JOURNAL OF MEDICAL ENTOMOLOGY

J. Med. Entomol. Vol. 20, no. 4: 347–364 21 July 1983

Published bimonthly by Department of Entomology, Bishop Museum, Honolulu, Hawaii, USA. Editorial committee: F.J. Radovsky, Executive Editor, JoAnn M. Tenorio, Editor, R.C. Axtell, B.N. Chaniotis, M.M. Crystal, D.M. Davies, R. Domrow, K.C. Emerson, A. Fain, H. Hoogstraal, J. Kitzmiller, H. Kurahashi, L.E. LaChance, L.A. Magnarelli, C.J. Mitchell, M. Nadchatram, W.A. Nelson, M.W. Service, R. Traub, J.E.M.H. van Bronswijk. Devoted to all branches of medical entomology from the world standpoint, including systematics of insects and other arthropods of public health and veterinary significance.

REVIEW ARTICLE

BITING MIDGES (DIPTERA: CERATOPOGONIDAE) AND HUMAN HEALTH[1]

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Abstract. The relationship of biting midges (Ceratopogonidae) to human health is reviewed and discussed. The treatment is broad and contains historical, distributional, and clinical information concerning the diseases in question, a comprehensive discussion of the vectors, including non-ceratopogonids, and epidemiological data. The emphasis is on recent work, with special attention to current knowledge of the transmission of the filarial parasite *Mansonella ozzardi* and Oropouche virus.

The period since Kettle (1965) published a review of Ceratopogonidae as vectors of human and animal disease has witnessed significant advances in knowledge in both fields. To survey both human and veterinary aspects now seems outside the scope of a single article, and we have confined ourselves here to the former. In the discussion we have included non-ceratopogonid vectors, which will make the paper useful to a larger readership than would be reached if the concern were strictly with ceratopogonids. Epidemiological aspects of some diseases, mansonellosis for example, could not be discussed intelligibly without consideration of the *Simulium* spp. that also transmit the parasite. Both clinical and historical information is also briefly included.

At the last major revision (Wirth et al. 1974), there were 3870 species of Ceratopogonidae assigned among 60 genera. Only 4 of these, *Culicoides, Leptoconops, Forcipomyia,* and *Austroconops,* are known to feed on man, or indeed on any warm-blooded vertebrate. *Culicoides* is much the most important genus with respect to both animal and human health. Species of *Leptoconops* are important pests of man and bite viciously in many parts of the world. One species has recently been shown capable of supporting development of a filarial parasite (discussed later), but it is as pests that *Leptoconops* spp. are principally important (Rioux et al. 1968, Duval 1971, Linley & Davies 1971, Laurence & Mathias 1972). Within the genus *Forcipomyia,* the subgenera *Lasiohelea* and *Dacnoforcipomyia* contain species reported attacking man, but neither are recorded as vectors of human pathogens. *Austroconops* is a rare monospecific genus (Wirth & Lee 1958) restricted to the southwest of Western Australia and is not known to be involved in transmission of disease organisms.

The disease organisms transmitted to man by ceratopogonids fall into 2 groups: Nematoda (Filarioidea) and viruses. We deal with the filarial parasites first.

NEMATODA (FILARIOIDEA)

*Dipetalonema (Acanthocheilonema) perstans**

This filarial worm is indigenous to Africa. It is widespread throughout all the sub-Saharan and central areas of the continent, extending from Gabon and Angola in the west to Kenya and Mozambique in the east. *Dipetalonema perstans* does not appear to occur in southern Africa. Originally introduced with the slave trade, the parasite has entered the Americas and occurs along the northern portion of South America from Colombia through Venezuela, Guyana, Surinam, and French Guiana. It has also been found in Mexico (Yucatán), Trinidad, and some of the other Caribbean islands. *Dipetalonema perstans* does not occur in the

[1] Florida Agricultural Experiment Station Journal Series No. 4085. The views of the authors do not purport to reflect the positions of the Department of the Army or the Department of Defense.

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* See Addendum

Pacific area or Asia. Complete distributional information and associated literature may be found in Hawking (1977, 1979), supplemented by a recent report of heavy infections with *D. perstans* in Gabon (Richard-Lenoble et al. 1980).

Although infecting large numbers of people, *D. perstans* has generally been regarded as nonpathogenic. This description persists, perhaps because clinical symptoms have been difficult to associate specifically with the presence of the organism and symptoms have often been inconsistent, even when *D. perstans* was known to be present with or without other complicating infections. Human populations studied in endemic areas have often been infected with several parasites. However, while variation between individuals may be very great, certain serious symptoms are undoubtedly associated with *D. perstans* in some individuals (Holmes et al. 1969). Based upon the most reliable documentations, in the sense that they identify symptoms specifically related to the presence of *D. perstans* (Strohschneider 1956, Adolph et al. 1962, and particularly Wiseman 1967), the principal clinical manifestations are eosinophilia, pruritis, transient swelling, liver pain, joint pain, fever, and fatigue. There are 2 reports of possible fatalities (Bourgnignon 1937, Foster 1956). Two cases of suspected *D. perstans* microfilariae in human cerebrospinal fluid (Dukes et al. 1968) were later considered more likely to be zoonotic infections with a monkey filaria, *Meningonema* (Hawking 1977). Particularly serious human symptoms were reviewed by Hawking (1977) from Rhodesia (Gelfand & Bernberg 1959, Baker et al. 1967), but the evidence, especially in the 2nd study, does not seem very convincing and Holmes et al. (1969) later found Rhodesian cases to be clinically mild. Other references to the pathogenicity of *D. perstans* will be found in the various papers cited in this section.

The New World vectors of the parasite have not been identified specifically, but there are many anthropophilic species of *Culicoides*. Wirth & Blanton (1973), for example, named 17 species collected from human bait in the Amazon region. In Africa, changing taxonomic views, when combined with biological information and recent work on species distribution and larval habitats, raise questions of vector identity that cannot as yet be completely answered. Early work establishing the originally recognized vectors of *D. perstans* (and *D. streptocerca*) has been described succinctly by Kettle (1965). Involved were studies by Sharp (1927, 1928), Chardome & Peel (1949), Henrard & Peel (1949), Hopkins (1952), Hopkins & Nicholas (1952), and Duke (1956). *Culicoides austeni* Carter, Ingram & Macfie, *C. grahamii* Austen, and *C. inornatipennis* Carter, Ingram & Macfie were all shown to be capable of supporting development of microfilariae to the infective (3rd) stage, but *C. grahamii* was not as effective a vector as the other two. The dispersal of *C. austeni* and *C. grahamii* from larval habitats in rotting banana material was studied by Nicholas (1953). Of these vectors, the identities of *C. grahamii* and *C. inornatipennis* remain uncontroversial, but that of *C. austeni* is problematical. Insects collected as *C. austeni* for studies of vector competence in the work by Nicholas (1953) were taken either as adults and tested for freedom from parasites, or in the immature stages from rotting banana stumps and litter (Hopkins & Nicholas 1952). The work was in West Africa, mostly in the Cameroons, and the *Culicoides* species were identified at that time from material in the British Museum (Natural History). In identifying their specimens from Nigeria and the Cameroons, Nicholas et al. (1955) were confused by the possible synonymy of *C. austeni* with *C. milnei* Austen on examining the type series of both species. They preferred to retain the name *C. austeni.* However, on looking through the same specimens in conjunction with their work on the *Culicoides* of East Africa, Khamala & Kettle (1971) recognized 4 species among the *austeni* and *milnei* material (*C. milnei, C. austeni, C. vitshumbiensis* Goetghebuer, and *C. hortensis* Khamala & Kettle). The difficulty of the situation thus becomes evident. That banana litter formed the larval habitat does not help clarify the situation because, if pertinent literature of the past 20 years or so is abstracted (about 12 publications dealing with larval habitats and zonal distribution), there is no reference to *any* of these 4 species having been taken from banana debris. Records of species reared from specific habitats admittedly are not abundant, but some surveys have been made, especially in Kenya and Nigeria.

