Journal of the American Mosquito Control Association, 24(4):502–507, 2008

POTENTIAL FOR NORTH AMERICAN MOSQUITOES TO TRANSMIT RIFT VALLEY FEVER VIRUS[1]

MICHAEL J. TURELL,[2] DAVID J. DOHM,[2] CHRISTOPHER N. MORES,[2,7] LUCAS TERRACINA,[3] DENNIS L. WALLETTE, JR.,[4] LAWRENCE J. HRIBAR,[5] JAMES E. PECOR[6] AND JAMIE A. BLOW[2,8]

ABSTRACT. The rapid spread of West Nile viral activity across North America since its discovery in 1999 illustrates the potential for an exotic arbovirus to be introduced and widely established across North America. Rift Valley fever virus (RVFV) has been responsible for large outbreaks in Africa that have resulted in hundreds of thousands of human infections and major economic disruption due to loss of livestock and to trade restrictions. However, little is known about the potential for North American mosquitoes to transmit this virus should it be introduced into North America. Therefore, we evaluated selected mosquito species from the southeastern United States for their ability to serve as potential vectors for RVFV. Mosquitoes were fed on adult hamsters inoculated 1 day previously with RVFV. These mosquitoes were tested for infection and ability to transmit RVFV after incubation at 26°C for 7–21 days. None of the species tested (*Aedes taeniorhynchus, Ae. vexans, Culex erraticus, Cx. nigripalpus, Cx. quinquefasciatus,* and *Cx. salinarius*) were efficient vectors after they fed on hamsters with viremias ranging from $10^{4.1}$ to $10^{6.9}$ plaque-forming units (PFU)/ml. However, *Ae. taeniorhynchus, Ae. vexans,* and *Cx. erraticus* all developed disseminated infections after they fed on hamsters with viremias between $10^{8.5}$ and $10^{10.2}$ PFU/ml, and both *Ae. vexans* and *Cx. erraticus* transmitted RVFV by bite. These studies illustrate the need to identify the ability of individual mosquito species to transmit RVFV so that appropriate decisions can be made concerning the application of control measures during an outbreak.

KEY WORDS Rift Valley fever, vector, transmission, North America

INTRODUCTION

As illustrated by the introduction of West Nile virus into the United States in 1999 and its subsequent spread across North America, exotic arboviruses have the potential to be introduced and become established in North America and to cause significant disease and economic disruption. Of particular concern is Rift Valley fever virus (RVFV), which has been responsible for numerous outbreaks of severe disease in ruminants and humans in sub-Saharan Africa over the past 70 years (Meegan and Bailey 1988, Gerdes 2004). Although originally limited to sub-Saharan Africa, an outbreak in Egypt in 1977 caused an estimated 200,000 human cases as well as having devastating effects on the sheep and cattle industries (Laughlin et al. 1979, Meegan 1979). The detection of RVFV on the Arabian Peninsula (Jupp et al. 2002, Shoemaker et al. 2002, Balkhy and Memish 2003, Madani et al. 2003) has raised very real concerns regarding the agricultural and medical impact this zoonotic disease agent might have if it were to continue to spread (House et al. 1992).

Although Rift Valley fever (RVF) is predominately a problem in domestic ruminants, in which infection in pregnant animals usually results in abortion and infection of newborn animals is nearly always fatal, humans are also susceptible to infection (Easterday et al. 1962, Meegan and Bailey 1988). In humans, most infections result in an undifferentiated febrile disease and, rarely, encephalitis; however, about 1% of the infections result in hemorrhagic complications, which are often fatal. In addition, ocular sequellae can occur and cause retinal damage, including blindness (Siam et al. 1980, Al-Hazmi et al. 2005).

Rift Valley fever virus is a member of the genus *Phlebovirus,* in the family *Bunyaviridae,* and most viruses in this genus are associated with sand flies

[1] Research was conducted in compliance with the Animal Welfare Act and other federal statutes and regulations relating to animals and experiments involving animals and adheres to principles stated in the Guide for the Care and Use of Laboratory Animals, National Research Council, 1996. The facility where this research was conducted is fully accredited by the Association for Assessment and Accreditation of Laboratory Animal Care International. The views of the authors do not necessarily reflect the position of the Department of Defense or the Department of the Army.

