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Chapter 18

BUNYAMWERAL FEVERS: BUNYAMWERA, ILESHA, GERMISTON, BWAMBA, AND TATAGUINE

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I. INTRODUCTION

Bwamba, Bunyamwera, Germiston, Ilesha, and Tataguine viruses, all members of the family Bunyaviridae,[1-4] are endemic in Africa and have never been recognized elsewhere. All these viruses can cause mild febrile illness, with or without rash; none is responsible for important epidemic disease or has a great social or economic impact. Interest in the study of these viruses centers on both their epidemiology and their structure. Among the five viruses, Bunyamwera, Bwamba (including Bwamba-related virus such as Pongola), and Tataguine have been isolated most frequently.

II. HISTORICAL BACKGROUND

A. Discovery of Agents

In 1941, Smithburn et al.[5] first described the isolation of Bwamba virus. In 1946, they[6] reported the first isolation of Bunyamwera virus, which is considered the prototype of the Bunyamwera group; it was recovered in Uganda from a pool of *Aedes* mosquitoes.

In 1957, Ilesha virus was isolated in a Nigerian village of the same name from blood of a 9-year-old girl.[7] Germiston virus was recognized by Kokernot et al.[8] in 1960 in South Africa. The first isolation of Tataguine was made by Brès et al.[9] from a mixed pool of *Culex* and *Anopheles* mosquitoes collected in Senegal in 1961.

B. History of Human Cases and Geographical Spread

Bwamba virus was first recognized in the setting of a small outbreak in western Uganda; nine cases were confirmed by virus isolation.[5] Bwamba infection has been recognized by virus isolation from humans in Uganda,[5] Nigeria,[10] Cameroon,[11] Central African Republic,[11,12] Kenya, Tanzania,[5] and South Africa.[13] Identification of human infections by Bwamba group viruses was reported in Ethiopia by Ota et al. (unpublished) and, more recently (1978), in Kenya.[14] In Bwamba County (Uganda), where Bunyamwera virus was first isolated, 2.9 to 30% of the human population was found with neutralizing antibodies.[5]

Tataguine virus is present in Senegal, where 57% of the inhabitants were found to have antibodies, and eight strains were isolated from inhabitants of Dakar suburbs.[9] It has also been found in Cameroon,[15] Nigeria,[16] the Central African Republic,[12,17] and Ethiopia.[18]

After its first isolation in Nigeria,[7] Ilesha virus was found in Cameroon, Senegal, and the Central African Republic.[11,12] Ilesha virus, but not Bunyamwera,[19] is considered to be endemic in southern Ethiopia.

None of the five viruses seems to be responsible for epidemics. They are, nevertheless, distributed widely in all of tropical Africa.

C. Social and Economic Impact

Generally responsible for only mild diseases, these viruses have a very limited recognized social and economic impact.

III. VIRUS CHARACTERISTICS

Bunyaviruses from the Western Hemisphere show serological cross reactions with those from Africa.[20] Nevertheless, it is quite easy to identify each African virus using either complement fixation (CF) or mouse protection neutralization (MPN).[21]

The genomes of Bunyaviridae consist of a single strand of RNA comprising three segments. Experimental recombination shows that genetic material can be exchanged between members of Bunyamwera group and other Bunyaviruses. This could explain the occurrence of "mosaic" strains of different Bunyaviruses with similar geographic and ecologic distribution, as mentioned by Iroegbu.[22] The reassortment of RNA segments can explain the diversity of members of the Bunyaviridae family, as well as the cross reaction observed using serological tests.

Germiston viral polypeptides have been studied by Ozden and Hannoun[23] consist of three major structural polypeptides (mol wt 125 × 10^6, 27 × 10^6, 18 × 10^6) and one minor larger protein (mol wt 185 × 10^3).

A. Antigenic Relationships

Bunyamwera virus is closely related to other members of Bunyamwera group, but a prior infection by any member of this serogroup does not prevent human infection with another virus of the same group. Ukauwa virus is now considered to be a strain of Bunyamwera virus.