The identity of vectors is further complicated by the observations by Murphy (1961). In The Gambia, he found 2 forms of *C. austeni.* The most common form was found in mangrove swamps, was autogenous, and mated without flight; the other form was taken inland from banana litter, was anautogenous, and mated only in flight (in swarms). Murphy also found a 2nd, darkly colored species, unidentified but very closely related to *C. austeni.* It was abundant in mangrove swamps and was also autogenous.

At present, the analysis that most nearly resolves these problems of identity is that by Cornet et al.

(1974), who suggest the following interpretation. The species described as *C. austeni* by Khamala & Kettle (1971) is, they believe, *C. zuluensis* De Meillon. The species Cornet and his collaborators recognize as *C. austeni* is restricted to a coastal distribution in mangrove swamps and is one of the autogenous species collected by Murphy (1961). The other, darker autogenous species mentioned by Murphy is *C. obscuripennis* Clastrier & Wirth (Clastrier & Wirth 1961). The inland form from banana litter, studied by Nicholas et al. (1955) and Murphy (1961), is, they suggest, *C. hortensis* or *C. krameri* Clastrier or a mixture of the two. Since *C. austeni* as recognized by Cornet et al. (1974) is autogenous, it seems unlikely that it has much potential as a vector.

Another report of possible concern is that by Phelps & Mokry (1976). Working in Rhodesia, where *D. perstans* is known to be present (Baker et al. 1967), they found filarial nematodes in the head capsules of *C. pycnostictus* Ingram & Macfie and *C. ravus* De Meillon among specimens of 15 species dissected. However, the filariae could have been parasites of birds or other animals, especially in the case of *C. pycnostictus,* which has been shown to be primarily an avian feeder (Nevill & Anderson 1972).

In conclusion, the species that probably are vectors in Africa are the following: *C. grahamii, C. inornatipennis,* and perhaps *C. milnei, C. hortensis, C. krameri,* and *C. vitshumbiensis.* Less likely, but possible, vectors are *C. pycnostictus* and *C. ravus.* There is considerable need to intensify collection and taxonomic study of these and other African species, in combination with extended investigations of their habits and habitats. Difficulties currently arising from an incomplete knowledge of the vectors are compounded by the recognition that *D. perstans* itself is probably a complex of species (Hawking 1977).

*Dipetalonema streptocerca**

This parasite is restricted to Africa, where its distribution, although incompletely known, includes Ghana, Nigeria, and Zaire (Hawking 1977). It infests the skin and is generally less intense in its prevalence than is *D. perstans. Dipetalonema streptocerca* seems legitimately to be considered nonpathogenic for man, with symptoms of the skin perhaps occasionally seen (Colbourne et al. 1950). *Dipetalonema streptocerca* can complete development in *C. grahamii* females that are concurrently supporting development of *D. perstans* larvae (Duke 1954). Ancillary information germane to the role of *C. grahamii* as a vector has recently been provided by Auriault (1977a,b), who has studied the biting and gonotrophic cycles.

Culicoides grahamii has remained a well-defined species taxonomically (Khamala & Kettle 1971). It has been collected in the larval stage from banana litter (Hopkins 1952, Nicholas et al. 1955), but judging from more recent literature, *C. grahamii* is capable of exploiting a wide range of habitats and was collected in all ecological zones recognized in surveys in East Africa (Khamala 1971) and Nigeria (Dipeolu 1976).

*Mansonella ozzardi**

This worm is indigenous to the Americas and occurs in Argentina, Bolivia, Colombia, Brazil (Amazon basin), Guyana, Surinam, French Guiana, Venezuela, Trinidad, and several other islands in the West Indies. More detailed distributional information is given by Hawking (1979) and for the Antilles especially by Ripert et al. (1977).

Clinically, infection with *M. ozzardi* is thought not to produce significant pathology, although Marinkelle & German (1970) indicate symptoms, possibly attributable to infection, encountered among Amerindians in Colombia. The chief complaint was severe articular pain, and varying degrees of eosinophilia also were present. Pruritis observed in this population was probably not due to *M. ozzardi,* since histological examination of infected skin (Ewert et al. 1981) showed virtually no evidence of cutaneous disease.

The discovery of microfilariae of *M. ozzardi* in the skin has been quite recent. The parasite was previously assumed to be present only in the blood and diagnosis was made from aliquots of venous or capillary blood. However, Moraes (1974), surveying newly discovered cases of onchocerciasis in Brazilian Amerindians, found *M. ozzardi* in bloodless skin snips and pointed out the possibility of erroneous diagnosis in the absence of confirmed identifications of the parasites (Moraes 1976). The concern for positive identification, where distributions of *Onchocerca* and *Mansonella* overlap, has been echoed by Ewert et al. (1981). The presence of *M. ozzardi* in the skin has been confirmed in other cases (Nathan et al. 1978, Nathan 1979, Lightner et al. 1980, Raccurt et al. 1980); in fact, 2 of 170 cases seen by Lightner et al. (1980) were positive by skin biopsy only, as were 2 of 24 cases

* See Addendum.

in the study by Nathan (1979). Raccurt et al. (1980) discuss why diagnosis by blood samples is the preferred method as surveys continue and until more is known of the presence and behavior of the microfilariae in various tissues.

Even though the microfilariae are present in the skin, they remain mostly in the capillaries of the dermal papillae (Moraes et al. 1978, da Silva et al. 1978, Raccurt et al. 1980), although a few have been noted in the perivascular spaces, the dermal interstitium, and epidermis (Ewert et al. 1981). Nelson & Davies (1976) suggested that the distribution of microfilariae in the skin might be adapted to the preferential feeding sites of the vectors, and Nathan (1979) tested this possibility in Trinidad where the vector *Culicoides phlebotomus* (Williston) (Nathan 1978) feeds predominantly on the legs and ankles. Skin snips from shoulder, hip, and calf showed no consistent pattern of distribution and no sign of heavier infestation in the legs. Nathan & Raccurt (1979) did find higher concentrations of microfilariae in capillary blood from the ear lobe than from the finger. The significance of this was not clear, but for survey purposes indicated the need for standardization of a site for capillary blood sampling. Attempts have been made to treat infections with diethylcarbamazine, but with no apparent benefit (Bartholomew et al. 1978).

In recent years, important and more detailed work has been done on the epidemiology of mansonellosis, principally in 3 localities: Haiti, Trinidad, and the Amazon regions of Brazil and Colombia. In Haiti and Trinidad the vectors are ceratopogonids, whereas in the Amazon, simuliids have been incriminated. The view has been expressed for some time and by several authors (e.g., Nelson & Pester 1962, Shelley & Shelley 1976, Hawking 1979) that the Brazilian and West Indian forms of *M. ozzardi* may represent different species.* For present purposes, studies with the West Indian form will be reviewed first. Illustrations that collate data from both regions are presented so that direct comparison is possible.

In Haiti, infection is limited to, but widely spread throughout, coastal areas, coincident with the mangrove-swamp habitat of the principal vector, *Culicoides furens* (Poey) (Ripert et al. 1977, Raccurt & Lowrie 1979). *Culicoides furens* was the first ceratopogonid to be incriminated as a vector of *M. ozzardi* by Buckley (1933, 1934) working in St. Vincent. Raccurt & Lowrie (1981) have also shown that both *Culicoides barbosai* Wirth & Blanton and *Leptoconops becquaerti* (Kieffer) are capable of supporting development of the microfilariae to the infective stage, but for reasons discussed shortly, these species seem unlikely to play a significant role as vectors. Mansonellosis occurs along the north coast of Trinidad and is particularly prevalent in the westernmost communities (Nathan et al. 1979). The vector, *Culicoides phlebotomus,* breeds in sandy rivers where they cross the beaches and is the only *Culicoides* so far found responsible for transmission. *Leptoconops becquaerti* also supported development of the parasite to the 1st larval stage, but the experiments were incomplete and insufficient to assess its role as a secondary vector.

Infected human populations in both areas are essentially similar; both are coastal fishing communities very near the habitat of the particular vector species. Nathan et al. (1979), in a survey of 10 communities in Trinidad, found 214 of 4489 persons (4.8%) of both sexes with blood positive for microfilariae, while in Haiti the overall infection rate was considerably higher, with 188 of 1165 (16.1%) positive (Raccurt et al. 1980). The distribution of the parasite among different age segments of the population was similar in the 2 localities (Fig. 1).