[2] Virology Division, US Army Medical Research Institute of Infectious Diseases, 1425 Porter Street, Fort Detrick, MD 21702-5011.

[3] Department of Mosquito and Rodent Control, Calcasieu Parish Police Jury, Lake Charles, LA 70615.

[4] Tangipahoa Mosquito Abatement District, 15483 Club Deluxe Road, Hammond, LA 70403.

[5] Florida Keys Mosquito Control District, 503 107th Street, Marathon, FL 33050.

[6] Division of Entomology, Walter Reed Army Institute of Research, Silver Spring, MD 20907.

[7] Present address: Department of Pathobiological Sciences, School of Veterinary Medicine, Louisiana State University, Skip Bertman Drive, Baton Rouge, LA 70803.

[8] Present address: Armed Forces Pest Management Board, Forest Glen Annex, Walter Reed Army Medical Center, 6900 Georgia Avenue, NW, Washington, DC 20307-5001.

Report Documentation Page

Form Approved
OMB No. 0704-0188

1. REPORT DATE	2. REPORT TYPE	3. DATES COVERED
1 DEC 2008	**N/A**	**-**

4. TITLE AND SUBTITLE	5a. CONTRACT NUMBER
Potential for North American mosquitoes (Diptera:Culicidae) to transmit Rift Valley fever virus. J Mosq Control Asso 24:502-507	5b. GRANT NUMBER
	5c. PROGRAM ELEMENT NUMBER
6. AUTHOR(S)	5d. PROJECT NUMBER
Turell MJ Dohm DJ Mores CN Terracina L Wallette DL Jr Hribar LJ Pecor JE Blow JA	5e. TASK NUMBER
	5f. WORK UNIT NUMBER
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)	8. PERFORMING ORGANIZATION REPORT NUMBER
United States Army Medical Research Institute of Infectious Diseases, Fort Detrick, MD	**TR-08-044**
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)	10. SPONSOR/MONITOR'S ACRONYM(S)
	11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION/AVAILABILITY STATEMENT

Approved for public release, distribution unlimited

13. SUPPLEMENTARY NOTES

14. ABSTRACT

The rapid spread of West Nile viral activity across North America since its discovery in 1999 illustrates the potential for an exotic arbovirus to be introduced and widely established across North America. Rift Valley fever virus (RVFV) has been responsible for large outbreaks in Africa that have resulted in hundreds of thousands of human infections and major economic disruption. However, little is known about the potential for North American mosquitoes to transmit this virus should it be introduced into North America. Therefore, we evaluated selected mosquito species from the southeastern US for their ability to serve as potential vectors for RVFV. Mosquitoes were fed on adult hamsters inoculated 1 day previously with RVFV. These mosquitoes were tested for infection and ability to transmit RVFV after incubation at 26oC for 7-21 days. None of the species tested (Aedes taeniorhynchus, Ae. vexans, Culex erraticus, Cx. nigripalpus, Cx. quinquefasciatus, and Cx. salinarius) were efficient vectors after they fed on hamsters with viremias <106.9 plaque-forming units (PFU)/ml. However, Ae. taeniorhynchus, Ae. vexans, and Cx. erraticus all developed disseminated infections after they fed on hamsters with viremias between 108.5 and 1010.2 PFU/ml, and both Ae. vexans, and Cx. erraticus transmitted RVFV by bite. These studies illustrate the need to identify the ability of individual mosquito species to transmit RVFV so that correct decisions can be made concerning the application of appropriate control measures during an outbreak.

15. SUBJECT TERMS

Rift Valley fever virus, mosquito vector, transmission, North American mosquitoes

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			
unclassified	**unclassified**	**unclassified**	**SAR**	**7**	

Table 1. Source and colonization history of mosquitoes captured at dry-ice–baited miniature light traps from April to June 2004 and evaluated for their vector competence for Rift Valley fever virus.