Bwamba virus cross reacts by neutralization test (N) with Pongola virus[24] nevertheless, it clearly differs in a quantitative reciprocal manner. Johnson et al.[14] demonstrated that a Bwamba virus variant isolated in Kenya could present antigenic characteristics of Pongola virus.

Originally, Germiston virus was described as having significant reciprocal neutralization (adult mouse N test) with Bunyamwera; nevertheless, these two viruses are readily distinguished by hemagglutination inhibition (HI) and CF, as mentioned by Kokernot et al.[8]

Ilesha virus cross reacts to some degree with Bunyamwera by the N test;[25] by HI, the closest relationship is with Cache Valley.[26] By CF test, Ilesha virus is related more closely to Bunyamwera and Cache Valley than to other members of the Bunyamwera serogroup.[27]

B. Host Range

Table 1 summarizes the available data concerning natural host range, as evidenced by virus isolation and antibody tests. All five viruses have been isolated from humans, whereas data on other vertebrate species are scanty. Susceptibility of laboratory hosts is variable. All viruses are pathogenic for newborn mice by the intracerebral (i.c.) route; Bunyamwera and Germiston viruses are the most pathogenic, causing death in weaned mice by the peripheral route. Tataguine virus is the least pathogenic and does not produce illness in suckling mice inoculated i.c. or weaned mice by any route. Susceptibility of hamsters, rabbits, and other experimental hosts is described in the *International Catalogue of Arthropod-Borne Viruses*.[27]

C. Methods for Assay

All of the viruses may be assayed by i.c. inoculation of newborn mice. Cell culture systems can be used, but the cytopathic effect is not always easy to see and is sometimes absent. Susceptibilities of some cell culture systems are given in Table 2.

Techniques for virus isolation and assay using mosquito cell cultures or mosquitoes inoculated by the intrathoracic route are described below. The sensitivity and specificity of various infectivity assays are different for each virus. The N test performed with Tataguine virus in VERO cells has a high specificity and is more sensitive than the MPN test.[28]

IV. DISEASE ASSOCIATIONS

A. Humans

The most frequent symptoms are fever, headache, arthralgia. The hallmark of nearly all cases is their brief duration (4 or 5 days) and benign nature: no fatalities are recorded.

Physical examination is generally normal except, at times, for the presence of conjunctivitis or stiffness of the neck.[11,12] Convalescence is characterized by marked asthenia lasting 8 to 10 days.

Bwamba virus is responsible for a relatively severe form of generalized infection. Exanthem is nearly always present, and it is frequently associated with meningeal involvement.[74] A case of myocarditis has been reported.[11] Intestinal tract involvement, especially diarrhea, is also seen.[74]

Bunyamwera virus is generally responsible for pediatric infection. Children present with fever, headache, joint pains, and rash. Recovery occurs in less than 7 days. In some cases, visual disturbances and vertigo have been observed.[14] When infection occurs accidentally or in immunologically compromised patients, severe encephalitis can be observed.[29]

Ilesha virus infections are characteristically mild, with the primary symptom being a feeling of malaise. Fever, when present, is generally less than 39°C. A transient discrete exanthem is seen in about half of the cases.[30] Recovery occurs without sequelae, but asthenia persists for 8 to 10 days.

Tataguine virus is associated with mild disease in children and more severe symptoms in adults. Rash is present; however, fever is usually less than 39°C. Some patients complain of marked headache, gastrointestinal symptoms, and a florid, nonpruritic rash.[10]

Only two laboratory-acquired Germiston virus infections have been reported from South Africa; both were characterized by mild disease without specific symptoms and with recovery occurring in 37 hr to 3 days.[8]

B. Wild and Domestic Animals

No natural disease has been reported.