The microfilarial count increased with age and was higher in males than in females, with especially pronounced differences between individuals aged from the late twenties to late fifties (Fig. 1). The reasons for these age and sex differences are controversial. Nathan et al. (1979) argue, plausibly, that Trinidadian men are exposed to infective bites as they tend their boats on the beaches in the early morning, when *C. phlebotomus* is very active (Fig. 3). Women and children, avoiding the insects and remaining in the village, are supposedly less exposed. However, in the Haitian communities, where spatial relationships between vector and human populations seem even more intimate, men and women are thought to be equally exposed during peak activity periods (Fig. 3) of the vector, *C. furens,* at sunrise and sunset (Raccurt et al. 1980). Most transmission may, in fact, occur at night during sleep (Lowrie & Raccurt 1981). This is possible not only because the vector is active throughout the night (Fig. 3), but because *C. furens* will enter houses in search of blood (Porter 1959).

After microfilariae have been ingested in the blood meal, they enter the abdomen of the insect, begin to develop through 3 stages (Buckley 1934), and ultimately migrate to the thorax and hence to the head capsule. Buckley (1934) found that 6 days were needed for maturation in *C. furens* at 27–30

* See Addendum.

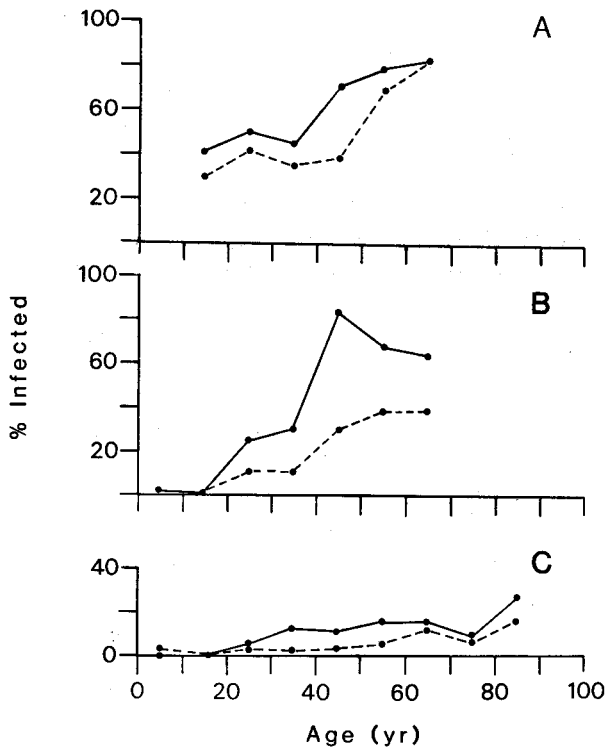

FIG. 1. Infection rates, by age, of *Mansonella ozzardi* in human populations: **A**, Colombia (Lightner et al. 1980); **B**, Haiti (Raccurt et al. 1980); **C**, Trinidad (Nathan et al. 1979). Solid line = ♂, broken line = ♀.

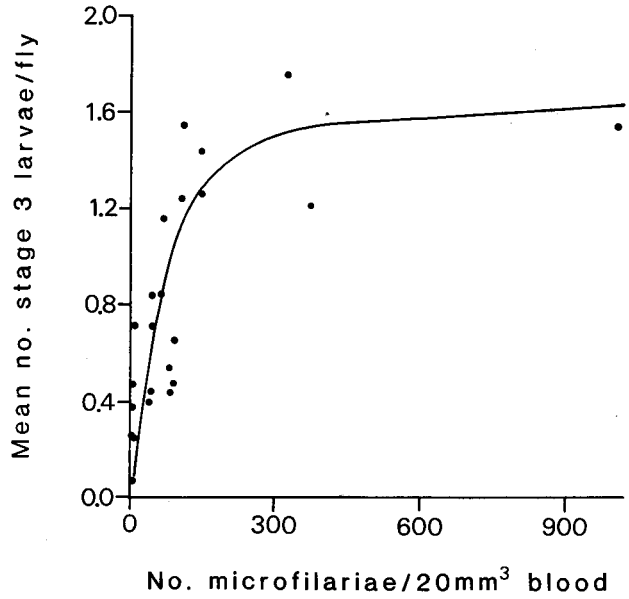

FIG. 2. Mean numbers of infective (stage 3) *Mansonella ozzardi* larvae from *Culicoides furens* ♀ fed on volunteers showing different levels of microfilaremia. Data from Lowrie & Raccurt (1981).

°C, and the same period was observed in *C. phlebotomus* at 25–31 °C (Nathan 1981a). Lowrie & Raccurt (1981) recorded 9 days for *C. furens* in Haiti, but their experimental temperature was probably somewhat lower. Based on the duration of the gonotrophic cycle, Nathan (1981a) constructed an interpretation of the feeding and oviposition cycles to show that transmission to the human population is by 2-parous flies (feeding for the 3rd time) aged about 6 days. As deduced from the growth stages of filariae observed in wild-caught, infected flies, it was estimated that only about 10% of infected insects lived to 6 days of age and thus became capable of transmitting the parasite. Survival of *C. furens* adults has not been examined in Haiti. However, development of the ovaries following a blood meal requires about 48 h at 27 °C (Linley 1966), from which it follows that transmission must again occur after the 2nd oviposition. The vector efficiency of *C. furens* populations would be affected by the incidence of autogeny, which was not assessed in the Haitian population, but is known to be variably expressed in this species (Linley 1965, Linley et al. 1970). Autogenous females would be expected to transmit very infrequently, since they would do so only after the 3rd oviposition.

Man-baited and truck-trap collections enabled Nathan (1981a) to determine the natural infection rate in the insect population. In females attempting to bite, the overall rate (all larval stages of the parasite) was 0.8% and 5 females of 6659 contained 3rd (infective) stage larvae for an infectivity rate of 0.08%. Truck traps yielded an overall rate of 1.3%, with 9 of 7028 (0.13%) infective. Taking the lowest of these rates (0.08%), it was estimated that a person spending 1 h during the early morning biting peak of *C. phlebotomus* (Nathan 1981b) would accrue 38 infective bites per year. This was considered a conservative estimate, since it did not include bites experienced at other times of day.

Culicoides phlebotomus is a larger midge than *C. furens*. The female wing lengths given by Wirth & Blanton (1974) are 1.03 and 0.91 mm, respectively. There is some evidence that *C. phlebotomus* can sustain higher worm loads per individual than *C. furens*. In *C. phlebotomus* females biting man and taken in truck traps, 2.6–2.8 filariae per infected insect were typical (Nathan 1981a), regardless of the stage of the parasite's development. Among females fed experimentally on a subject with 39 microfilariae/25 μl blood, 55.1% became infected, and worm loads remained quite consistent (average 3/fly) throughout development of the worm. Lowrie &

Raccurt (1981) were able to obtain considerably more experimental feedings on 3 separate occasions, for a total of 23 groups of *C. furens* fed on Haitian volunteers with microfilaremias ranging from 2–1001 microfilariae/20 mm^3 of blood. The results were considered separately according to each occasion, and in 2 of them the worm load seemed generally related directly to the level of microfilaremia (Lowrie & Raccurt 1981). However, if all data are combined, a procedure to which there seems no obvious objection, the relationship is distinctly nonlinear (Fig. 2). It suggests that regardless of the number of microfilariae ingested, the worm load at the 3rd larval stage is limited to between 1 and 2 larvae per female, fewer than in *C. phlebotomus*. The maximum load for *C. phlebotomus* cannot be assessed from data now available, as there was only a single volunteer and the microfilaremia was relatively low compared to some of the Haitian subjects.

In Haiti, *Culicoides barbosai* was considered much less efficient, and *Leptoconops becquaerti* a very poor vector compared with *C. furens* (Raccurt & Lowrie 1981), since both species yielded fewer 3rd-stage larvae per surviving fly when fed on infected volunteers.

