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ECOLOGICAL INTERACTION OF WILDLIFE, MAN, AND A VIRUS OF THE VENEZUELAN EQUINE ENCEPHALOMYELITIS COMPLEX IN A TROPICAL FOREST

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Abstract: Venezuelan equine encephalomyelitis (VE) virus was isolated 18 times from blood of *Oryzomys* in tropical forests near Belem, Brazil. Rainfall, *Culex* population levels, and nonimmune *Oryzomys* population levels were analyzed during 1962 to 1964 and 1968 to 1970 for association with transmission of VE virus. A positive correlation between VE virus transmission in *Oryzomys* and the abundance of nonimmune animals was determined. Infection of man with rodent-associated viruses such as VE probably occurs during crepuscular hours when *Culex (Melanoconion)* mosquitoes and rodents are active.

INTRODUCTION

Venezuelan equine encephalomyelitis (VE) is a mosquito-borne disease of man and horses which periodically breaks out in South and Central America. There are several VE subtypes, including Mucambo and Pixuna from northern South America and a more recently recognized virus from Florida, U.S.A.[3,7] These form the VE complex. While VE virus itself is the member of the complex which causes recognized human and equine epidemics, the other members infect man sporadically and serve as models in our attempt to understand the reservoir state of classical VE. Mucambo is the VE subtype which is enzootic in the rodent population of the Amazon region of Brazil.

Interaction of VE virus with the rodent host and the influence of rainfall and mosquito population levels on virus transmission are illustrated from data collected by the Belem Virus Laboratory, Brazil, in the nearby Utinga Forest and in the Aura Forest, about 3 km distant from Utinga.

METHODS

In the Utinga Forest, wild rodents were captured, bled, marked, released, and periodically recaptured during the period July 1962 to November 1964. Altogether, 8750 samples were obtained from 1389 animals. Approximately half the animals were *Proechimys guyannensis oris* Thomas, the spiney rat, and a further third were *Oryzomys capito goeldii* Thomas, the rice rat. Animals were tested for viremia at each capture and, when possible, for hemagglutination-inhibiting (HI) antibody at 3-week intervals by methods previously described.[6]

At Aura, where a similar mammal recapture program was carried out from August 1968 through December 1970, 1648 samples were obtained from 820 animals. Of these animals, one third were *Proechimys* and one third were *Oryzomys*.

Three parameters were analyzed for their effect on VE virus transmission in *Oryzomys:* 1) rainfall, 2) *Culex* mosquito prevalence, and 3) relative abundance of nonimmune *Oryzomys*.

Rainfall data from 1962 through 1964 were from the government weather station near, but not in, Utinga Forest and must be considered an approximation of actual rainfall in the forest. Data from 1968 through 1970 were collected in Aura Forest at the actual site of mammal trapping.

The *Culex* mosquito prevalence from 1962 through 1964, based on the numbers of *Culex* aspirated from Causey hoods baited with mice and exposed daily in Utinga Forest, was used as a measure of *Culex* population. The collections were early morning samples and consisted almost entirely of mosquitoes of the subgenus *Melanoconion* which are rodent-feeders. The *Culex* prevalence from 1968 through 1970 was estimated from captures of *Culex* in blower traps operated in Aura Forest 4 days each month with mouse bait.

The relative abundance of nonimmune *Oryzomys* was an estimate of relative population levels calculated by Petersen's formula[5] to which was added the relative number of trapped juveniles whose birth dates could be retrospectively estimated by their weight when first captured. The percentage of animals with HI antibody was subtracted to arrive at the relative abundance of nonimmunes.

Petersen's formula was modified in an attempt to compensate for rising and declining populations with varying influx of young animals. Young animals were shown by grid trapping in Utinga Forest to have a lesser range than older animals. The trap spacing was such that *Oryzomys* were usually 3 to 4 months old when they first appeared in the traps. Age was estimated by first constructing age-weight curves using data on recaptured juveniles, and then by determining the birth date of juvenile animals on the basis of weight at initial capture.