C. Diagnostic Procedures

1. Virus Isolation

Viremia is usually of short duration (24 to 48 hr); Ilesha virus, however, has been isolated 4 days after onset of disease.[12] The greatest number of successful virus isolations has been made using both i.c. and subcutaneous (s.c.) or intraperitoneal (i.p.) inoculation of suckling mice (usually 1 to 2 days of age). Material for inoculation consists of blood, serum, or plasma from humans and mammals or of arthropod pools diluted in Hanks' balanced salt solution or similar solution containing a source of protein (bovine albumin or serum). The virus can be identified at the time of harvest of a mouse brain tissue using the Chrom Elisa Technic of Lhuillier and Sarthou,[31] or it may be established by passage and identified by use of an appropriate serological test (CF or N test).

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Less experience has been accumulated with the use of cell culture systems for the isolations of these viruses. Continuous cell lines, such as VERO or BHK-21 or *Aedes albopictus* (C6/36), may be useful. Inoculated C6/36 cells can be tested between days 2 and 10 for virus by indirect immunofluorescence (IFA) with specific ascitic fluids or held for as long as 2 weeks with infectious virus detectable by an appropriate technique such as mouse inoculation.

The viruses under consideration can also be isolated by intrathoracic inoculation of *Toxorhynchites* mosquitoes.[32] Antigen is demonstrated by testing mosquito head squashes by IFA or pooled mosquitoes by CF.

Detection of virus in serum by enzyme immunoassay, as described for yellow fever,[33] could represent a new approach to diagnosis, but is limited by the probable short duration and low titer of viremia in cases of most bunyaviral infections.

2. Serological Diagnosis

One must consider the serological cross reactivity within the Bunyaviridae family.[27] The "original antigenic sin" phenomenon applies to humans infected with multiple Bunyamwera-group viruses. Usually, a battery of viruses and tests is required to clarify the diagnosis.[34]

Serological response to infection can be low and/or without seroconversion in the case of Tataguine virus;[12] infection can be serologically diagnosed by the N test but not the CF test.[11] Methods such as IgM antibody detection, described for other arboviruses, could represent a new approach to serodiagnosis.

V. EPIDEMIOLOGY

A. Geographic Distribution and Seroepidemiology

The distribution of these viruses determined by virus isolation has been described (in Section II.B) and is shown in Table 3. The table also shows the results of seroprevalence surveys in various countries of Africa.

No serological or virological evidence for activity of any of these agents has been found in North Africa, with the exception of Egypt, where a low prevalence (0.9%) of N test antibodies to Bunyamwera but no antibodies to Bwamba virus was reported.[35]

In tropical parts of West Africa, the prevalence of BUN antibodies has been high in many countries.[36] In Equatorial Guinea, the prevalence of antibodies to Bunyamwera and Bwamba viruses appeared to be higher than to most other arboviruses.[37] In contrast, a low prevalence of Bunyamwera antibodies was reported in Togo, Benin, Mali, Niger, and Chad.[38,39]

In Central Africa, 100% of the populations inhabiting the rainforest area of the Congo had antibodies after 10 years of age, whereas only 8.0% of persons in the same age group in Gabon were immune.[40] Considering Bunyamwera antibody prevalence in Africa as a whole, it is apparent that it is highest in the tropics, low in North Africa, and shows a sharp decline in the Republic of South Africa, being 0% in Cape Province and 45% in North Natal.[41]

In East Africa, Bunyamwera and Bwamba viruses appear to be endemic in Uganda, Tanzania, and Mozambique.[41,42] A low prevalence of BUN antibodies has been found in Madagascar, with the highest percentage positive on the north coast.[43]

In South Africa, Bunyamwera and Bwamba antibody prevalences are significantly higher in the Simbu Pan area of North Natal than in other regions. Natal seems to be the southern limit for arboviruses, as they require a tropical ecology. Germiston virus seems to be active only in Austral Africa, with a specific importance in Angola and Botswana. In South Africa, Germiston antibodies are found in livestock.[36,44]