When mansonellosis was discovered in South America, there was considerable initial confusion as to what the vector(s) might be. Buckley (1934) had demonstrated development of the parasite in *C. furens*, and in Argentina, Romaña & Wygodzinsky (1950) obtained a high experimental infection rate (35.3%) and were able to observe maturation of *M. ozzardi* to the 2nd larval stage in *Culicoides paraensis* (Goeldi) before high mortality of the flies terminated the experiment. Mirsa et al. (1952) found an apparent natural infection of *M. ozzardi* in *Culicoides pifanoi* Ortiz in Venezuela, but Rachou (1957) and Cerqueira (1959) did not find any natural infections among several species of *Culicoides* in Brazil. Moreover, Cerqueira did find *Simulium amazonicum* Goeldi (actually a misidentification, see below) naturally infected and was able to achieve infection experimentally. Garnham & Walliker (1965) further supported the vector status of *S. amazonicum* on circumstantial grounds. In preliminary work, Shelley (1975) identified a suitable study area, and Shelley & Shelley (1976) were able experimentally to confirm that *M. ozzardi* microfilariae migrated to the thoracic muscles of this insect. Evidence from naturally infected specimens bore out the conclusion that development was completed to the infective stage. They obtained an experimental infection rate of only 7.1% but attributed this to the low microfilaremias of the human volunteers. No natural infections were found in 687 mosquitoes [*Mansonia amazonensis* (Theobald)] or 154 *Culicoides* (sp. indet.). *Mansonia amazonensis* proved incapable of supporting larval development of the worm after experimental feedings.

Recently, Shelley et al. (1980) were able to make a more comprehensive examination of the vector status of a number of hematophagous Diptera in the Brazilian Amazon. These included *Simulium amazonicum*, another undescribed species (*Simulium* sp.), *Mansonia amazonensis*, *Lepiselaga crassipes* (Fabr.) (Tabanidae), and *Culicoides insinuatus* Ortiz & Leon. The undescribed *Simulium* sp. is the same as that mistakenly identified by Cerqueira (1959) as *S. amazonicum* (Shelley et al. 1982). Only the *Simulium* species were dissected for natural infections and specimens of both were positive. Experimental infections were achieved from a Ticuna Indian volunteer showing 112 microfilariae/20 mm^3 fingertip blood. An overall infection rate of 44.8% was achieved in the *S. amazonicum* and 69.2% in the *Simulium* sp. Third-stage larvae appeared in both species 7–9 days after the blood meal. No developing filariae were found in dissections of the other dipteran species, although it was confirmed for all 3, by examination of the gut contents, that microfilariae had been ingested. Similarly, Cerqueira (1959) found that microfilariae died shortly after ingestion by the mosquito *Culex p. quinquefasciatus* Say. Another *Simulium* species, in the *S. sanguineum* Knab group and closely related to *S. amazonicum*, has been studied in Colombia (Tidwell et al. 1980). Two male subjects having average microfilaremias of 128 and 373/20 mm^3 finger blood were used as bait. Microfilariae appeared in the thoracic musculature of the engorged *Simulium* within 2 h following intake of blood. Third-stage larvae were found as early as 5 days later and most had developed to this stage by 6 days. After 6 days the distribution of 3rd-stage larvae was 52% in the head, 34% from the thorax, and 14% from the abdomen.

In addition to the *Simulium* species, females of *Culicoides caprilesi* Fox and a few *C. insinuatus* were collected after engorgement on the subject with the lower microfilaremia. Among the *C. caprilesi*, 40% dissected soon after the blood meal contained microfilariae, but after 6 days only 1 of 93 *Culicoides caprilesi* had an advanced stage 2 larva, and 4 *C. insinuatus* showed no infection. *Culicoides caprilesi* was not considered to be a vector of any importance in the area.

Different authors have recorded quite widely varying natural infection rates among the potential simuliid and tabanid vectors. Cerqueira (1959) found 16.5% of *Simulium* sp. carrying *M. ozzardi* in various developmental stages and, remarkably, 21.4% of the tabanid *Lepiselaga crassipes.* This latter species, when tested experimentally by Shelley et al. (1980), showed no infective forms in 56 specimens dissected 9 days after the blood meal, even though it was confirmed that microfilariae had been ingested. Shelley & Shelley (1976) found only 0.99% of *S. amazonicum* naturally infected at 2 villages on the Purus River, Amazonas State, but later, on the upper reaches of the Solimões River where Moraes et al. (1978) had shown mansonellosis to be most prevalent, Shelley et al. (1980) recorded 3.5% of *S. amazonicum* infected and 9.7% of *Simulium* sp. In Colombia, Tidwell et al. (1980) discovered that 3.4% of wild-caught *Simulium* sp. (*sanguineum* group) were infected with apparent 3rd-stage *M. ozzardi* larvae.

Simulium flies are larger than *Culicoides,* so it is not surprising to find that they seem able to bear greater worm loads. Referring only to stage 3 larvae, the data of Shelley et al. (1980) allow loads of 1.8–5.0 larvae/fly to be resolved for *S. amazonicum,* and 4.4–6.2/fly for *Simulium* sp. Experimentally infected flies of the *S. sanguineum* group in Colombia contained averages of 2.5 or 6.8 larvae per fly, depending on the microfilaremia of the volunteer, and 1 fly had 22 larvae (Tidwell et al. 1980). In contrast, *Culicoides furens* appeared capable of supporting only 1–2 mature larvae per individual, and *C. phlebotomus* 2–3 (earlier discussion).

In some areas in which the parasite is endemic in South America, extremely high levels of infection have been revealed in the human population. In a location with one of the highest levels, the eastern area of Comisaría del Vaupes, Colombia, Marinkelle & German (1970) found over 96% of the adult Amerindians positive, and in the same place 10 years later, Lightner et al. (1980) found 49% of the population (all ages) infected. Near the frontier of Brazil and Colombia with Peru, Moraes et al. (1976) found 45.7% of the Indian population infected. Lower rates have been seen elsewhere, for example 9.17 and 5.9% (Shelley 1975) at 2 villages on the Purus River (Amazonas). The infection rate generally increases with age (Fig. 1) in both sexes (Fros 1956, Moraes et al. 1976, Lightner et al. 1980) and in some populations shows further similarity with the West Indian (*Culicoides*-transmitted) form (Fig. 1) in that infection in males is higher than in females (Lightner et al. 1980). However, this is not always the case (Batista et al. 1960, Shelley 1975).

While the microfilariae of *M. ozzardi* may be found in the skin, they also circulate in the peripheral blood. Some consideration has been given in the literature to the question of whether there is a diel periodicity in the concentration of blood-borne microfilariae that might coincide with the biting cycle of the vector. There are 5 publications that address this question (Rachou & Lacerda 1954, Moraes 1959, Restrepo et al. 1962, Nathan et al. 1978, Raccurt et al. 1980), and they are summarized for comparison in Fig. 3. The biting cycles of the particular vectors, abstracted from sources indicated, are superimposed. The activity data for *Simulium sanguineum* are taken from Lacey & Charlwood (1980, fig. 6) and are the result of observations at Uruá, western Pará State, Brazil. This locality does not match either data set for microfilarial periodicity (Amazonas: Rachou & Lacerda 1954; Colombia: Restrepo et al. 1962). However, it adequately illustrates the diurnal behavior of *S. sanguineum* and the tendency for attack to be concentrated in the morning hours (Lacey & Charlwood 1980).

Restrepo et al. (1962) examined 1 case and sampled blood once during each time period. They detected a pronounced fluctuation in microfilaremia, with a peak coincident with the biting activity of *S. sanguineum* (Fig. 3), a species closely related to a *S. sanguineum* group species thought to be a vector in Colombia (Tidwell et al. 1980). More comprehensive data were obtained by Rachou & Lacerda (1954) from 47 patients who showed only a very weakly defined periodicity, with slightly higher counts generally during the daylight hours. Data obtained from 33 patients by Moraes (1959) were quite similar; the highest counts of microfilariae tended to occur during the early part of the day (about 0700–1400 h) and the minimum at 1800 h (Fig. 3). Moraes also divided his data (patients) into 4 groups according to increasing microfilaremia, and he found that the apparent diel fluctuation was consistent among all groups. In none of these studies were the data subjected to statistical tests; Rachou & Lacerda (1954) concluded that there was no periodicity.