RESULTS

Initial HI studies established that both *Proechimys* and *Oryzomys* had antibody, and that naturally infected viremic rodents developed HI antibody which persisted for life.[6] Both *Proechimys* and *Oryzomys* were thus considered possible reservoirs in Utinga Forest and it is assumed, since HI antibody persists, that lack of HI antibody indicates susceptibility to infection.

Subsequently, in the Utinga and Aura Forest studies reported here, VE virus was isolated 18 times from *Oryzomys* and only seven times from all other animals. Viremia titers in *Oryzomys* captured in Utinga Forest ranged between 1.2 and 5.0 log LD_{50}/0.02 ml (Table 1). This high frequency of isolation from *Oryzomys* and the substantial viremia titers implicate *Oryzomys capito* as a prime suspect in the search for vertebrate hosts in the transmission cycle of VE virus.

Figure 1 shows the estimated birth dates of juvenile *Oryzomys,* 1962 to 1964. Most animals were born in the first 4 months of each year. The animals estimated to have been born during the prior 3 months were added to the relative population as determined by Petersen's formula prior to subtracting the percentage of immune animals. The resulting relative abundance of nonimmune *Oryzomys* is still a crude estimate, but represents the most accurate determination the data allow.

Figure 2 shows the associations of rainfall, *Culex* population levels, and nonimmune *Oryzomys* population levels to VE virus infections in Utinga Forest. Peak rainfall occurred during the first 4 months of the year, and *Culex* prevalence correspondingly increased shortly after the beginning of the rainy season.

There was a large increase in nonimmune *Oryzomys* population in early 1963. This reflected the high birth rate between January and April. The increase in susceptibles correlated with a hyperendemic period of VE virus activity.

In 1964, although rainfall and *Culex* populations were apparently ample, there were relatively fewer nonimmune *Oryzomys* and there was less evidence of VE virus transmission to *Oryzomys.*

Figure 3 illustrates for Aura Forest the dynamics of VE virus transmission. Rainfall in 1969 and 1970 was as usual followed by large increases in *Culex* populations. During the latter half of 1968 and the first half of 1969, as had been observed in 1963 in Utinga, there was an ample nonimmune *Oryzomys* population, reflecting a high birth rate. Transmission of VE virus to *Oryzomys* was observed. In 1970, however, in spite

TABLE 1. VE virus isolations from animals of Utinga Forest, Belem, Brazil, July 1962 to November 1964.

Animal	Date	Titer of viremia, log LD_{50}/0.02 ml
Proechimys guyannensis	31 Aug. 62	Not done
Metachirops opossum	18 Sept. 62	Not done
Oryzomys capito	12 Mar. 63	Not done
Oryzomys capito	21 May 63	Not done
Oryzomys capito	17 June 63	3.0
Oryzomys capito	19 June 63	>3.5
Oryzomys capito	29 July 63	Not done
Oryzomys capito	30 July 63	2.0
Oryzomys capito	2 Aug. 63	Not done
Oryzomys capito	23 Aug. 63	3.6
Oryzomys capito	18 Oct. 63	5.0
Oryzomys capito	29 Jan. 64	3.7
Oryzomys capito	7 Feb. 64	1.2
Proechimys guyannensis	27 Aug. 64	<1.0