Species	Location	Generation[1]
Aedes vexans	Indian River County, FL	P_0/F_1
Ae. taeniorhynchus	No Name Key, FL	P_0
Culex nigripalpus	Sarasota and Indian River, FL	P_0/F_1
Cx. (Melanoconion) erraticus	Sarasota, FL, Lake Charles, LA	P_0
Cx. salinarius	Lake Charles, LA	P_0
Cx. quinquefasciatus	Sarasota, FL, Lake Charles, LA	P_0/F_1

[1] P_0, field-collected mosquitoes; F_1, first generation progeny of field-collected mosquitoes.

in nature. Although laboratory studies indicate that various African sand flies can transmit RVFV after feeding on viremic hamsters (Turell and Perkins 1990, Dohm et al. 2000) and that South American sand flies were able to transmit RVFV after intrathoracic inoculation (Hoch et al. 1984), this virus has been associated almost exclusively with mosquitoes in nature, with the virus isolated from at least 40 species of mosquitoes in 8 genera (Meegan and Bailey 1988, Fontenille et al. 1998). Because methods of control vary for different mosquito species, it is necessary to identify which species are competent vectors and might be involved in the natural transmission cycle so that the appropriate control measures can be employed. Laboratory studies indicate that numerous species of mosquitoes are susceptible to oral infection and are able to transmit RVFV by bite (McIntosh et al. 1973b, 1980, 1983; Gargan et al. 1988, Meegan and Bailey 1988, Turell et al. 1996). However, only a limited number of mosquito species from North America have been evaluated for their potential to transmit RVFV (Gargan et al. 1988).

To determine which mosquito species might serve as potential vectors should RVFV be introduced into North America, we captured live mosquitoes in Florida and Louisiana and transported them to the United States Army Medical Research Institute of Infectious Diseases (USAMRIID), where they were evaluated for their potential to serve as natural vectors of RVFV. We selected this region as a starting point for evaluating North American mosquito species because of available mosquitoes. Rift Valley fever virus is a select agent and a Biological Safety Level (BSL)-3 agriculture facility with vaccination or a BSL-4 facility is required to work with it.

MATERIALS AND METHODS

Mosquitoes

Mosquitoes were captured in dry-ice–baited Centers for Disease Control miniature light traps (John W. Hock Co., Gainesville, FL) in Indian River County, Sarasota, and No Name Key, FL, and Lake Charles, LA, from late April through early June 2004 (Table 1). These mosquitoes were placed in screen-topped 3.8-liter cardboard containers, which were individually sealed in plastic bags. The sealed bags were added to a cardboard box before being placed in a shipping container for transport to USAMRIID. Upon arrival at USAMRIID, the mosquitoes were provided apple slices and placed in an incubator maintained at 26°C with a photoperiod of 16:8 (light:dark) h until tested for their susceptibility to RVFV.

Viruses and virus assay

The ZH501 strain of RVFV, isolated in 1977 from the blood of a 10-year-old Egyptian girl who had a fatal RVFV infection (Meagan 1979), was used throughout this study. This strain was passed twice in fetal rhesus monkey lung cells and once in Vero (African green monkey kidney) cells before use in this study.

Mosquito specimens were triturated in 1 ml of diluent (10% heat-inactivated fetal bovine serum in Medium 199 with Earle's salts [Invitrogen, Inc., Carlsbad, CA] and antibiotics) and then frozen at −70°C until tested for infectious virus by a plaque assay on Vero cell monolayers. Serial 10-fold dilutions of each specimen were tested on 6- or 12-well plates as described by Gargan et al. (1983). Viral titers were expressed as $\log_{10}$ plaque-forming units (PFU) per specimen.

Determination of vector competence

Adult female Syrian hamsters (Harlan Sprague Dawley, Indianapolis, IN) were inoculated intraperitoneally with 0.2 ml of a suspension containing about 10^4 PFU of RVFV to provide a source of viremic blood. These hamsters were anesthetized with a ketamine, xylazine, and acepromazine suspension 1 day after inoculation and placed individually (i.e., 1 per cage) on top of cages each containing 50–100 mosquitoes that had been deprived of a sucrose source for about 24 h. Immediately after mosquito feeding, a blood sample was collected from the anesthetized hamsters by cardiac puncture and the hamsters were then euthanized by CO_2 exposure. The blood suspensions (0.2 ml of blood added to 1.8 ml of diluent) were frozen at −70°C until assayed on Vero cell monolayers (as described above) to

determine viremias at the time of mosquito feeding. After exposure to the viremic hamsters, nonengorged mosquitoes were removed and destroyed by placing them in a freezer at −20°C. Engorged mosquitoes were provided apple slices, or a 10% sucrose solution on a gauze pledget as a carbohydrate source, and held at 26°C with a photoperiod of 16:8 (light:dark) h until tested for infection, dissemination, and transmission. Approximately 1 wk after the infectious blood meal, moist toweling or a water dish was added to each cage to stimulate oviposition. Eggs obtained from *Aedes vexans* (Meigen), *Culex nigripalpus* Theobald, and *Culex quinquefasciatus* Say were hatched and larvae reared to provide an F_1 generation that was also tested for their susceptibility to RVFV as described above.

