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ENZOOTIC ACTIVITY OF RIFT VALLEY FEVER VIRUS IN SENEGAL

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Abstract. In two areas of Senegal where previous evidence of Rift Valley fever (RVF) virus circulation was detected, Barkedji in the Sahelian bioclimatic zone and Kedougou in the Sudano-Guinean zone, a longitudinal study of the enzootic maintenance of RVF virus was undertaken from 1991 to 1993. Mosquitoes, sand flies, and ticks were collected and domestic ungulates were monitored with serologic surveys. Rift Valley fever virus was not isolated in Kedougou. In Barkedji, RVF virus was isolated from *Aedes vexans* and *Ae. ochraceus* mosquitoes collected in traps near ground pools and cattle droves and from one healthy sheep. Sand flies were not involved in the maintenance cycle. Seroconversions were recorded in three (1.9%) of 160 monitored sheep and goats. The interepizootic vectors appeared to belong to the *Aedes* subgenus *Neomelaniconion* in East Africa, and to the subgenus *Aedimorphus* in West Africa. Epizootics in East Africa are associated with an increase in rainfall. However, factors associated with epizootics remain unknown for West Africa.

Rift Valley fever (RVF) virus, a member of the family Bunyaviridae, genus *Phlebovirus,* is responsible for epizootics in ungulates resulting in abortions and deaths of newborns. It also causes human hemorrhagic fever epidemics throughout sub-Saharan Africa and Egypt.[1,2] The virus is thought to be zoonotically transmitted by infected mosquitoes.[1]

The RVF virus was first isolated in western Africa from *Aedes (Aedimorphus) dalzieli* mosquitoes in October 1974 in southeastern Senegal.[3] Other isolations were reported from *Ae. (Adm) cumminsii* and *Mansonia uniformis* mosquitoes in Burkina Faso, from *Culex antennatus* mosquitoes and *Culicoides sp.* in Nigeria, and from bats in Guinea.[4–7] Active RVF virus transmission in humans and domestic ungulates was even recorded during a period of drought in southern Mauritania and Mali in 1982–1985.[8] Large RVF outbreaks in western Africa had not been reported prior to the southern Mauritanian epizootic/epidemic in 1987.[9] Serologic data had established an extension of the epizootic throughout Senegal and The Gambia.[10,11] The virus was still active in southern Mauritania in 1988 but was not recovered from mosquitoes in northern Senegal.[12,13] Likewise, it was not isolated in a longitudinal study examining 490,000 mosquitoes collected on a monthly basis from 1989 to 1990 in the lower Senegal River basin. These mosquitoes mostly belonged to *Mansonia, Culex, Anopheles,* and halophilic *Aedes* species (Hervy JP, unpublished data).

Since 1989, successive serosurveys conducted on selected ruminants in Senegal showed a progressive decrease of RVF antibody prevalence.[14–16] However, the detection of RVF immunoglobulin G (IgG) and IgM antibodies in a few, young sheep and goats indicated the existence of an enzootic transmission of RVF virus in northern Senegal.[17] Many mosquito species have been implicated as epizootic vectors. Two subgenera of *Aedes* mosquitoes, *Aedimorphus* and *Neomelaniconion,* referred to as flood-water breeding *Aedes,* have been considered as possible vectors.[1,18]

MATERIALS AND METHODS

Entomologic survey. From 1991 to 1993, an entomologic survey was conducted in two areas selected because of previous evidence of RVF virus transmission: Kedougou (12°11′N, 12°33′W) located in southeastern Senegal and Barkedji (15°17′N, 14°17′W) in northern Senegal (Figure 1).[19,20] The study included captures of mosquitoes, sand flies, biting midges, and ticks.

The Kedougou area, located in the Sudano-Guinean bioclimatic zone, is characterized by a rainy season from May through October and an annual average rainfall of 1,100 mm (1,123 mm in 1991, 935 mm in 1992, and 1,111 mm in 1993). Arthropod collections were performed each year in July, October, and November from human bait from 5:30 PM to 10:30 PM with Centers for Disease Control (CDC) miniature dry ice (CO_2) light traps (John W, Hock Co., Gainesville, FL) and animal (sheep or chicken)–baited intermittent light traps.[19,20] Mosquitoes were sorted and pooled by species, sex, location, and date in the field. Pools of arthropods (≤ 100 mosquitoes) were placed in liquid nitrogen, then at stored −70°C until treatment.

The Barkedji area, located in the Sahelian Ferlo region, is characterized by a short rainy season from July to September with an annual average rainfall of 350 mm (215 mm in 1991, 347 mm in 1992, and 343 mm in 1993) (Figure 2). Temporary ground pools, filled soon after the first rains, remain the unique water resources for up to four months into the dry season.[21] Mosquitoes were collected at the edge of three temporary ground pools, 6 km apart, on a monthly basis, using the same techniques as above.