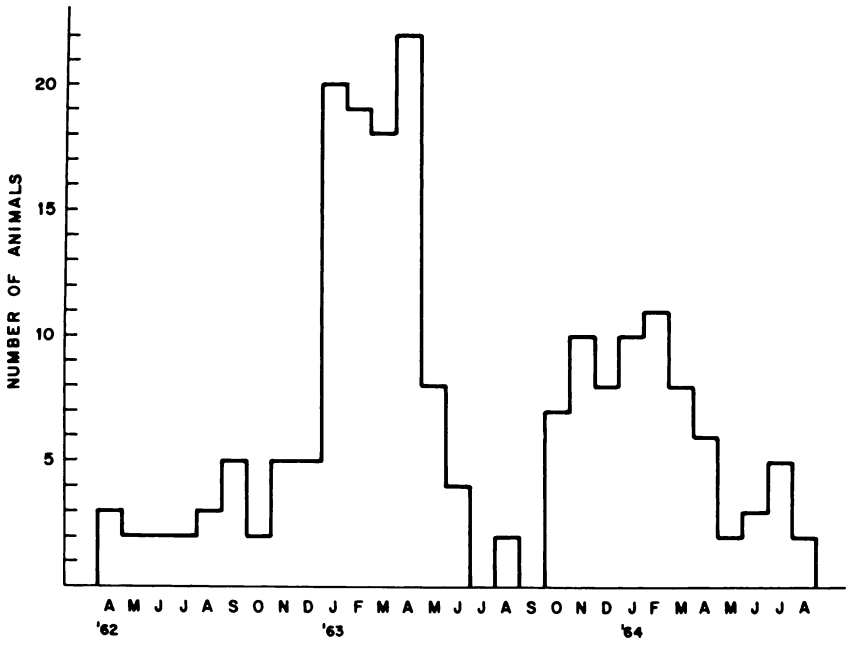

FIGURE 1. Estimated birth dates of juvenile **Oryzomys capito** captured between June 1962 and November 1964.

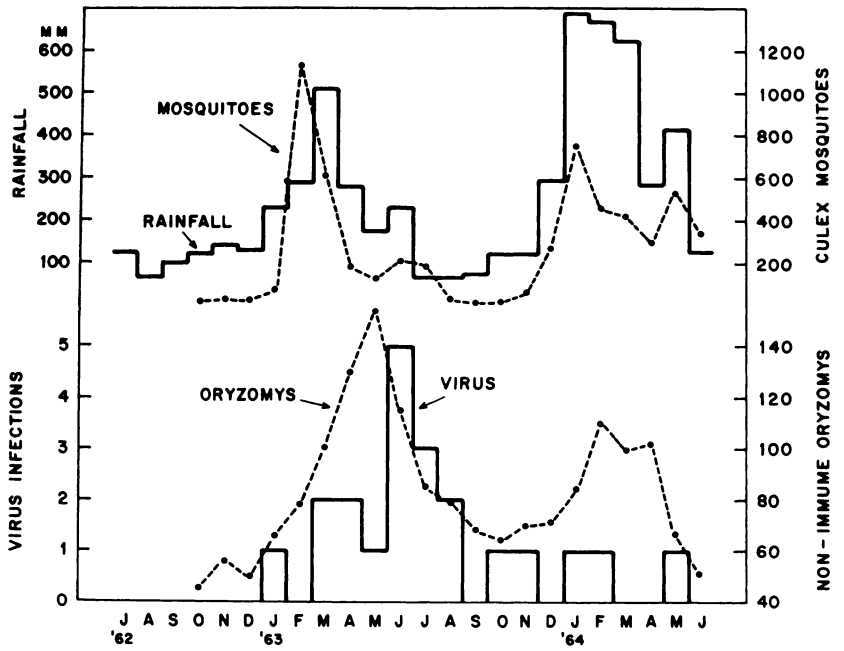

FIGURE 2. Relationship of rainfall, **Culex**, and **Oryzomys** to VE virus infections in Utinga Forest.