To determine if the mosquitoes could transmit virus by bite, mosquitoes were allowed to feed on susceptible hamsters either individually or in small groups of 2–5 mosquitoes each. We considered death (or euthanasia when moribund) of these hamsters to indicate viral transmission, because RVFV infection consistently is fatal to hamsters. Nearly all RVFV-infected hamsters die or become moribund 1–3 days after virus exposure (M. Turell, unpublished data). Presence of virus was verified by isolating virus from brain tissue from a subset of the dead or euthanized hamsters (data not shown). Immediately after each transmission trial, mosquitoes were killed by freezing at −20°C for 5 min and identified to species; their feeding status was confirmed, and their legs and bodies triturated separately in 1 ml of diluent. These suspensions were then frozen at −70°C until assayed for virus.

The extent of viral infection in mosquitoes was determined by assaying a mosquito's body separately from its legs. If virus was detected in its body, but not its legs, the mosquito was considered to have a nondisseminated infection limited to its midgut. In contrast, if virus was detected in both the body and leg suspensions, the mosquito was considered to have a disseminated infection (Turell et al. 1984). Because some of the mosquitoes were tested for transmission in small pools, it was not always possible to determine which mosquito (or mosquitoes) in a pool actually transmitted virus by bite. Therefore, if more than one mosquito with a disseminated infection fed in a pool (only occurred 3 times in this study), data from that pool were not used to calculate the transmission rate, regardless of hamster survival.

The infection rate was the percentage of orally exposed mosquitoes that contained virus. The dissemination rate was the percentage of orally exposed mosquitoes (regardless of their infection status) that contained virus in their legs, and the transmission rate was the percentage of orally exposed mosquitoes that refed (regardless of their infection status) that transmitted virus by bite. We used the extended Wald method of calculating 95% confidence intervals (Agresti and Coull 1998).

RESULTS

Hamster viremias

Viremias in the 13 hamsters used to expose mosquitoes to RVFV ranged from $10^{4.1}$ to $10^{10.2}$ PFU/ml of virus in the blood ($10^{2.6}$ to $10^{7.7}$ PFU of virus ingested per mosquito, respectively). Because these viremias represent low, moderate, and high natural viremia levels, we arbitrarily grouped the mosquitoes into those exposed to low ($10^{4.1–4.9}$ PFU/ml), moderate ($10^{6.5–6.9}$ PFU/ml), high ($10^{8.5–8.6}$ PFU/ml), or very high ($10^{10.1–10.2}$ PFU/ml) viremias.

Susceptibility to infection

For those species in which both P_0 and F_1 mosquitoes were tested, infection and dissemination rates were similar in both generations (data not shown). Therefore data for the 2 generations were combined for analysis. When exposed to viremias ranging from $10^{4.1}$ to $10^{6.9}$ PFU/ml, infection rates were low in all species tested except *Aedes taeniorhynchus* (Wiedemann) (43%; Table 2). However, when fed on hamsters with viremias $\geq 10^{8.5}$ PFU/ml, all species tested became infected, with *Ae. vexans, Culex erraticus* (Dyar and Knab), and *Culex salinarius* Coq. being highly susceptible with infection rates >75% and *Cx. quinquefasciatus* being only moderately susceptible with an infection rate of 26% (Table 2).

Viral dissemination

As with infection, viral dissemination rates were low when mosquitoes were exposed to a viremia $\leq 10^{6.9}$ PFU/ml, with only a single *Ae. vexans* and *Cx. nigripalpus* and 2 *Ae. taeniorhynchus* having virus detected in their legs (Table 2). However, with the exception of *Cx. quinquefasciatus*, virus was readily detected in the legs of mosquitoes that fed on hamsters with viremias $\geq 10^{8.5}$ PFU/ml.

