fever. Proceedings of a Workshop on Rift Valley Fever, pp. 92–99, March 18–21, 1980, Herzlia, Israel
76. McIntosh BM, Jupp PG, Anderson D, Dickinson DB. 1973. Rift Valley fever. 2. Attempts to transmit virus with seven species of mosquito. *J. S. Afr. Vet. Assoc.* 44:57–60
77. McIntosh BM, Jupp PG, Dos Santos I. 1978. Rift Valley fever. *Arthropod-Borne Virus Inf. Exchange* 35:3–9
78. McIntosh BM, Jupp PG, Dos Santos I, Barnard BJ. 1980. Vector studies on Rift Valley fever virus in South Africa. *S. Afr. Med. J.* 58:127–32
79. McIntosh BM, Jupp PG, Dos Santos I, Rowe AC. 1983. Field and laboratory evidence implicating *Culex zombaensis* and *Aedes circumluteolus* as vectors of Rift Valley fever virus in coastal South Africa. *S. Afr. J. Sci.* 79:61–64
80. Meegan JM. 1979. The Rift Valley fever epizootic in Egypt 1977–78. 1. Description of the epizootic and virological studies. *Trans. R. Soc. Trop. Med. Hyg.* 73:618–23
81. Meegan JM, Bailey CL. 1989. Rift Valley fever. In *The Arboviruses*, ed. TP Monath, 39:51–76. Boca Raton, FL: CRC Press
82. Meegan JM, Khalil GM, Hoogstraal H, Adham FK. 1980. Experimental transmission and field isolation studies implicating *Culex pipiens* as a vector of Rift Valley fever virus in Egypt. *Am. J. Trop. Med. Hyg.* 29:1405–10
83. Miller BR, Godsey MS, Crabtree MB, Savage HM, Al-Mazrao Y, et al. 2002. Isolation and genetic characterization of Rift Valley fever virus from *Aedes vexans arabiensis*, Kingdom of Saudi Arabia. *Emerg. Infect. Dis.* 8:1492–94
84. Mims CA, Gillet JD. 1956. RVF virus transmission experiments. *East Afr. Virus Res. Inst. Annu. Rep.* 6:20–21

85. Morvan J, Rollin PE, Roux J. 1992. Rift Valley fever in Madagascar in 1991. Sero-epidemiological studies in cattle. *Rev. Elev. Med. Vet. Pays Trop.* 45:121–27
86. Moutailler S, Krida G, Schaffner F, Vazeille M, Failloux A-B. 2008. Potential vectors of Rift Valley fever virus in the Mediterranean region. *Vector Borne Zoonotic Dis.* 8:749–53
87. Mweya CN, Holst N, Mboera LEG, Kimera SI. 2014. Simulation modelling of population dynamics of mosquito vectors for Rift Valley fever virus in a disease epidemic setting. *PLOS ONE* 9:e108430
88. Nash D, Mostashari F, Fine A, Miller J, O'Leary D, et al. 1999. West Nile Outbreak Response Working Group. 2001. The outbreak of West Nile virus infection in the New York City area in 1999. *N. Engl. J. Med.* 344:1807–14
89. Niu T, Gaff HD, Papelis YE, Hartley DM. 2012. An epidemiological model of Rift Valley fever with spatial dynamics. *Comput. Math. Methods Med.* 2012:138757
90. Pepin M, Bouloy M, Bird BH, Kemp A, Paweska J. 2010. Rift Valley fever virus (Bunyaviridae: Phlebovirus): an update on pathogenesis, molecular epidemiology, vectors, diagnostics and prevention. *Vet. Res.* 41:61
91. Peters CJ, Linthicum KJ. 1994. Rift Valley fever. In *Handbook of Zoonoses*, ed. GW Beran, pp. 125–38. Boca Raton, FL: CRC Press. 2nd ed.
92. Peters CJ, Meegan JM. 1981. Rift Valley fever. In *CRC Handbook Series in Zoonoses. Section B*, Vol. I: *Viral Zoonoses*, ed. GW Beran, pp. 403–20. Boca Raton, FL: CRC Press
93. Pretorius A, Oelofsen MJ, Smith MS, van der Ryst E. 1997. Rift Valley fever virus: a seroepidemiologic study of small terrestrial vertebrates in South Africa. *Am. J. Trop. Med. Hyg.* 57:693–98
94. Ratovonjato J, Olive M-M, Tantely LM, Andrianaivolambo L, Tata E, et al. 2011. Detection, isolation, and genetic characterization of Rift Valley fever virus from *Anopheles* (*Anopheles*) *coustani*, *Anopheles* (*Anopheles*) *squamosus*, and *Culex* (*Culex*) *antennatus* of the Haute Matsiatra region, Madagascar. *Vector Borne Zoonotic Dis.* 11:753–59
95. Roger M, Girard S, Faharoudine A, Halifa M, Bouloy M, et al. 2011. Rift Valley fever in ruminants, Republic of Comoros, 2009. *Emerg. Infect. Dis.* 17:1319–20
96. Ropelewski CF, Halpert MS. 1987. Global and regional scale precipitation patterns associated with the El Niño/Southern Oscillation. *Mon. Weather Rev.* 115:1606–26
97. Sang R, Kioko E, Lutomiah J, Warigia M, Ochieng C, et al. 2010. Rift Valley fever virus epidemic in Kenya, 2006/2007: the entomologic investigations. *Am. J. Trop. Med. Hyg.* 83(S2):28–37
98. Seufi AM, Galal FH. 2010. Role of *Culex* and *Anopheles* mosquito species as potential vectors of Rift Valley fever virus in Sudan outbreak, 2007. *BMC Infect. Dis.* 10:65
99. Shimshony A, Barzilai R. 1983. Rift Valley fever. *Adv. Vet. Sci. Comp. Med.* 27:347–425
100. Smithburn KC, Haddow AJ, Gillett JD. 1948. Rift Valley fever. Isolation of the virus from wild mosquitoes. *Br. J. Exp. Pathol.* 29:107–21
101. Swanepoel R, Blackburn NK, Efstratiou S, Condy JB. 1978. Studies on Rift Valley fever in some African murids (Rodentia: Muridae). *J. Hyg.* 80:183–96
102. Swanepoel R, Coetzer JAW. 2004. Rift Valley fever. In *Infectious Diseases of Livestock*, ed. JA Coetzer, RC Tustin, pp. 1037–70. Cape Town, S. Afr.: Oxford Univ. Press
103. Tucker CJ. 1989. Comparing SMMR and AVHRR data for drought monitoring. *Int. J. Remote Sens.* 10:1663–72
104. Tucker CJ, Hielkema JU, Roffey J. 1985. The potential of satellite remote-sensing of ecological conditions for survey and forecasting desert locust activity. *Int. J. Remote Sens.* 6:127–38
105. Turell MJ, Bailey CL, Beaman JR. 1988. Vector competence of a Houston, Texas strain of *Aedes albopictus* for Rift Valley fever virus. *J. Am. Mosq. Control Assoc.* 4:94–96
106. Turell MJ, Bailey CL, Rossi CA. 1984. Increased mosquito feeding on Rift Valley fever virus-infected lambs. *Am. J. Trop. Med. Hyg.* 33:1232–38
107. Turell MJ, Britch SC, Aldridge RL, Kline DL, Boohene C, Linthicum KJ. 2013. Potential for mosquitoes (Diptera: Culicidae) from Florida to transmit Rift Valley fever virus. *J. Med. Entomol.* 50:1111–17
108. Turell MJ, Britch SC, Aldridge RL, Xue R-D, Smith ML, et al. 2015. Potential for *Psorophora columbiae* and *Psorophora ciliata* mosquitoes (Diptera: Culicidae) to transmit Rift Valley fever virus. *J. Med. Entomol.* 52:1111–16