Viruses were isolated on AP-61 (*Ae. pseudoscutellaris*) and Vero cell line cultures, and were detected by immunofluorescence assay using specific mouse immune ascitic fluids as previously described.[22] Identification of the viruses was done using complement fixation and neutralization tests.[13,23] Engorged mosquitoes collected in the traps were preserved for blood meal studies.

Serosurveys. In Barkedji, a longitudinal serologic survey for RVF virus antibodies was conducted in domestic ungulates on a bimonthly basis, starting in March 1992. Four nonnomadic herds of sheep and goats, settled near the different temporary pools selected for mosquito captures, were selected for this serosurvey. Tagged animals (40 young females in each herd) were bled by venipuncture. Infesting

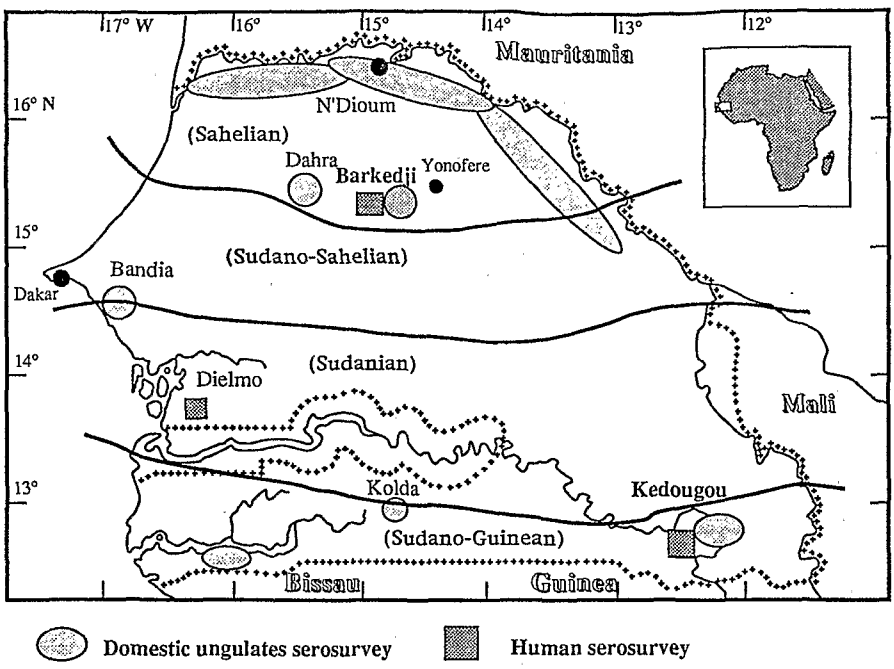

FIGURE 1. Locations of Barkedji and Kedougou study sites and other areas where serosurveys of domestic ungulates and humans were undertaken in 1992–1993 in Senegal.

ticks were collected and tested for presence of virus by inoculation into suckling mice. Clinical data and abortions were recorded from the herdsmen at each visit. It is possible that missing animals were replaced by new young females. Similar studies were not conducted in Kedougou.

Blood specimens were allowed to clot and were then centrifuged. Sera were stored at 4°C until tested for RVF IgG/IgM antibodies using an immunocapture enzyme-linked immunosorbent assay (ELISA) and seroneutralization tests as previously described.[14, 17, 24] Briefly, for RVF IgG detection, a β-propiolactone-inactivated suckling mouse liver antigen (RVF virus strain Dak Ar D 38861) was captured by a specific RVF mouse immune ascitic fluid adsorbed on polystyrene plates (Immunlon II; Dynatech Laboratories, Inc., Alexandria, VA). Test samples were added at a dilution of 1:100 and binding of specific IgG antibodies was detected with peroxidase-labeled, affinity-purified, anti-species antibodies (Kirkegaard & Perry Laboratories, Inc., Gaithersburg, MD) using *o*-toluidine (Sigma, La Verpilliere, France) as a chromogenic substrate. For IgM detection, plates were coated with an anti-species IgM (μ) antibody (Kirkegaard & Perry). The test samples at a dilution of 1:100 were added, followed by the RVF antigen. Specific binding was revealed using an RVF mouse immune ascitic fluid and peroxidase-labeled, affinity-purified, anti-mouse antibodies. A neutralizing antibody test was performed using Vero cell monolayers infected with an RVF Smithburn viral suspension with a titer of $10^{6.5}$ plaque-forming units/ml at a dilution of 1:1,600. Antibody-positive sera were determined by the absence of a cytopathogenic effect of the same serum dilution of 1:160.