Tataguine virus has a wide geographic distribution encompassing West and Central Africa. In Nigeria, the highest prevalence of antibodies was found in the derived savannah zone (61%) followed by lowland rain forest (42%).[45]

B. Incidence

Evidence for human infections has been accumulated by many studies and is summarized in Table 3.[13,15,36,40,43,44,46,50] After chikungunya, Semliki Forest, Sindbis, yellow fever, Uganda S, West Nile, Wesselsbron, and Zika, Bunyamwera and Bwamba appear as the ninth and tenth most frequent arboviruses infecting humans in the African continent.[36] In the Central African Republic, Bwamba was the virus most often isolated from human cases of arboviral infection.[13] Tataguine virus also appears to be a very common human infection in some areas, e.g., Nigeria,[45] where multiple virus isolations have been made from febrile patients.[46]

C. Seasonal Distribution

Bunyamwera virus has been isolated in either the middle or, more frequently, at the end of the rainy season. In the Central African Republic, Ilesha and Bwamba viruses were most often isolated during the dry season. All Tataguine virus isolations were made during the dry season. Germiston strains from Kenya were isolated at the beginning of the rainy season.[47]

D. Risk Factors

Bunyamwera antibodies seem to appear earlier in age in females than in males, and the seroprevalence in females remains higher lifelong.[49,50]

VI. TRANSMISSION CYCLES

A. Evidence from Field Studies

1. Vectors

Bunyamwera virus has been isolated from mosquitoes belonging to three genera (*Aedes*, *Mansonia*, and *Culex*), but *Aedes* spp. appear to play the predominant role in transmission. Multiple strains of the virus were recovered from *Ae. circumluteolus* collected at the same time and place as a naturally infected human in South Africa.[55] The wide array of mosquito species which have yielded virus suggests that high viremia levels may occur in a variety of different vertebrate hosts.

Little field evidence has been accumulated for Ilesha virus; the virus has been isolated from *Anopheles gambiae* in the Central African Republic and Senegal and from *Cx. thalassius* in Senegal.[57] Germiston virus has been recovered repeatedly from *Cx. rubinotus* in South Africa,[8,58] Zimbabwe,[58] Mozambique,[58] Kenya,[57] and Uganda.[59] High minimum infection rates in this species suggest that transovarial transmission of the virus may occur.

Bwamba virus has been isolated from *An. funestus* in Uganda,[27] Senegal, Nigeria,[27] Central African Republic,[57] and the Ivory Coast[57] and from *An. gambiae* and *Ae. furcifer* in Senegal.[57] Twelve strains of Bwamba virus were isolated during field studies in riverine forest in Nigeria in 1971,[60] the majority from *Ae. (Neomelanoconion)* spp. and *Ae. (N.) circumluteolus*, but also from *An. coustani* and *Ma. uniformis*. The *Aedes* were captured on human bait, suggesting that these mosquitoes, as well as anophelines, may be responsible for human infections.

Tataguine virus has been isolated principally from anopheline mosquitoes. Multiple strains have been recovered from *An. gambiae* in Cameroon,[15] Central African Republic, Senegal, and Ethiopia; from *An. funestus* in Nigeria, Central African Republic,[27,57] and Ethiopia; and from *An. nili* in Senegal.[27,57] An isolate has also been made from *Coquillettidia aurites* in Cameroon.[27,57] The association between virus isolations from humans and anopheline mosquitoes suggests that transmission occurs in the domestic habitat with humans serving as a viremic host.[15]

2. Vertebrate Hosts

No isolations of Bunyamwera, Ilesha, Bwamba, or Tataguine viruses have been made from naturally infected vertebrates. Germiston virus has been isolated on multiple occasions from rodents (Table 4): from *Dasymys incomtus* in Kenya[48] and from *Rattus rattus*, *Arvicanthus niloticus*, *Lophuromys sikapusi*, and *L. flavopunctatus* in Uganda.[59,61] An isolate was made from a mongoose (*Herpestes ichneumon*) in Kenya.[48] Multiple isolations of Germiston virus were also made from sentinel hamsters in South Africa and Mozambique.[62]