Viral transmission

Aedes vexans and *Cx. erraticus* transmitted RVFV by bite to susceptible hamsters (Table 3). However, because so few of the other species developed a disseminated infection and subsequently fed on a hamster, we have few data about potential salivary gland barriers (Kramer et al. 1981) in these species.

Table 2. Infection and dissemination rates for mosquitoes orally exposed to Rift Valley fever virus.

Species	Days of extrinsic incubation[1]											
	7			14–17			≥19			All days combined		
	N	I.R.[2]	D.R.[3]	*N*	I.R.	D.R.	*N*	I.R.	D.R.	*N*	I.R.	D.R.
Infectious dose = $10^{4.1-4.6}$ PFU/ml												
Aedes vexans				10	10	0	2	50	50	12	17 (3.5–46.0)	8 (0.1–37.5)
Culex quinquefasciatus							40	3	0	40	3 (0.1–14.0)	0 (0.0–10.4)
Cx. nigripalpus				10	0	0	72	4	0	82	4 (0.1–10.7)	0 (0.0–5.4)
Cx. salinarius	10	20	0	3	0	0	18	0	0	31	6 (0.8–21.8)	0 (0.0–13.1)
Infectious dose = $10^{6.5-6.9}$ PFU/ml												
Cx. (Melanoconion) erraticus				19	5	0	18	11	0	37	8 (2.1–22.0)	0 (0.0–11.2)
Cx. salinarius				10	0	0	7	0	0	17	0 (0.0–21.6)	0 (0.0–21.6)
Cx. nigripalpus				10	20	10	10	10	0	20	15 (4.4–36.9)	5 (0.1–25.4)
Ae. taeniorhynchus	10	50	10	4	25	25				14	43 (21.3–67.5)	14 (2.8–41.2)
Infectious dose = $10^{8.5-8.6}$ PFU/ml												
Ae. vexans				10	80	30				10	80 (47.9–95.4)	30 (10.3–60.8)
Cx. quinquefasciatus				73	26	0				73	26 (17.3–37.2)	0 (0.0–6.0)
Infectious dose = $10^{10.1-10.2}$ PFU/ml												
Ae. vexans				15	93	73	17	100	82	32	97 (82.9–99.9)	78 (61.0–91.6)
Cx. (Mel.) erraticus				14	79	64				14	79 (51.7–93.2)	64 (38.6–83.8)
Cx. salinarius				8	88	38				8	88 (50.8–99.9)	38 (13.5–69.6)

[1] *N*, number tested; I.R., infection rate; D.R., dissemination rate.
[2] I.R. = percentage of mosquitoes containing virus in their bodies.
[3] D.R. = percentage of mosquitoes, regardless of their infection status, containing virus in their legs.

DISCUSSION

Despite both the medical and economic risk that RVFV poses to North America, there have been few studies on the potential for North American mosquitoes to transmit RVFV (Gargan et al. 1988; Turell et al. 1985, 1988). This is the first study to evaluate field-collected mosquitoes from the southeastern United States for their ability to transmit this virus. Both *Ae. vexans* and *Cx. erraticus* readily developed disseminated viral infections and transmitted RVFV by bite after oral exposure to relatively high viremias ($\geq 10^{8.5}$/ml of blood). In addition, although *Ae. taeniorhynchus* was not tested at the higher viremia levels, it was the most susceptible species tested when fed on hamsters with viremias ranging from $10^{6.5}$ to $10^{6.9}$ PFU/ml, and based on studies with colonized *Ae. taeniorhynchus*, this species is a relatively efficient vector of RVFV (Turell et al. 1985). Although the only *Ae. taeniorhynchus* with a disseminated infection that fed on a susceptible hamster in the present study did not transmit virus by bite, previous studies with both field-

Table 3. Transmission of Rift Valley fever virus by mosquitoes captured in the southeastern United States that had been exposed to virus by feeding on viremic hamsters.