Additional studies of RVF antibody distribution in domestic animals and humans were undertaken in 1991–1993 in different bioclimatic zones (Figure 1). In the Sahelo-Sudanian zone, Bandia area, individually tagged sheep and

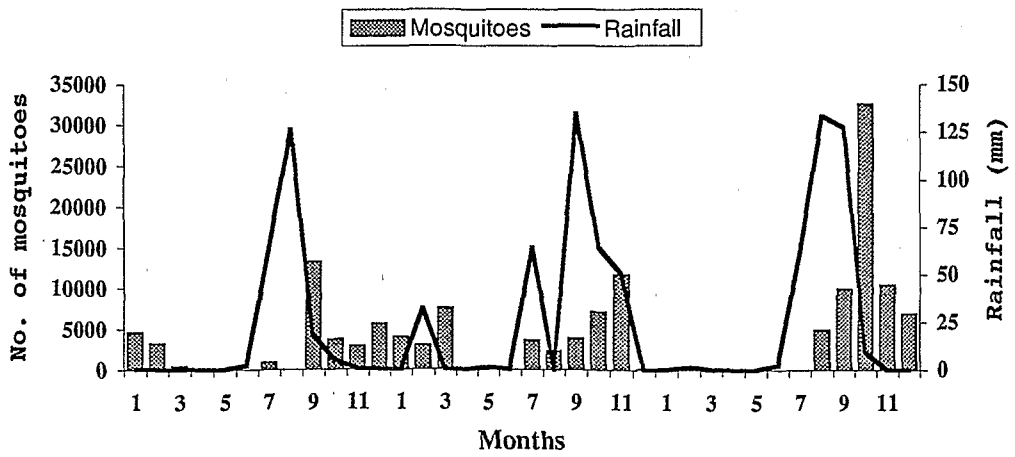

FIGURE 2. Monthly distribution of rainfall and mosquito captures from January 1991 to December 1993 in Barkedji, Senegal.

TABLE 1

Number of collected mosquitoes, number of inoculated pools, and number and proportion of *Aedes* sp. collected in Barkedji and Kedougou from 1991 to 1993

Location	Year	No. of mosquitoes	No. of pools	*Aedes* sp. No. (%)	*Aedes* pools No. (%)
Barkedji	1991	34,327	1,042	11,233 (32.7)	338 (32.4)
	1992	42,804	1,534	5,782 (13.5)	352 (22.9)
	1993	64,810	2,023	6,091 (9.4)	304 (15.0)
Subtotal		141,941	4,599	23,106 (16.3)	994 (21.6)
Kedougou	1991	48,377	1,264	28,579 (59.1)	730 (57.8)
	1992	37,685	1,364	26,889 (71.4)	804 (58.9)
	1993	71,992	2,493	31,224 (43.4)	1,136 (45.6)
Subtotal		158,054	5,121	86,692 (54.8)	2,670 (52.1)
Total		299,995	9,720	109,798 (36.6)	3,664 (37.7)

cows were monitored from March 1992 to October 1993 for RVF IgG/IgM antibodies. Other studies were undertaken in randomly chosen herds of cattle, sheep, and goats in the Sudano-Guinean zone (Casamance region and Kedougou area) in July 1992 and September 1993, in the Sudanian zone (Dielmo area) in September 1993, and in the Sahelian zone (Senegal River basin and Dahra area) from August 1993 to January 1994 (Figure 1). Sera were collected from animals and tested for RVF antibodies by immunocapture ELISA and the neutralization test.

In humans, serologic studies were undertaken in the Kedougou, Barkedji, and Dielmo areas (Figure 1). A nonrandomized study in the general population in eight locations in the Kedougou area was undertaken at the end of the rainy season from 1991 to 1993.[25] Informed consent was required for every individual to be included in the study prior to blood collection. In the Dielmo village, located in the Sahelain zone and selected for convenience, sera from the inhabitants were collected in July 1991, 1992, and 1993 in concordance with other pre-existing research.[26] In Barkedji, all children 7–14 years of age attending the primary school were included in the study (the only school in this 600-km^2 area) in April 1993. Sera were tested for RVF IgG/IgM antibodies by immunocapture ELISA. The chi-square Pearson test was used for statistical analysis.

RESULTS

Entomologic investigations. From 1991 to 1993, a total of 134,695 female mosquitoes (4,374 pools) and 7,246 male mosquitoes (225 pools) comprising 39 species (six genera) were captured in Barkedji, and 152,191 female (5,006 pools) and 5,863 male (115 pools) mosquitoes from 67 species (six genera) were captured in Kedougou (Table 1). Fourteen species of *Aedes* were identified; six species belonged to the subgenus *Aedimorphus* and represented 90.1% of the *Aedes* sp. captured and one species belonged to the subgenus *Neomelaniconion.* In addition, 105,931 sand flies (951 pools) and 1,290 biting midges (32 pools) were captured in Barkedji, and 1,232 sand flies (11 pools) and 20 biting midges (one pool) were captured in Kedougou.

In Barkedji, the temporal pattern of mosquito abundance was highly seasonal, corresponding to the abundance of rainfall (Figure 2). In 1991, 32.7% of the mosquitoes captured were *Aedes* spp., 13.5% in 1992, and 9.4% in 1993. *Aedes (Adm) vexans* were most abundant, followed by *Ae. (Adm) ochraceus* (Table 2). *Aedes (Adm) vexans* and *Ae. (Neo) mcintoshi* were proportionally more abundant at the beginning of the rainy season.