The interpretation of serological evidence for involvement of vertebrate hosts with Bunyamwera group viruses is limited somewhat by the problem of antigenic cross reactivity among members of the serogroup. Neutralizing antibodies to Bunyamwera, Bwamba, and Germiston viruses have been found in domestic livestock (Table 4). A high seroprevalence to Germiston virus has also been found in rodents, in accord with virus isolation data implicating them as hosts. A high prevalence of antibodies to Tataguine virus in humans and absence of antibodies in wild and domestic animals[45] further support the conclusion that humans serve as hosts in the transmission cycle. Antibodies to Bwamba virus have been found in birds in South and East Africa.[27,48] A high prevalence of antibodies in monkeys was reported in Uganda.[63] In South Africa, the seroprevalence in humans to Bwamba and Bunyamwera viruses was significantly higher than in monkeys, birds, or domestic livestock.[41]

B. Evidence from Experimental Studies

1. Vectors

After intrathoracic inoculation, Bunyamwera virus has been shown to replicate in *Ae. vexans*,[64] *Ae. canadensis*,[64,65] *Ae. aegypti*,[64-66] *Ae. triseriatus*,[65,66] *Psorophora ferox*,[66] *Cx. pipiens*,[27] and *An. quadrimaculatus*.[27] Oral infection of and transmission by *Ae. aegypti* has been demonstrated for both Bunyamwera[64,65,67] and Ilesha viruses.[67]

Cx. rubinotus has been shown to become infected after feeding on virus and to transmit virus to hamsters.[27] Bwamba virus has been shown to infect *Ae. aegypti*, *An. quadrimaculatus*, and *Cx. pipiens* after intrathoracic inoculation,[27] but susceptibility to oral feeding has not been investigated. *Cx. pipiens* fed on virus-soaked pledgets become infected with but are incapable of transmitting Tataguine virus.[68]

2. Vertebrate Hosts

Wild African rodents (*Arvicanthus abyssinicus* and *Cricetomys gambianus*) experimentally infected with Bunyamwera virus develop viremias sufficient to infect mosquitoes, suggesting that rodents could play a role in natural transmission cycles.[69] Similar results have been reported for *Tadarida* bats.[70] Monkeys develop viremia, fever, and inapparent or mild illness.[71] Germiston virus behaves similarly in experimental animals, including rodents (*Arvicanthus niloticus*[59] and *Tatera brantsi*[72]) and monkeys.[71] Little or no useful information is available regarding experimental infections with Bwamba or Ilesha viruses.

C. Summary

Except for Germiston virus, for which convincing evidence of a rodent-*Cx.* rubinotus cycle is available, the natural history of the viruses under consideration remains obscure. All viruses are mosquitoborne; for Ilesha, Bwamba, and Tataguine viruses, anophelines appear to be the principal vectors, especially endo- and anthropophilic species (*An. gambiae* and *funestus*). A human-*Anopheles* cycle is suggested for Tataguine and possible Bwamba viruses, but a role for wild vertebrate hosts cannot be excluded.

VII. ECOLOGICAL DYNAMICS

A. Macro- and Microenvironment

Bunyamwera virus serological studies show virus activity in association with rivers and riverine forests.[73] HI antibody prevalence in Nigeria was higher in forest and savannah zones than in swampy areas.[36] Virus activity in the Central African Republic is associated with gallery forests in the moist savannah zone.[49,50]

Ilesha antibody prevalence in the endemic area of Nigeria appears to be highest in the savannah than in the plateau and rain forest, and the virus seems to be more active in rural than in urban communities.[73] Germiston virus isolations have been associated with irrigated areas, where cattle have a high antibody prevalence. Tataguine antibodies have been found in highest prevalence in the derived savannah vegetational zone.[45]

VIII. PREVENTION AND CONTROL

Vaccines are not available. Because of their relatively low pathogenicity and lack of economic impact, specific efforts have not been developed to reduce the incidence of these diseases.