109. Turell MJ, Byrd BD, Harrison BA. 2013. Potential for populations of *Aedes j. japonicus* to transmit Rift Valley fever virus in the USA. *J. Am. Mosq. Control Assoc.* 29:133–37
110. Turell MJ, Dohm DJ, Geden CJ, Hogsette JA, Linthicum KJ. 2010. Potential for stable flies and house flies (Diptera: Muscidae) to transmit Rift Valley fever virus. *J. Am. Mosq. Control Assoc.* 26:445–48
111. Turell MJ, Dohm DJ, Mores CN, Terracina L, Wallette DL Jr, et al. 2008. Potential for North American mosquitoes to transmit Rift Valley fever virus. *J. Am. Mosq. Control Assoc.* 24:502–7
112. Turell MJ, Kay BH. 1998. Susceptibility of selected strains of Australian mosquitoes (Diptera: Culicidae) to Rift Valley fever virus. *J. Med. Entomol.* 35:132–35
113. Turell MJ, Linthicum KJ, Patrican LA, Davies FG, Kairo A, Bailey CL. 2008. Vector competence of selected African mosquito (Diptera: Culicidae) species for Rift Valley fever virus. *J. Med. Entomol.* 45:102–8
114. Turell MJ, Perkins PV. 1990. Transmission of Rift Valley fever virus by the sand fly, *Phlebotomus duboscqi* (Diptera: Psychodidae). *Am. J. Trop. Med. Hyg.* 42:185–88
115. Turell MJ, Presley SM, Gad AM, Cope SE, Dohm DJ, et al. 1996. Vector competence of Egyptian mosquitoes for Rift Valley fever virus. *Am. J. Trop. Med. Hyg.* 54(2):136–39
116. Turell MJ, Wilson WC, Bennett KE. 2010. Potential for North American mosquitoes (Diptera: Culicidae) to transmit Rift Valley fever virus. *J. Med. Entomol.* 47:884–89
117. USDA-ARS. 2015. *Rift Valley Fever Monitor*. **http://www.ars.usda.gov/Business/Docs.htm?docid=23464**
118. Van Velden DJJ, Meyer JD, Olivier J. 1977. Rift Valley fever affecting humans in South Africa. A clinicopathological study. *S. Afr. Med. J.* 51:867–71
119. Weinbren MP, Mason PJ. 1957. Rift Valley fever in a wild field rat (*Arvicanthis abyssinicus*): a possible natural host. *S. Afr. Med. J.* 31:427–30
120. Weinbren MP, Williams MC, Haddow AJ. 1956. Isolation of an atypically reacting strain of Rift Valley fever virus from Lunyo, on the Entebbe Peninsula. *East African Virus Research Institute Annual Report*, pp. 21–22. Entebbe, Uganda: East African High Comm.
121. Weiss KE. 1957. Rift Valley fever - a review. *Bull. Epizoot. Dis. Afr.* 5:431–58
122. Williams MC, Woodall JP, Knight EK, Corbet PS, Haddow AJ. 1960. An outbreak of Rift Valley fever occurring near Entebbe. *East African Virus Research Institute*, *Annual Report*, *1959–1960*, pp. 24–28. Entebbe, Uganda: East African High Comm.
123. Woodall JP. 1964. The viruses isolated from arthropods at the East African Virus Research Institute in the 26 years ending December 1963. *Proc. E. Afr. Acad.* 2:141–46

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