TABLE 2

Number of *Aedes dalzieli, Ae. mcintoshi, Ae. ochreaceus,* and *Ae. vexans* mosquitoes collected in Barkedji and Kedougou areas in 1991–1993, proportion among the *Aedes* spp. mosquitoes, number of inoculated pools and Rift Valley fever (RVF) virus isolations

Location/species	Year	No.	% among *Aedes* sp.	No. of pools	No. of RVF virus isolations
Barkedji					
Ae. dalzieli	1993	10	0.1	5	
Ae. mcintoshi	1991	281	2.5	16	
	1992	258	4.5	35	
	1993	21	0.3	7	
Subtotal		560	2.4	58	
Ae. ochraceus	1991	282	2.5	21	
	1992	634	11.0	47	
	1993	1,269	21.0	58	3
Subtotal		2,185	9.5	126	3
Ae. vexans	1991	10,232	91.2	248	
	1992	4,204	72.7	122	
	1993	4,142	68.4	123	10
Subtotal		18,578	80.5	493	10
Kedougou					
Ae. dalzieli	1991	10,122	35.4	192	
	1992	5,064	18.8	130	
	1993	10,650	34.1	273	
Subtotal		25,836	29.8	595	
Ae. mcintoshi	1991	28	0.2	4	
	1992	62	0.2	18	
	1993	136	0.4	28	
Subtotal		226	0.3	50	
Ae. ochraceus	1991	59	0.2	8	
	1992	41	0.2	12	
	1993	520	1.7	44	
Subtotal		620	0.7	64	
Ae. vexans	1991	367	1.3	13	
	1992	17	0.1	5	
	1993	754	2.4	50	
Subtotal		1,138	1.3	68	

In the first weeks following the flooding in July 1991, 28% of the mosquitoes captured with light traps (15 night-traps), were *Ae. (Neo) mcintoshi,* compared with 0.1% in September and October and none in November. *Culex poicilipes* appeared later, toward the middle of the rainy season. *Mimomyia splendens, Aedeomyia africana,* and *Mansonia africana* were more abundant at the end of the rainy season. In addition, 194 *Ae. (Adm) minutus* (0.8% of the total of *Aedes* spp.), 32 *Ae. (Adm) argenteopunctatus* (0.1%), and 21 *Ae. (Adm) fowleri* (0.1%) were captured from 1991 to 1993.

Other mosquito species, previously reported as possible RVF virus vectors,[1] were also captured: 940 *Culex* group *univittatus* (54 pools), 5,356 (212 pools) *Cx. neavei,* and 1,716 *Mansonia uniformis* (107 pools). Sand flies were abundant during the dry season from November through April, with a maximum in January, the coldest month.

In the Kedougou area, mostly *Aedes* mosquitoes comprised 59.1% of the mosquito collections in 1991, 71.4% in

TABLE 3

Rift Valley fever virus antibody prevalence in monitored domestic ruminants in the Barkedji and Bandia areas (March 1992–March 1994)

Location	Animal species		Mar 1992	Oct 1992	Mar 1993	Oct 1993	Jan 1994	Mar 1994
Barkedji								
	Sheep/goat	No. tested	60	160	160	160	160	160
		IgG %	5.0	2.5	3.1	3.7	5.6	5.6
		IgM %	0	0	0	0	0	0
		NT* %	0	0	0	0	1.9	1.9
Bandia								
	Sheep	No. tested	9	5	5	3		
		IgG %	0	0	0	0		
	Cattle	No. tested	25	23	21	18		
		IgG %	0	0	0	0		

* NT = neutralization test.

1992, and 43.4% in 1993 (Table 1). Twenty-two species of *Aedes* were identified; 11 species belonged to the subgenus *Aedimorphus* representing 61.1% of the *Aedes* sp. captured, and one species belonged to the subgenus *Neomelaniconion.* Sheep bait, sleeping under an unclosed mosquito net, and mosquito CO_2 light traps allowed the capture of many *Ae. dalzielil* at the beginning of the rainy season, and a few specimens later in the year. *Aedes dalzieli* were predominant, while *Ae. vexans, Ae. mcintoshi,* and *Ae. ochraceus* were present but less abundant (Table 2). In addition, from 1991 to 1993, 10,478 *Ae. minutus* (6.6% of the *Aedes* spp), 4,749 *Ae. fowleri* (3.0%), and 4,689 *Ae. argenteopunctatus* (3.0%) were captured. The remaining *Aedes* mosquito species constituted 56.6% of the *Aedes* captures. Other species included 360 *Cx. neavei* (40 pools) and 12,987 *Mansonia uniformis* (266 pools).

Virus isolations. In Barkedji, RVF virus was isolated from seven of 38 pools of *Ae. vexans* (1,187 females) and from three of 30 pools of *Ae. ochraceus* (852 females) in October 1993. The virus was recovered from three of 10 pools of *Ae. vexans* (333 females) in November 1993 (Table 2). Mosquitoes were captured in CO_2/CDC light traps around three temporary ground pools and near cattle droves. Viruses were isolated on AP-61 cells but not on suckling mice or Vero cells. No isolations were reported from phlebotomine sand flies, biting midges, or from 1, 717 ticks (402 pools) collected in 1992–1993 on monitored animals. In Kedougou, RVF virus was not isolated from the captured mosquitoes, phlebotomine sand flies, and biting midges.