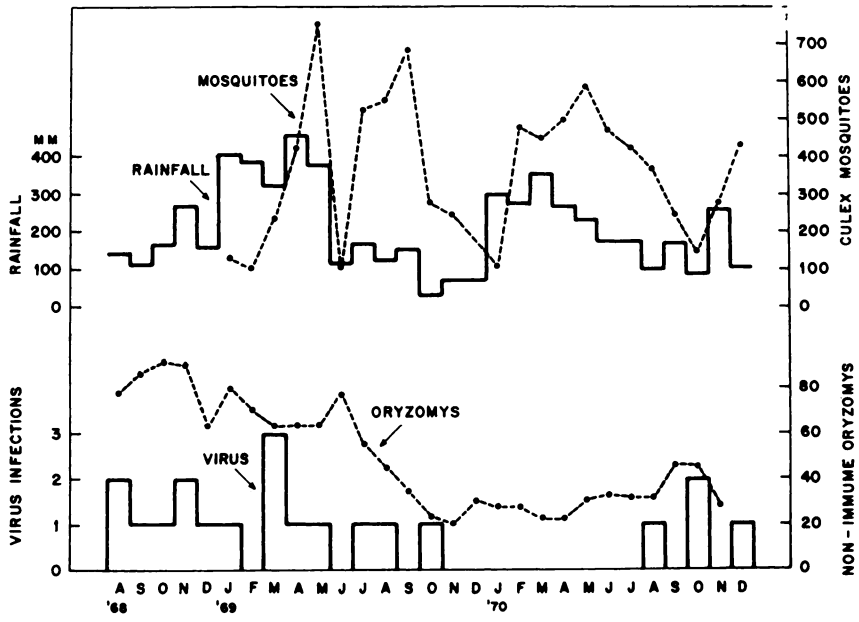

FIGURE 3. Relationship of rainfall, **Culex**, and **Oryzomys** to VE virus infections in Aura Forest.

of normal rainfall and *Culex* mosquito populations, the number of nonimmunes had fallen to very low levels, and VE virus transmission was not detected until very late in the year with an influx of young animals and a rise in the nonimmune *Oryzomys* population.

DISCUSSION

There appears to be a positive correlation between VE virus transmission in the *Oryzomys* population and the abundance of nonimmune animals. These data are consistent with the observations in 1964 in Trinidad where a rodent population crash was associated with cessation of VE virus transmission in spite of continued presence of *Culex* mosquitoes.[4]

It should be noted, however, that while the *Culex* mosquito level may be high without transmission being detected if the *Oryzomys* population is low, the reverse may also be true; i.e. transmission may not occur if the *Culex* level is low and the *Oryzomys* population is high. *Culex* levels dropped precipitously for a short period in June 1969 (possibly because of lack of rainfall) and in spite of ample nonimmunes, there is a hint that VE virus transmission ceased.

Humans are infected with VE virus in the Amazon region.[2] The precise mechanism of infection by mosquitoes is not known, although proximity to tropical rain forest either because of residence or occupation clearly increases the risk of infection.[1,2] The normally rodent-feeding *Culex (Melanoconion)* are likely vectors since they readily attack human beings during crepuscular hours when man enters the forest.

Field workers of the Belem Virus Laboratory have been infected with rodent-associated viruses (although not VE). One such field worker had been employed for 5 years capturing mosquitoes during daytime. When switched to crepuscular captures in 1964, he was immediately infected with Murutucu virus and in another month with Catu virus, both of these being rodent-associated as is VE virus. In another instance, an entomologist and two associates working in the Aura Forest at dawn in 1970, were infected with Caraparu, Oriboca, and Catu viruses, respectively, all rodent-associated viruses like VE. One would predict by analogy and by what is known of the forest hosts and vectors that VE virus also is transmitted to man during crepuscular hours by *Culex (Melanoconion)* mosquitoes in the Amazon region.

VE virus has been used as an example of an agent which infects wildlife and which is directly dependent in multiple facets of its epidemiology on the ecological factors governing the tropical rain forest. There is scant evidence in 16 years of study of rodents with this infection, of overt disease in wildlife.[8] Although this is a wildlife disease conference, it is hoped that apologies are not necessary for discussing infection which is benign in wildlife, because in the final analysis this is probably the rule rather than the exception with wildlife parasites.

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