Species	Viremia	Transmission Rate[1]	Transmission (D) rate[2]
Aedes taeniorhynchus	$10^{6.5-6.9}$	0/4 (0%)	0/1 (0%)
Ae. vexans	$10^{8.5-8.6}$	1/4 (25%)	1/1 (100%)
Ae. vexans	$10^{10.1-10.2}$	3/13 (23%)	3/9 (33%)
Culex (Melanoconion) erraticus	$10^{6.5-6.9}$	0/11 (0%)	NT[3]
Cx. (Mel.) erraticus	$10^{10.1-10.2}$	2/6 (33%)	2/4 (50%)
Cx. nigripalpus	$10^{4.1-4.9}$	0/5 (0%)	0/1 (0%)
Cx. quinquefasciatus	$10^{4.1-4.9}$	0/29 (0%)	NT
Cx. quinquefasciatus	$10^{8.5-8.6}$	0/13 (0%)	NT
Cx. salinarius	$10^{4.1-4.9}$	0/7 (0%)	NT
Cx. salinarius	$10^{6.5-6.9}$	0/13 (0%)	NT
Cx. salinarius	$10^{10.1-10.2}$	0/2 (0%)	NT

[1] Number of mosquitoes transmitting virus/number of virus-exposed mosquitoes that refed (percentage transmitting).
[2] Number of mosquitoes transmitting virus/number of mosquitoes with a disseminated infection that refed (percentage transmitting).
[3] NT, not tested.

collected and colonized *Ae. taeniorhynchus* indicate that this species has a moderate salivary gland barrier and about 50% of this species with a disseminated infection would transmit RVFV by bite (Turell et al. 1985, Turell and Bailey 1987, Gargan et al. 1988). The infection and dissemination rates observed for *Ae. taeniorhynchus, Ae. vexans,* and *Cx. erraticus* are consistent with those reported for African vectors of RVFV (Jupp and Cornel 1988, Turell et al. 1996, 2007, 2008). Therefore, these species should be considered as potential vectors of RVFV should this virus be introduced into areas where these species are found.

The *Culex* (*Culex*) species tested were relatively incompetent vectors of RVFV, with only a single *Cx. nigripalpus* that fed on a hamster with a viremia of $10^{6.9}$ PFU/ml and none of 73 *Cx. quinquefasciatus* that fed on a hamster with a viremia of $10^{8.5}$ PFU/ml developing a disseminated infection. In contrast, several African members of this subgenus are relatively efficient vectors of RVFV, including *Culex pipiens* L., *Culex zombaensis* Theobald, *Culex perexiguus* Theobald, *Culex antennatus* (Becker), *Culex tritaeniorhynchus* Giles, and *Culex poicilipes* (Theobald) (Gad et al. 1987, 1989; Jupp and Cornel 1988, Jupp et al. 2002, Meegan et al. 1980, Turell et al. 1996, 2007), with reported infection rates ranging from 60% to 95%. The apparent inability of *Cx. quinquefasciatus* to transmit RVFV in this study is similar to the results of a recent study in which *Cx. quinquefasciatus* captured in Kenya failed to transmit virus by bite despite feeding on hamsters with viremias ranging from $10^{9.7}$ to $10^{10.3}$ PFU/ml (Turell et al. 2007). This is surprising because of the close relationship between this species and *Cx. pipiens,* the incriminated vector during the outbreak in Egypt in 1977–79 (Meegan et al. 1980). Is the poor vector competence of North American *Cx. quinquefasciatus* due to a difference in vector competence between *Cx. pipiens* and *Cx. quinquefasciatus*, or is it a difference between North African and North American members of these species? This illustrates the need to identify which mosquito species found to be infected in nature are actually able to transmit that virus by bite. Once a species has been confirmed as a vector, appropriate control measures can be implemented to reduce the number of those mosquitoes, especially during an outbreak.

Although most of the *Culex* (*Culex*) species tested in this study were inefficient vectors of RVFV, *Cx. salinarius* was susceptible when fed on a hamster with a viremia of about $10^{10.1}$ PFU/ml, and its viral infection and dissemination rates were similar to those reported by Gargan et al. (1988) for this species. We were unable to determine if *Cx. salinarius* had a salivary gland barrier because none of the *Cx. salinarius* with a disseminated infection took a second blood meal. However, nearly half of those with a disseminated infection transmitted RVFV by bite when fed on a susceptible hamster in an earlier study (Gargan et al. 1988). The only species of *Culex* (*Melanoconion*) tested, *Cx.* (*Mel.*) *erraticus,* was a moderately efficient vector, with vector competence similar to that of *Ae. vexans*. This is the first member of this subgenus to be evaluated for its ability to transmit RVFV, and its relative efficiency as a vector indicates that other members of this subgenus should be evaluated.