Serosurveys. In Barkedji, the longitudinal serosurvey of individually tagged female sheep and goats started in March 1992 with 60 animals from four different herds. From August 1992 through March 1994, 160 animals were monitored. In a few lambs and kids (≤ four months old), which were newly included in this study as replacements for missing animals, RVF IgG antibodies were detected by ELISA and then disappeared within two months (Table 3). In January 1994, RVF IgG and neutralizing antibodies were recorded in three of 160 animals (1.9%), but RVF IgM antibodies were not detected (Table 3). These three ewes belonged to the same herd of 28 sheep and 12 goats, and one was pregnant. No abortions were reported by herdsmen among the monitored animals from October 1993 to January 1994. A retrospective attempt for viral isolation from the sera of these three sheep collected in October 1993 was successful with the serum of one animal inoculated into suckling mice.

Serosurveys conducted in domestic ruminants in Senegal in different bioclimatic zones showed the presence of RVF IgG antibodies in 38 (5.8%) of 654 bovines and 36 (4.6%) of 778 small sheep and goats without IgM antibodies (Table 4). No RVF antibodies were detected among the monitored cattle and sheep in Bandia from March 1992 to October 1993 (Table 3).

TABLE 4

Rift Valley fever antibody prevalence in domestic ruminants in three different bioclimatic zones in Senegal (1992–1994) by enzyme-linked immunosorbent assay and neutralization test (NT)*

Bioclimatic zone	Location	Animal species	Date	No. tested	RVF positive No. (%)		
					IgG	IgM	NT
Sahelian							
	Senegal River basin	Cattle	8–9/93	483	36 (7.5)	0	38 (7.9)
		Sheep	1/94	200	10 (5.0)	0	4 (2.0)
	Dahra	Cattle	11/93	96	0 (0.0)	0	0 (0.0)
Sudanian							
	Dielmo-N'Diop	Sheep	9/93	91	7 (7.7)	0	ND
	Dielmo-N'Diop	Cattle	9/93	75	2 (2.7)	0	ND
Sudano-Guinean							
	Casamance	Sheep/goat	9/93	296	8 (2.7)	0	ND
	Kedougou	Sheep/goat	7/92	191	11 (5.8)	0	1.05

* ND = not done.

TABLE 5

Rift Valley fever (RVF) virus IgG antibody prevalence in the general population and children (5–15-years old) in Senegal from the Kedougou and Barkedji areas and Dielmo village

Location	Date	General population		Children (5–15-years old)	
		No. tested	RVF IgG %	No. tested	RVF IgG %
Kedougou	10/91	517	3.09	138	0.72
	10/92	457	2.41	135	0.00
	10/93	412	2.67	162	1.24
Barkedji	4/93			245	6.12
Dielmo	7/91	257	3.11	46	2.17
	7/92	254	1.96	46	2.17
	7/93	249	2.01	87	0.00

In the human population in the Kedougou area, the overall RVF IgG antibody prevalence was low (2.7%), without significant variations from 1991 to 1993 (Table 5). The prevalence was similar among the overall population of Dielmo from 1991 to 1993 in an annual monitoring effort. A significantly higher RVF IgG antibody prevalence was observed in children in Barkedji compared with children from the Kedougou area in 1993 ($\chi^2 = 5.8$, degrees of freedom = 1, $P < 0.02$) (Table 5).

DISCUSSION

A possible focus of RVF virus activity was suspected in Barkedji, where serologic data had shown RVF antibodies in non-nomadic, small, young ruminants in March 1992 (Table 3). The entomologic and serologic surveys undertaken in the area allowed the isolation of RVF virus from one sheep and two new Culicidae vectors (*Ae. vexans* and *Ae. ochraceus*) in October–November 1993.[27] The mosquito abundance was highly seasonal and closely related to the temporal pattern of rainfall.[20,28] Entomologic surveys conducted around the temporary ground pools showed an abundance of mosquito species able to transmit RVF virus to vertebrates in the weeks following the flooding.

The likely potential mosquito vectors belonged mostly to the *Aedes* (*Neomelaniconion*) and *Ae.* (*Aedimorphus*) subgenera.[1] *Aedes dalzieli, Ae. mcintoshi, Ae. ochraceus, Ae. vexans, Ae. minutus, Ae. fowleri,* and *Ae. argenteopunctatus* mosquitoes represented 93.4% of the *Aedes* collected in Barkedji and 44.6% in Kedougou. The predominance of *Ae. vexans* and *Ae. ochraceus* mosquito species in Barkedji was observed in 1989–1990 in Yonofere (Figure 1).[28] Blood meal studies for *Ae. ochraceus* and *Ae. vexans* have shown their preferences for sheep and bovines, but *Ae. vexans* was able to feed on a large variety of vertebrates, including humans.[13,27] Only a few specimens of other potential vectors (*Ae. minutus, Ae. fowleri,* and *Ae. argenteopunctatus*) were captured in Barkedji.