In Trinidad the average microfilaremia from 8 subjects (Nathan et al. 1978) showed a weak fluctuation (Fig. 3), which was not reflected consistently in the data from separate individuals and did not reach a peak with the morning biting ac-

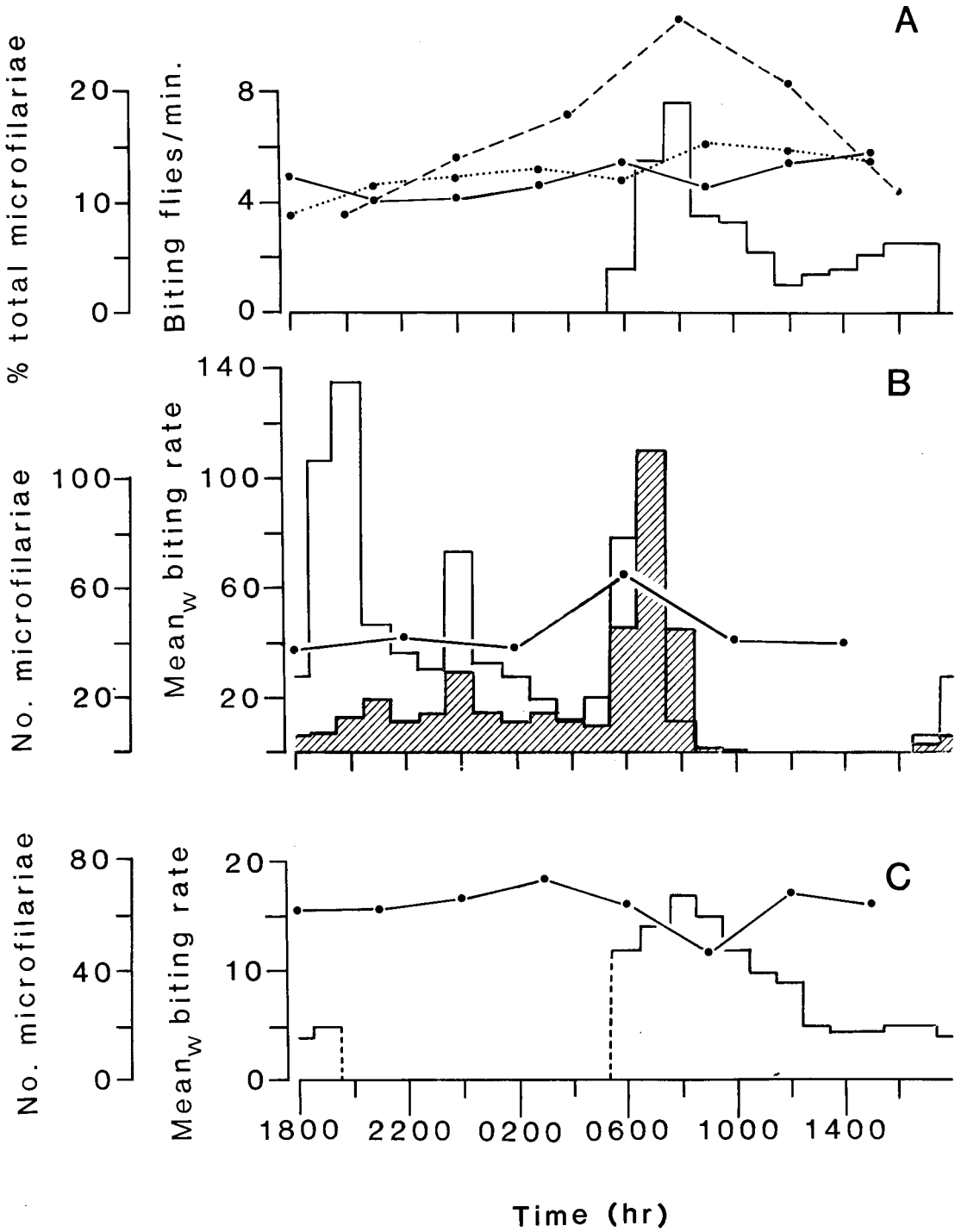

FIG. 3. Possible diel periodicity of *Mansonella ozzardi* microfilaremia (lines) compared with the biting cycle(s) of the vector(s) (histograms): **A**, Amazon basin (solid line, Restrepo et al. 1962; broken line, Rachou & Lacerda 1954; dotted line, Moraes 1959; histogram, *Simulium sanguineum*, Lacey & Charlwood 1980) **B**, Haiti (solid line, Raccurt et al. 1980; open histogram, *Culicoides furens*, Kettle 1969; shaded histogram, *C. barbosai*, Kettle 1969). **C**, Trinidad (solid line, Nathan et al. 1978; histogram, *Culicoides phlebotomus*, Nathan 1981b).

tivity of *C. phlebotomus* (Nathan 1981b). The opposite trend, in fact, appeared to prevail. The biting cycle data were not complete through the hours of darkness, but *C. phlebotomus* is well known to be a diurnal species and thus most of its activity was probably recorded by Nathan (1981b). By analysis of variance, Nathan et al. (1979) concluded that there was no periodicity in the microfilaremia.

When 7 male and 7 female volunteers were examined in Haiti (Raccurt et al. 1980), 9 of the 14 showed a maximum microfilaremia at 0600 h and this was reflected in the average count (Fig. 3), which was otherwise uniform at other times in the diel. *Culicoides furens,* the principal vector in Haiti, exhibits a burst of activity around sunset, continues to bite to some extent through the night, and intensifies its activity again at dawn (Fig. 3). Kettle's (1969) data for the biting cycle are used and may be considered particularly accurate because they were corrected for meteorological variables. The possible secondary vector in Haiti, *C. barbosai,* bites at a low level during the night and has a single peak of activity (Kettle 1969) coincident with that of *C. furens* at dawn (Fig. 3). The microfilarial count was therefore maximal during a peak activity of both vectors. However, the fluctuation in microfilaremia was not pronounced in its range, and Raccurt et al. (1980) concluded that there was no evidence of periodicity. The data sets from both Haiti and Trinidad may be considered distribution-free within each sample period, if for no other reason than the nonequality of microfilaremias between sampled volunteers. We therefore reexamined the differences between sample periods using a nonparametric test. Friedman's method for randomized blocks (Sokal & Rohlf 1969), based on the ranked data, computes X^2 as the analogue of χ^2. The value obtained for the combined Trinidad data ($X^2 = 4.083$) does not approach significance. The Haitian data for females give $X^2 = 9.694$ and for males $X^2 = 10.204$, closely approaching significance in both cases ($\chi^2 = 11.07, P = 0.05$), and the combined data are highly significant ($X^2 = 15.22$, $P < 0.01$). This result does not prove the occurrence of periodicity, but rather that the microfilaremias may be assumed unequal with virtual certainty. However, it is not unreasonable to suppose that since *M. ozzardi* is geographically restricted by island boundaries, it has to some extent become adapted to the biting cycles of these 2 vectors. Perhaps the segments of the *C. furens* and *C. barbosai* populations that bite at dawn are those of an age-spectrum optimal for transmission.

While work with both the West Indian and South American forms of *M. ozzardi* continues, so do questions concerning the taxonomic status of the parasite.* Vector compatibility does not, at least on the basis of very limited observations, indicate an obvious difference between the 2 forms. Lowrie et al. (1982) fed colony *Culicoides variipennis* (Coq.) on a Patas Monkey infected with the Colombian (=*Simulium*-transmitted) strain of *M. ozzardi* and found that the microfilariae were ingested in 20% of the engorged flies and completed development to the infective stage in 9–11 days. This somewhat prolonged period was attributed to lower insectary temperatures. Somewhat earlier, Mellor (1976) had membrane-fed both *C. variipennis* and *C. nubeculosus* (Mg.) on infected blood from a Trinidadian donor and observed production of infective larvae in both species after 8 days. Microfilariae inoculated intrathoracically into *C. variipennis, C. nubeculosus, C. riethi* Kieffer, and *Aedes aegypti* (L.) appeared to be developing normally, even in the mosquito, but unfortunately the rather toxic inoculum brought the observations to a premature conclusion.