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Table 1
ORIGIN OF VIRUS ISOLATIONS (ISOL) AND ANTIBODIES (AB) AGAINST FIVE ARBOVIRUSES (BUNYAMWERA, BWAMBA, GERMISTON, ILESHA, AND TATAGUINE)

	BUN		BWA		GER		ILE		TAT	
Origin	Isol	AB	Isol	AB	Isol	AB	Isol	AB	Isol	AB
Man	+[a]	+	+	+	+	+	+	+	+	+
Chimpanzee	ND[b]	+	ND	ND	ND	ND	ND	ND	ND	ND
Monkeys	ND	+	ND	ND	ND	ND	ND	ND	ND	ND
Domestic animals	ND	+	ND	ND	ND	ND	ND	ND	ND	ND
Rodents	ND	+	ND	ND	+	+	ND	ND	ND	ND
Birds	ND	+	ND	+	ND	ND	ND	ND	ND	ND
Donkeys	ND	ND	ND	+	ND	ND	ND	ND	ND	ND
Cattle	ND	ND	ND	ND	ND	+	ND	ND	ND	ND
Hamster (sentinel)	ND	ND	ND	ND	+	ND	ND	ND	ND	ND
Arthropods	+	+	+	+	+	+	+	+	+	+

[a] Positive isolation or presence of antibodies.
[b] ND, no data.

Job 4386SSSS29 Galley — — — — — — — — 12-21-87 11-06-07

Table 1

ORIGIN OF VIRUS ISOLATIONS (ISOL) AND ANTIBODIES (AB) AGAINST FIVE ARBOVIRUSES (BUNYAMWERA, BWAMBA, GERMISTON, ILESHA, AND TATAGUINE)

	BUN		BWA		GER		ILE		TAT	
Origin	Isol	AB	Isol	AB	Isol	AB	Isol	AB	Isol	AB
Man	+[a]	+	+	+	+	+	+	+	+	+
Chimpanzee	ND[b]	+	ND	ND	ND	ND	ND	ND	ND	ND
Monkeys	ND	+	ND	ND	ND	ND	ND	ND	ND	ND
Domestic animals	ND	+	ND	ND	ND	ND	ND	ND	ND	ND
Rodents	ND	+	ND	ND	+	+	ND	ND	ND	ND
Birds	ND	+	ND	+	ND	ND	ND	ND	ND	ND
Donkeys	ND	ND	ND	+	ND	ND	ND	ND	ND	ND
Cattle	ND	ND	ND	ND	ND	+	ND	ND	ND	ND
Hamster (sentinel)	ND	ND	ND	ND	+	ND	ND	ND	ND	ND
Arthropods	+	+	+	+	+	+	+	+	+	+

[a] Positive isolation or presence of antibodies.
[b] ND, no data.

DUPLICATE

Job 4386SSSS29 Galley 1x— — — — — — — — 12-21-87 11-06-07

Job 4386SSSS30 Galley — — — — — — — — 12-21-87 11-09-29

Table 2

SUSCEPTIBILITY OF CELL CULTURE SYSTEMS FOR FIVE BUNYAVIRUSES

Spinners

Spinner

	BUN		BWA		GER		ILE		TAT	
Cell system	Eff[a]	Day[b]	Eff	Day	Eff	Day	Eff	Day	Eff	Day
Chick embryo	CPE	2—5	CPE	2	PLQ	3	PLQ	4—5	ND[c]	ND
VERO	PLQ	5	PLQ	5	PLQ	3	PLQ	6	PLQ	6
BHK-21	CPE	4	CPE	2	PLQ	2	ND	ND	ND	ND
LLC-MK_2	PLQ	4	PLQ	3	PLQ	4	PLQ	4	NP[d]	NP

Note: Results given can vary with the virus passage history.

[a] Eff, effect in cell culture (CPE, cytopathic effects; PLQ, plaques).
[b] Day, mean of days after isolation.
[c] ND, no data.
[d] NP, no plaque.