In Kedougou, eight species of mosquitoes that previously had been found naturally infected with RVF virus were captured. *Aedes dalzieli* mosquitoes were predominant, and other potential *Aedes* vectors were more abundant than in Barkedji. Breeding places of *Ae. dalzieli* were small ground pools located in the flood plain of temporary rivers in the gallery forests. Hatches were particularly abundant in this region, occurring within a few days following the first rain, which normally arrives in June. It is important to note that the first captures of mosquitoes were in July, which could have masked a previous predominance of some *Aedes* species.

Sand flies were not involved in RVF virus maintenance, yet they allowed the replication of other viruses.[21–29] However, *Phlebotomus duboscqi,* one of the 11 identified sand fly species in Barkedji, has been shown experimentally to transmit RVF virus.[30] The role of *Culicoides* was not investigated.

Inter-epizootic vectors appeared to belong to the *Aedes* subgenus *Aedimorphus* in West Africa.[3,27] However, the presence of *Ae. mcintoshi,* the unique mosquito species of the *Neomelaniconion* subgenus captured in Kedougou and Barkedji, was confirmed in Senegal. It appeared in the first weeks following the flooding of temporary ground pools. In East Africa, RVF virus has been isolated from *Ae. mcintoshi* captured near shallow streamless depressions described as dambos in Kenya or broad vleis in Zimbabwe.[18,31] Isolations of the virus from male and female *Ae. mcintoshi* reared from larvae and never fed as adults provide strong evidence of transovarial transmission.[32] A possible annual emergence of infected mosquitoes may maintain the RVF enzootic foci, as was suggested in Zimbabwe.[31] Transovarially infected larvae emerge and develop into infected adults when their habitats are flooded. Females may then feed on nearby susceptible livestock. Others secondary mosquito vector populations may be orally infected from viremic domestic animals. The absence of isolations of RVF virus from susceptible mosquito species in Barkedji provided strong evidence of the role of *Ae. vexans* and *Ae. ochraceus* as potential enzootic vectors (Figure 3). Infected mosquitoes were captured near the three monitored ground pools, indicating a local dispersion of the virus at the end of the 1993 rainy season. In contrast, an undetectable circulation of the virus or an absence of emergence of infected mosquitoes was reported in 1991 and 1992. Flood water *Aedes* have drought-resistant eggs, which may be able to survive several years without hatching and then require one or more floodings to trigger their development.[29]

The RVF virus serologic surveys of domestic animals showed that a few animals that tested positive by ELISA were negative by the neutralization test because only sera with a neutralization test titer ≥ 160 were considered positive (Table 3).[14] Maternal IgG antibodies to RVF virus in lambs and kids were recorded, and they disappeared within two months. Nevertheless, the presence of unknown phleboviruses could produce cross-reactions by ELISA as previously reported in Burkina Faso.[33] An additional study performed on 25 RVF virus IgG-positive and 25 negative animal sera did not show cross-reactivity by immunocapture ELISA with phleboviruses previously isolated in West Africa, including Gabek Forest, Saint-Floris, or Gordil.

A study of the duration of RVF IgM antibodies after natural infection in cattle in Madagascar showed that only 27% of the cattle remained positive two months after the acute infection.[34] The absence of RVF IgM antibodies in monitored sheep in Barkedji in January 1994 indicated that infection had occurred in October or November 1993. This hypothesis was confirmed by the isolation of the virus from