Important advances in the understanding of mansonellosis are expected to continue. A model laboratory system is now available owing to the establishment of both Haitian and Colombian strains of *M. ozzardi* in Patas Monkeys (Orihel et al. 1981) and the ready availability of colony *C. variipennis.*

Other filarial parasites

It is well established that species of *Culicoides* (e.g., Collins & Jones 1978, El Sinnary & Hussein 1980) and other ceratopogonid genera (Ottley & Moorhouse 1980) transmit the pathogens causing onchocerciasis in horses and cattle. The human parasite, *Onchocerca volvulus,* normally transmitted by *Simulium,* will complete development in *Aedes aegypti* (Zielke et al. 1977), but when *O. volvulus* was fed to *C. nubeculosus,* either from an infected chimpanzee via membrane feeding or injected intrathoracically, no development was observed (Bianco et al. 1979). Three filarial parasites of monkeys have been reported to complete development in *Culicoides* species (Lowrie et al. 1978, Eberhard et al. 1979) and may provide useful models for future research.

VIRUSES

Oropouche

Oropouche is a Simbu group virus (family Bunyaviridae) first isolated in 1955 from a Trinidadian charcoal worker showing benign febrile illness (Anderson et al. 1961). In addition to the original isolation, the virus has been recognized from a number of localities in the Amazon region of Brazil, and particularly extensively in Pará State (Pinheiro et al. 1962, 1976, 1981a, LeDuc et al. 1981,

* See Addendum.

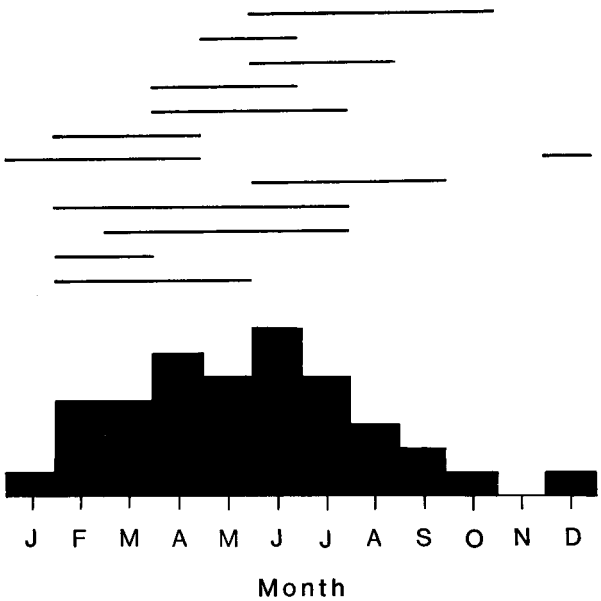

FIG. 4. Occurrence and duration of individual epidemics of Oropouche virus (upper) and combined monthly incidence (lower), summarized from Pinheiro et al. (1981a).

Roberts et al. 1981), but now also in Amazonas (Borborema et al., in prep.). Antibodies have been detected from monkeys in Colombia, but no human cases have been recorded (Dixon et al. 1981). Oropouche is the most important agent of human disease yet found to be associated with a ceratopogonid vector. According to Pinheiro et al. (1982), at least 165,000 persons were infected in numerous outbreaks in Pará State between 1961 and 1980, including 130,000 from 1978–1980. An additional 96,000 persons were presumed infected during the 1980–1981 outbreak that occurred in Manaus, Amazonas (Borborema et al., in prep.).

The incubation period of the disease is probably 4–8 days in natural infections (Pinheiro et al. 1981b). The principal manifestation is fever in over 90% of patients, with over 50% afflicted also with severe headache, myalgia, arthralgia, dizziness, and photophobia (Pinheiro et al. 1976). No fatalities are attributed to the disease, but illness to the point of prostration is seen (Pinheiro et al. 1976, 1981a). Meningitis is a recently reported complication, occurring in 4.1% of 292 cases examined (Pinheiro et al., in press). LeDuc et al. (1981) and Pinheiro et al. (1981a) point out that since Oropouche is in the Simbu serogroup, it is related to Akabane virus, which is known to cause abortion and terata in domestic animals (Inaba et al. 1975, Parsonson et al. 1975). To date, there is no such effect attributed to Oropouche infection in humans, but the possibility is of concern. The course of severe symptoms is usually a week or less, but in many instances recurrences may be experienced, which prolong illness for up to 2 weeks. Freitas et al. (1980) reported that at least 63% of persons who became infected in a 1979 outbreak developed clinical manifestations.

A description and summary of the epidemiology of numerous outbreaks of Oropouche in Brazil in the past 2 decades can be found in various publications (Pinheiro et al. 1962, 1968, 1976, 1981a, Dixon et al. 1981, LeDuc et al. 1981, Borborema et al., in prep.). Epidemics from 1961–1978 are reviewed by Pinheiro et al. (1981a). The proportion of the population infected, as determined from serological evidence, has tended to be high in these epidemics, ranging from 3.5–44% but above 20% in most. In epidemics centered in urban areas, the distribution of infection was found to be uneven (Dixon et al. 1981, Pinheiro et al. 1981a). Differences between the sexes are evident in some localities, but not in others. Pinheiro et al. (1976) found about equal proportions of each sex affected, but in 1 epidemic in Monjuí dos Campos, Pará State, 3 times as many women as men possessed antibody (Pinheiro et al. 1981a). Again, in an urban outbreak, Dixon et al. (1981) encountered an apparent infection rate among women that was twice the rate in men. Where studies have been possible, the weekly distribution of infection by time of onset has shown rapid increase for the first 3–4 weeks of an epidemic, followed by decline over a somewhat longer period (Pinheiro et al. 1976). If the data given by Pinheiro et al. (1981a) are summarized graphically (Fig. 4), it is seen that epidemics have occurred in every month of the year except November, but the combined monthly incidence shows that the disease is primarily one of the 1st half of the year and perhaps particularly of the period March–July.

In an attempt to detect the vector(s) and nonhuman reservoirs of the virus, extensive collections of insects and a large number of vertebrates were tested both during and in the absence of epidemics. Tests during 5 of the epidemics reviewed by Pinheiro et al. (1981a) were aimed both at recognition of circulating virus and the presence of antibody by hemagglutination inhibition. None of 3695 mammals and birds had circulating virus. There were, however, 58 of 3214 animals positive for antibody, including 3.3% of the domestic and wild birds, 1.8% of the monkeys, 25% of the car-

nivores, and 0.2% of the rodents tested. No antibodies appeared in any marsupials, bats, dogs, cats, pigs, sloths, or reptiles examined. Three virus isolations were made from about 20,000 *Culex quinquefasciatus* and 2 each from 2 epidemics and a total sample of about 16,000 *Culicoides paraensis*. LeDuc et al. (1981) also obtained 4 isolations from over 60,000 *C. paraensis* during an epidemic in northern Brazil.

During nonepidemic periods, 25 areas in the Amazon region were sampled (Pinheiro et al. 1981a). The highest antibody rates were obtained from monkeys (several genera) with 11.9% positive, birds with 2.8%, and sloths with 4.1%. Sera from carnivores, ungulates, and reptiles proved negative. Over 1 million insects, comprising Culicidae, phlebotomine sand flies, ticks, and other ectoparasites yielded, at test, a single isolation from a pool of forest-collected *Aedes serratus* Theobald. Isolations from arthropods in the entire survey series were therefore limited to *Cx. quinquefasciatus, C. paraensis,* and *Aedes serratus*. Outside the Amazon region, the only other isolation from another mosquito species had been a single instance from *Coquillettidia venezuelensis* (Theobald) in Trinidad (Anderson et al. 1961).