The viremias used in this study, $10^{4.1–10.2}$ PFU/ml, are consistent with viremias determined for natural infections with RVFV, where viremias in lambs and calves were as high as $10^{10.2}$ and $10^{9.2}$ mouse intracranial 50% lethal dose, respectively (McIntosh et al. 1973a). Therefore, the results obtained in our study should apply to the various mosquito species tested, should they feed on RVFV-infected cattle or sheep in a natural outbreak of RVF. Because this study focused on mosquitoes from the southeastern United States and mosquito populations vary in their ability to transmit viruses, the results of the present study may not apply to populations from other regions in North America. Additional studies are required to evaluate other potential vectors of RVFV in North America and to determine the role of other factors (e.g., environmental temperature) on the transmission of this pathogen.

ACKNOWLEDGMENTS

We thank Jill Hightower, Biologist, Calcasieu Parish Mosquito and Rodent Control, and Lee Ann Morgan, Entomologist, Tangipahoa Mosquito Abatement District, for their assistance in capturing the mosquitoes; J. Williams for caring for the hamsters; and K. Kenyon and J. Kondig for their critical reading of the manuscript. We also thank G. Korch, who was instrumental in initiating these projects.

This work was supported by a grant (04-0-DH-009) from the National Biodefense Analysis and Countermeasures Center.

REFERENCES CITED

Agresti A, Coull BA. 1998. Approximate is better than "exact" for interval estimation of binomial proportions. *Am Statistician* 52:119–126.

Al-Hazmi A, Al-Rajhi AA, Abboud EB, Ayoola EA, Al-Hazmi M, Saadi R, Ahmed N. 2005. Ocular complications of Rift Valley fever outbreak in Saudi Arabia. *Ophthalmology* 112:313–318.

Balkhy HH, Memish ZA. 2003. Rift Valley fever: an uninvited zoonosis in the Arabian Peninsula. *Int J Antimicrob Agents* 21:153–157.

Dohm DJ, Rowton ED, Lawyer PG, O'Guinn M, Turell MJ. 2000. Laboratory transmission of Rift Valley fever virus by *Phlebotomus duboscqi, Phlebo-*

tomus papatasi, Phlebotomus sergenti, and *Sergentomyia schwetzi* (Diptera: Psychodidae). *J Med Entomol* 37:435–438.

Easterday BC, Murphy LC, Bennett DG. 1962. Experimental Rift Valley fever in lambs and sheep. *Am J Vet Res* 23:1154–1163.

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* 4:289–293.

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* 81:694–698.

Gad AM, Hassan AN, Merdan AI. 1989. Transmission of Rift Valley fever virus by different geographic strains of *Culex pipiens* in Egypt. *J Egypt Public Health Assoc* 64:363–379.

Gargan TP II, Bailey CL, Higbee GA, Gad A, El Said S. 1983. The effect of laboratory colonization on the vector pathogen interactions of Egyptian *Culex pipiens* and Rift Valley fever virus. *Am J Trop Med Hyg* 32:1154–1163.

Gargan TP II, 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* 38:440–446.

Gerdes GH. 2004. Rift Valley fever. *Rev Sci Tech Off Int Epizoot* 23:613–623.

Hoch AL, Turell MJ, Bailey CL. 1984. Replication of Rift Valley fever virus in the sand fly *Lutzomyia longipalpis. Am J Trop Med Hyg* 33:295–299.

House JA, Turell MJ, Mebus CA. 1992. Rift Valley fever: present status and risk to the Western Hemisphere. *Ann N Y Acad Sci* 653:233–242.

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.

Jupp PG, Kemp A, Grobbelaar A, Lema P, Burt FJ, Alahmed AM, Al Mujalli D, Al Khamees M, Swanepoel R. 2002. The 2000 epidemic of Rift Valley fever in Saudi Arabia: mosquito vector studies. *Med Vet Entomol* 16:245–252.