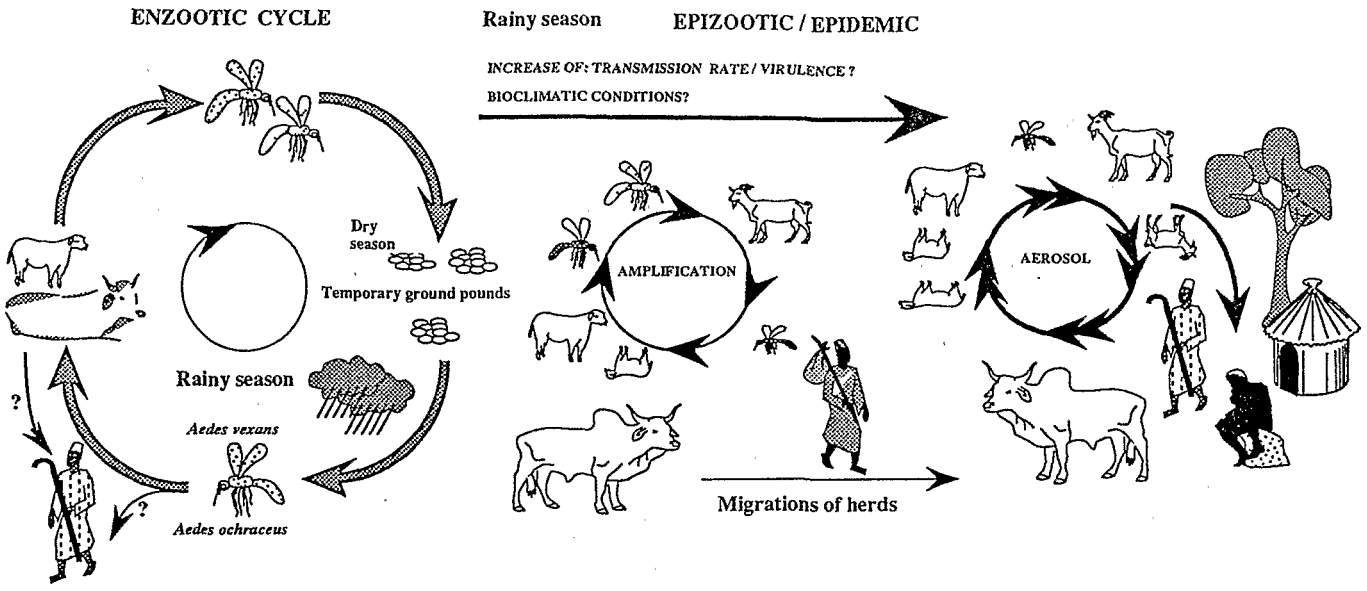

FIGURE 3. Possible cycles of Rift Valley fever in Sahelian western Africa with an enzootic virus maintenance around temporary ground pools involving *Aedes vexans* and *Ae. ochraceus* primarily vectors and epizootic/epidemic amplifications involving various mosquito species, movements of animals, and direct transmission (aerosol, contact with abortion products).

one animal in October. Such an isolation was fortuitous given that the duration of viremia is usually 2–4 days. However, during the Egyptian epidemic, the virus was isolated up to 10 days after initial onset in humans.[1] Conversely, another RVF virus isolation was reported from one healthy ox in Kolda (Casamance) in November 1993 (Thiongane Y, unpublished data). Vero cells and suckling mice, which are usually reported as sensitive models for RVF virus isolation, were not suitable for isolation of RVF virus from the mosquito pools, but they were useful for viral isolation from animal sera.

The distribution of RVF virus antibody prevalence in Senegal in domestic ruminants confirmed the hypothesis of the presence of several RVF loci in different bioclimatic zones without noticeable clinical disease. The low incidence of infection in domestic ungulates reported in Barkedji has also been previously observed in Kenya near the forest edge.[35] In Zambia, a sentinel herd exposed from 1982 to 1986 showed that RVF occurred every year at a low level.[36] When the enzootic circulation of RVF virus was detected here in Barkedji in October 1993, another RVF epizootic was reported in small ruminants in southeastern Mauritania, 250 km east of Barkedji.[37] An undetectable RVF enzootic maintenance in these areas or a possible new introduction of the virus was suggested. A serologic survey conducted in January 1994 in sheep, along the Senegalese left bank of the Senegal River basin, close to the Mauritanian infected areas, did not show any extension of the outbreak in Senegal (Table 3).

In humans, a low prevalence of RVF antibodies similar to that found in Senegal had been previously reported in coastal Kenya and in various locations of Mozambique.[38,39] In February–May 1989, a study conducted in Yonofere showed a overall RVF IgG prevalence of 22.3% in humans and a prevalence of 14.2% in the 5–19-year-old age group.[28] This probably resulted from a previous epizootic in 1987–1988 in which several cases of human hemorrhagic disease fatalities were reported. This previous outbreak could explain the higher prevalence of RVF antibodies recorded in schoolage children in 1993 in Barkedji.

The role of rodents in RVF maintenance is still not well-documented. Observations in South Africa seemed to demonstrate that rodents could be infected.[40,41] In Senegal, two of 268 *Mastomys* sp. were reported positive by immunofluorescence assay.[4] Other studies (Zeller HG, Duplantier JM, unpublished data) showed neutralizing RVF antibodies in two *Arvicanthis niloticus* among 70 rodents trapped in March 1990 in N'Dioum in northern Senegal, and an absence of RVF antibodies among 57 rodents (*Mastomys erythroleucus, Arvicanthis niloticus, Taterillus* sp., and *Desmodilliscus braueri*) trapped in January 1993 in Barkedji (Figure 1).