Field investigations indicated that *Cx. quinquefasciatus* and *C. paraensis* both served as vectors, but very few recoveries of virus were obtained from insects during the several epidemics. Consequently, experimental transmission was attempted. In experiments testing transmission between infected and clean hamsters, *Cx. quinquefasciatus* proved capable of effecting transmission (Hoch et al., unpubl. data, Pinheiro et al. 1982), but the threshold of infection for the mosquito was very high [$\geq$9.5 $\log_{10}$ suckling mouse lethal dose $(SMLD)_{50}$/ml], substantially above the viremia titers normally seen in Oropouche patients (Pinheiro et al. 1982). In contrast, and probably indicative of its principal role as the urban vector, *C. paraensis* became infected at a threshold of about 5.4 $\log_{10}SMLD_{50}$/ml, a titer encountered quite frequently among patients. Among 17 *C. paraensis* fed on 2 infected hamsters, 7 (41%) were found positive for the virus and 5 (50%) of 10 clean hamsters on which they fed contracted disease (Pinheiro et al. 1981b). Three animals were infected by a single bite. The possibility of mechanical transmission was investigated also, but results were negative (Pinheiro et al. 1981b). When 514 midges were exposed to 12 viremic patients but were not allowed to take a visible amount of blood, virus was not recovered from the insects (Pinheiro et al. 1982). In midges taking a full meal of infected blood, no correlation between the duration of the extrinsic incubation period and titer of ingested virus could be determined with certainty (Pinheiro et al. 1981b). However, transmission was accomplished 4–6 days after midges imbibed blood having a high viremic titer (8.9–9.9 $\log_{10}SMLD_{50}$/ml) and 8 days after a blood meal of lower virus concentration (7.2–7.7 $\log_{10}SMLD_{50}$/ml). The vector status of *C. paraensis* was confirmed (Pinheiro et al. 1982) when successful transmission from man to hamster was demonstrated. Infection of 12 (34%) of 35 midges was obtained from 6 of 7 patients circulating viremias in excess of 6.3 $\log_{10}SMLD_{50}$/ml, and of the 12 midges carrying virus, 6 transmitted to clean hamsters. Among 16 patients with viral titers of 5.3–6.2 $\log_{10}SMLD_{50}$/ml, only 5 infected the *Culicoides*; 15 (13%) of 115 of these flies became positive, and 6 (40%) transmitted successfully.

In synthesis, Pinheiro et al. (1981a) proposed a tentative epidemiological model for the disease (Fig. 5). The virus is considered primarily enzootic among sylvatic animals, perhaps monkeys, birds, and sloths, and between these it may be transmitted by sylvatic mosquitoes, although information is scanty. Notably, sylvatic species of *Culicoides* have not been tested. Man is probably the link to the urban cycle and the urban vertebrate reservoir, since domestic animals proved uniformly negative for antibodies. *Culex quinquefasciatus* may occasionally act as an urban vector, but *C. paraensis* principally fulfills this role, since it transmits from an infective blood meal of considerably lower virus titer. The threshold level is exceeded by about 10% of naturally infected persons (Pinheiro et al. 1981a). One rather anomalous finding has been the very low frequency of virus recoveries among *C. paraensis* populations, which have yielded an overall isolation rate of about 1:12,500 (Pinheiro et al. 1982).

Although Oropouche has emerged as the most important human disease transmitted by a ceratopogonid, relatively little is known of the biology of *C. paraensis*. The species is widely distributed throughout the eastern United States, Mexico, Central America, South America (to Argentina and Bolivia) and Grenada (Wirth & Blanton 1974). It has been found breeding in tree-hole debris (Snow et al. 1957, Breeland 1960, Smith 1965) and in Trinidad it was taken from rotting calabash and cacao pods (Williams 1964). The latter habitat is of some significance in the present context, as will be

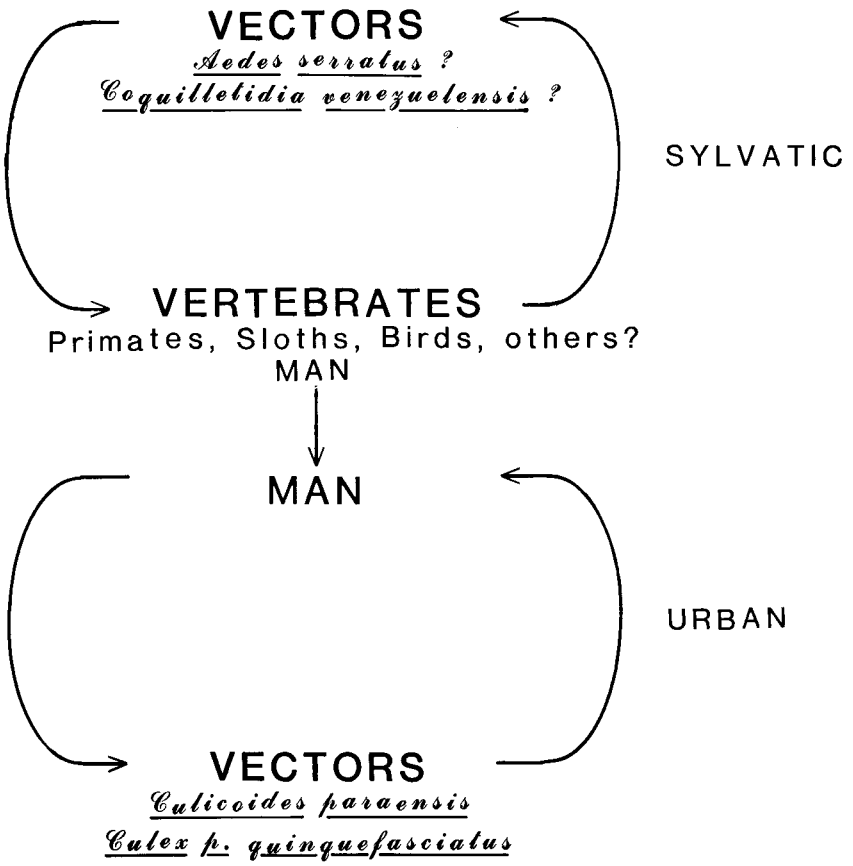

FIG. 5. Tentative epidemiological model for transmission of Oropouche virus disease (from Pinheiro et al. 1981a).

seen shortly. The larva is predaceous on smaller organisms, perhaps chiefly protozoans. A.L. Hoch (unpubl. data) was able to maintain a colony for 3 generations until the number of adults became insufficient to sustain the colony. Immature stages of *C. paraensis* were reared to adults in the laboratory with minimal difficulty.

Culicoides paraensis feeds readily on man (Romaña & Wygodzinsky 1950, Woke 1954, Wirth 1955, Hair & Turner 1968) and shows a diurnal activity pattern. Data abstracted from LeDuc et al. (1981) and Roberts et al. (1981) from Brazil reflect generally the same picture (Fig. 6), except that LeDuc's collections suggest a trimodal distribution. However, fluctuations could have been due to meteorological factors, which were not assessed. There is a gradual increase in activity throughout the day, culminating in a peak in the biting rate in the late afternoon. *Culicoides paraensis* also has the habit, probably of some significance in its vectorial role, of entering dwellings to bite (Pinheiro et al. 1976). The sylvatic hosts are unknown but may be presumed to be primates and sloths.