Kramer LD, Hardy JL, Presser SB, Houk EJ. 1981. Dissemination barriers for western equine encephalomyelitis virus in *Culex tarsalis* infected after ingestion of low viral doses. *Am J Trop Med Hyg* 30:190–197.

Laughlin LW, Meegan JM, Strausbaugh LJ, Morens DM, Watten RH. 1979. Epidemic Rift Valley fever in Egypt: observations on the spectrum of human illness. *Trans R Soc Trop Med Hyg* 73:630–633.

Madani TA, Al-Mazrou YY, Al-Jeffri MH, Mishkhas AA, Al-Rabeah AM, Turkistani AM, Al-Sayed MO, Abodahish AA, Khan AS, Ksiazek TG, Shobokshi O. 2003. Rift Valley fever epidemic in Saudi Arabia: epidemiological, clinical, and laboratory characteristics. *Clin Infect Dis* 37:1084–1092.

McIntosh BM, Dickinson DB, Dos Santos I. 1973a. Rift Valley fever. 3. Viremia in cattle and sheep. 4. The susceptibility of mice and hamsters in relation to transmission of virus by mosquitoes. *J S Afr Vet Med Assoc* 44:167–169.

McIntosh BM, Jupp PG, Anderson D, Dickinson DB. 1973b. Rift Valley fever. 2. Attempts to transmit virus with seven species of mosquito. *J S Afr Vet Med Assoc* 44:57–60.

McIntosh BM, Jupp PG, Dos Santos I, Barnard BJH. 1980. Vector studies on Rift Valley fever in South Africa. *S Afr Med J* 58:127–132.

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.

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* 73:618–623.

Meegan JM, Bailey CL. 1988. Rift Valley fever. In: Monath TP, ed. *The arboviruses: epidemiology and ecology.* Volume 4. Boca Raton, FL: CRC. p 61–76.

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* 29:1405–1410.

Shoemaker T, Boulianne C, Vincent MJ, Pezzanite L, Al-Qahtani MM, Al-Mazrou Y, Khan AS, Rollin PE, Swanepoel R, Ksiazek TG, Nichol ST. 2002. Genetic analysis of viruses associated with emergence of Rift Valley fever in Saudi Arabia and Yemen, 2000–01. *Emerg Infect Dis* 8:1415–1420.

Siam AL, Meegan JM, Gharbawi KF. 1980. Rift Valley fever ocular manifestations: observations during the 1977 epidemic in Egypt. *Br J Ophthalmol* 64:366–374.

Turell MJ, Bailey CL. 1987. Transmission studies in mosquitoes (Diptera: Culicidae) with disseminated Rift Valley fever virus infections. *J Med Entomol* 24:11–18.

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 Cont Assoc* 4:94–96.

Turell MJ, Gargan TP II, Bailey CL. 1984. Replication and dissemination of Rift Valley fever virus in *Culex pipiens. Am J Trop Med Hyg* 33:176–181.

Turell MJ, Lee JS, Richardson JH, Sang RC, Kioko EN, Agawo MO, Pecor J, O'Guinn ML. 2007. Vector competence of Kenyan *Culex zombaensis* and *Culex quinquefasciatus* mosquitoes for Rift Valley fever virus. *J Am Mosq Control Assoc* 23:378–382.

Turell MJ, Linthicum KJ, Patrican LA, Glyn Davies F, Kairo A, Bailey CL. 2008. Vector competence of selected African mosquito (Diptera: Culicidae) species for Rift Valley fever virus. *J Med Entomol* 45:102–108.

Turell MJ, Perkins PV. 1990. Transmission of Rift Valley fever virus by the sand fly, *Phlebotomus duboscqi* (Diptera: Psychodidae). *Am J Trop Med Hyg* 42:185–188.

Turell MJ, Presley SM, Gad AM, Cope SE, Dohm DJ, Morrill JC, Arthur RR. 1996. Vector competence of Egyptian mosquitoes for Rift Valley fever virus. *Am J Trop Med Hyg* 54:136–139.

Turell MJ, Rossi CA, Bailey CL. 1985. Effect of extrinsic incubation temperature on the ability of *Aedes taeniorhynchus* and *Culex pipiens* to transmit Rift Valley fever virus. *Am J Trop Med Hyg* 34:1211–1218.