Job 4386SSSS30 Galley 1x— — — — — — — — 12-21-87 11-09-29

Portrait page

Table 3

DETECTION OF IMMUNITY TO BWAMBA (BWA), BUNYAMWERA (BUN), ILESHA (ILE), GERMISTON (GER), AND TATAGUINE (TAT) VIRUSES IN HUMANS: ANTIBODY PREVALENCE AND VIRUS ISOLATIONS

	BWA	BUN	ILE	GER	TAT
North Africa					
Algeria	ND*	0.0	0.0	ND	ND
Egypt	0.0[b]	0.9[c]	ND	ND	ND
Libya	ND	0.0	ND	ND	ND
Morroco	ND	0.0	0.0	ND	ND
Tunisia	ND	0.0	0.0	ND	ND
West Tropical Africa					
Benin	ND	3.0	ND	ND	ND
Burkina faso	ND	17.0	ND	ND	*
Bameroon	*[d]	8.0*	*	ND	*
CAR	*	24.0*	*	ND	*
Chad	ND	0.0	ND	ND	ND
Gambia	+[e]	+	ND	ND	ND
Ghana	ND	+	6.5*	ND	ND
Guinea	ND	*	ND	ND	ND
Guinea-Bissau	43.0	11.0	3.0	ND	ND
Ivory Coast	ND	14.0	ND	ND	ND
Liberia	ND	19.0	ND	ND	ND
Mali	ND	2.0	ND	ND	ND
Niger	ND	3.0	ND	ND	ND
Nigeria	33.0—40.0*	0.0—23.0*	27.0—45.0*	10.0	26.0—61.0*
Senegal	ND	19.0*	*	ND	*
Sierra Leone	ND	•	ND	ND	ND
Togo	ND	0.5	ND	ND	ND
East Tropical Africa					
Ethiopia	+	1.8—19.2*	•	+	ND
Somalia	ND	*	ND	ND	ND
West Equatorial Africa					
Congo	ND	7.0—25.0	ND	ND	ND
Equatorial Guinea	+	+	ND	ND	ND
Gabon	ND	8	ND	ND	ND
Rwanda	ND	0.0—10.0	ND	ND	ND
East Equatorial Africa					
Kenya	*	+	•	*	ND
Tanzania	75.0*	11.1	ND	ND	ND
Uganda	44.0* 37.0*	*	*	ND	ND
Southern Tropical Africa					
Angola	+	52.0	ND	28.0	ND
Madagascar	ND	0.0—6.5	ND	ND	ND
Mozambique	24.7*	24.1*	ND	ND	ND
Zimbabwe	ND	15.9	ND	2.3	ND
Austral Africa					
Botswana	3.3	53.3	ND	90.1	ND
Namibia	ND	42.1	ND	56.0	ND
SAR	0.0—80.0	0.0—45.7*	ND	1.4*	ND

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4386-31-1

[a] ND, no data.
[b] Sera tested, but no evidence for antibody.
[c] Prevalence (%) of viral antibodies.
[d] Virus isolation.
[e] Presence of viral antibodies; prevalence not defined.

Table 4
EVIDENCE OF VIRUS CIRCULATION IN WILD AND DOMESTIC VERTEBRATE HOSTS

	Virus isolation		Serological evidence	
			Wild animals	Domestic animals
BWA	No		*Arvicanthys niloticus* *Boedon fuliginosus* *Varanus niloticus* *Turtur ater* Monkeys	Goast, sheep, cattle
BUN	No		Chimpanzee, cattle, sheep, rodents, nonhuman primates, goats	Goats, sheep
ILE	No		No	No
GER	*Herpestes ichneumon* *Dasymys incomtus* *Rattus rattus* *Lophuromys* spp. *Arvicanthus niloticus*		*Arvicanthus niloticus* Horses	Goats, sheep, cattle
TAT	No		No	No

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