It has been suggested that the 1987 epidemic/epizootic outbreak on the Senegal River had its origin in the Mauritanian and Malian Sahelian regions, and was associated with alterations in the ecology of the region with the irrigation projects and dam building and the development of new ecologic habitats for potential vector species.[42] However, the rapid decrease of antibody prevalence recorded from 1987 to 1992 in northern Burkina Faso and Senegal suggest an interepizootic period in Sahelian regions with maintenance of RVF virus mostly confined in more humid areas.[17] Epizootics in sub-Saharan Africa that occur simultaneously over geographic areas separated by several hundred kilometers are consistently reported.[1] They are associated with unusually heavy rainfall and large numbers of mosquitoes.[43] This same relationship between an increase in rainfall and RVF epizootics, as observed in East Africa, has not been established in Sahelian areas. For example, the RVF activity described in southern Mauritania in 1982–1985 occurred during a period of drought, and the 1993 epizootic was not associated with extensive rainfall.[8,37]

The enzootic maintenance of RVF virus in Barkedji was suspected when RVF epizootics coincidentally occurred in

Mauritania and in Egypt without a relationship with heavy rainfall.[37,44] Given the sporadic distribution in the past, risks of RVF transmission and epizootics during the next rainy season may appear in areas in which the virus has not been detected.[42] Furthermore, the extension of irrigation within the Senegal River basin with the Manantali and Diama dams may possibly increase the risks of RVF transmission in the future. This association has been observed in Egypt and may have been a contributing cause of the 1987 epidemic in Mauritania.[42] Otherwise, monitoring the enzootic circulation of RVF virus in West Africa Sahelian areas would be most effective in October–November.

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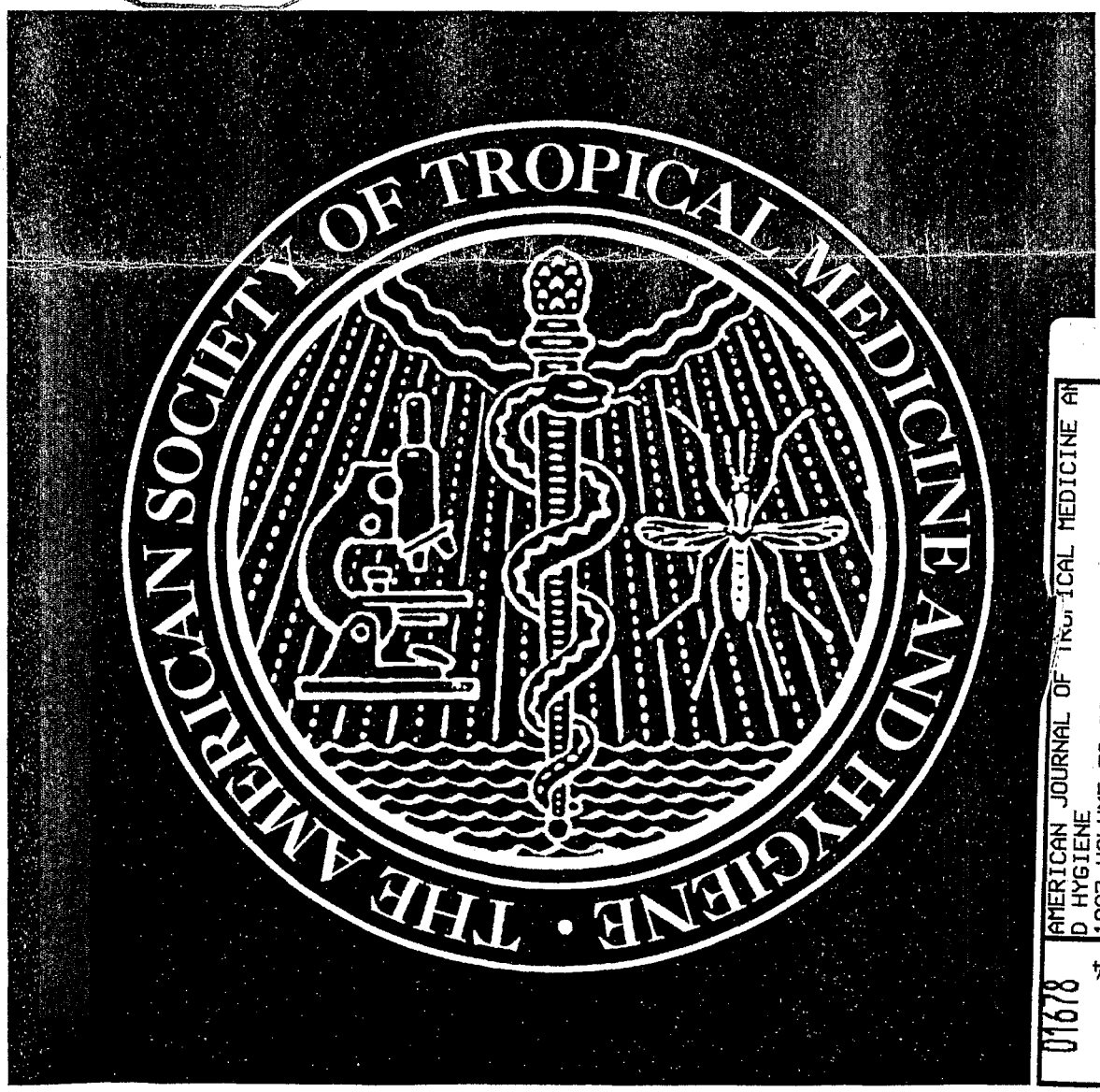

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