Culicoides paraensis is a forest species and in localities in Brazil, unmodified by man, may tentatively be presumed to breed in very limited habitats such as tree holes and occasionally in rotting fruit. However, it is remarkable that *C. paraensis* has never been taken in any of several hundred man-baited collections (ground and canopy) in forested areas removed from human dwellings (A.L. Hoch, unpubl. data). Even when *C. paraensis* was abundant around the buildings of a settlement, it was not collected in nearby forest. Regardless of how the midge populations are established, it is evident that as human habitations develop, the growing of banana and cacao creates an association that leads to enormous proliferation of the midge. It is this association that places *C. paraensis* in its key role as an urban vector. *Culicoides paraensis* larvae will invade banana stumps as they commence to rot (Fig. 7) and, equally importantly, will exploit opened and discarded cacao pods. These are often piled adjacent to human dwellings (Fig. 7) and create dense peridomestic populations of the midge. It is perhaps the density of these populations that accounts

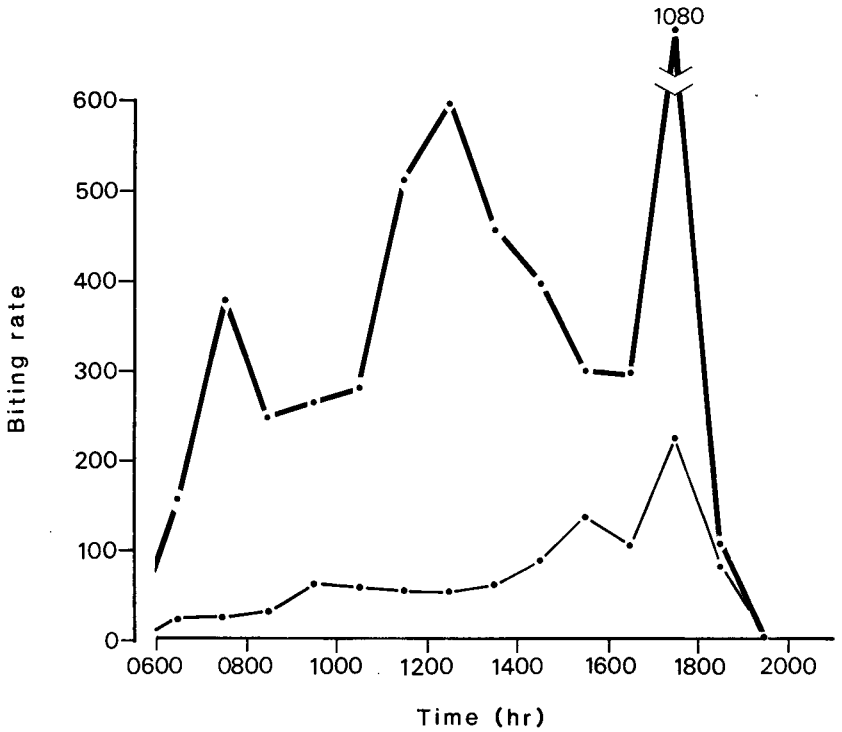

FIG. 6. Biting cycle of *Culicoides paraensis* (bold line, LeDuc et al. 1981; fine line, Roberts et al. 1981).

for the high incidence of human disease during epidemics, despite few virus isolations achieved from the insects so far. The factor(s) that stimulate oviposition by *C. paraensis* in rotting vegetable material are uninvestigated, but might repay curiosity.

Other viruses

There are a number of other viruses that cause human disease and with which biting ceratopogonids are tenuously connected. We consider these briefly.

Rift Valley fever

This arthropod-borne virus causes acute disease of sheep, cattle, a number of other animals, and humans. It appears to be restricted to Africa and nearby areas. Prior to 1977 only 4 human fatalities had apparently been recorded (Meegan et al. 1980), but extensive epizootics in Egypt in 1977 and 1978 resulted in large numbers of human cases with severe illness and possibly several hundred deaths (Laughlin et al. 1979). The disease has been shown to be mosquito borne, with 18 species implicated as possible vectors (Hoogstraal et al. 1979), and especially *Culex p. pipiens* L. Hoogstraal (1978) expressed doubt as to the role of biting midges as vectors. However, Davies & Highton (1980) isolated the virus from 1 pool of *Culicoides* (sp. indet.) collected near cattle pens in Kenya. Since any specimens containing blood had been removed from these pools, they thought that contaminated mouthparts of the midges might have been the source of virus. Nonetheless, that *Culicoides* are the most numerous insects feeding on cattle in Kenya (Davies & Highton 1980) indicated the need for further study. Lee (1979) discovered 2 isolates from pools containing several *Culicoides* species in Nigeria. In contrast, Hoogstraal et al. (1979) obtained no isolates from *Culicoides* during the Egyptian epidemic, and in Natal, McIntosh et al. (1980) similarly failed to isolate virus from many thousands of *Culicoides* (sp. indet.) but made 18 isolations from 4 mosquito species. A recent test with *C. variipennis* (Jennings et al. 1982) has shown that this species, at least, is not susceptible to the virus. Thus, the connection of biting midges with Rift Valley fever virus is weak, but the question remains open.

Congo and Dugbe viruses

Congo virus was first isolated in Africa in 1956 (Simpson et al. 1967). Although the disease seems relatively benign in Africa, with only 1 death recorded (Causey et al. 1970), it was found to be

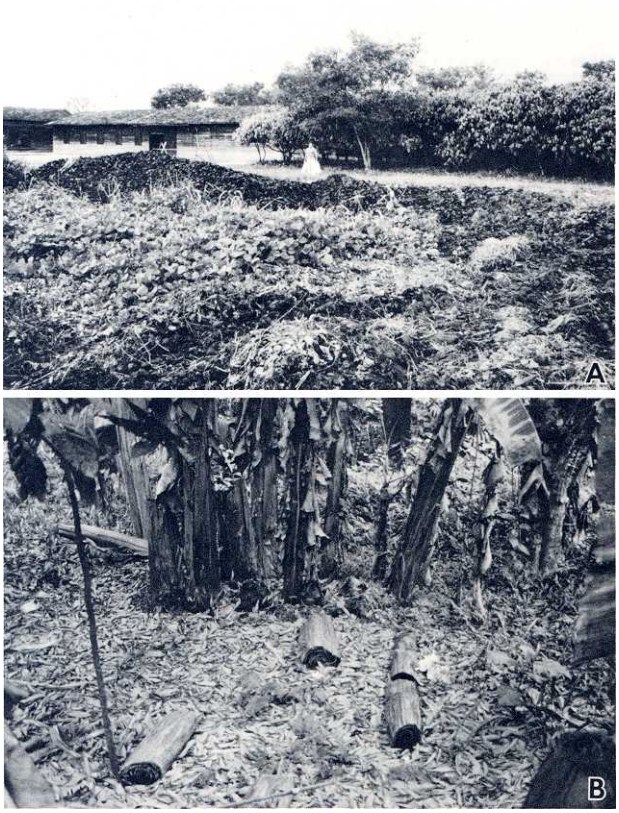

FIG. 7. Larval habitats of peridomestic populations of *Culicoides paraensis* in Brazil: **A**, piles of discarded rotting cacao pods; **B**, rotting banana stems.

serologically very close to Crimean hemorrhagic fever virus (Casals 1969), which causes serious human illness in the USSR and Bulgaria. The virus is primarily tick borne (Causey et al. 1970, Fabiyi 1973). In Nigeria, however, isolations were made from *Culicoides* (sp. indet.) by Causey et al. (1970) and Lee (1979). Little is known of Dugbe virus, first isolated in Nigeria (Causey et al. 1971) and, again, recovered on 3 occasions from pooled *Culicoides* sp. by Lee (1979).

Encephalitis viruses

Available data suggest that ceratopogonids play a limited role, if any, in the transmission of these diseases. Wu & Wu (1957) isolated Japanese B encephalitis virus from *Lasiohelea taiwana* Shiraki, and Karstad et al. (1957) obtained 1 isolation of eastern equine encephalitis virus from pooled *Culicoides* in Georgia, USA. On the other hand, Mitchell et al. (1979) secured no isolations from over 10,000 *Culicoides* sp. tested during an epidemic of eastern equine encephalitis in the Dominican Republic, and Scanlon (1960) failed to transmit this virus experimentally using *Culicoides obsoletus* Meigen.

CONCLUSIONS

The proven association of *Culicoides paraensis* with transmission of Oropouche virus elevates the medical importance of biting midges to a new level. The biology of *C. paraensis* is incompletely known and its particular association with current agricultural practices requires much more thorough investigation. More detailed knowledge should be sought regarding the conditions conducive to oviposition and larval development in banana stumps and cacao pods in various stages of decomposition. Substantial but perhaps not complete control might be achieved with quite simple physical manipulations of the man-made larval habitats. Remarkably small proportions of *C. paraensis* populations sampled have been found to be carrying virus, and as Pinheiro et al. (1981a) point out, it must be ascertained whether only limited segments of the insect populations are capable of transmission.

The uniform importance of voucher material, deposited in conjunction with any planned study, is amply illustrated by problems that currently exist with regard to the midge-transmitted filarial parasites. The identity of African vectors of *Dipetalonema perstans* is in a state of confusion. Comprehensive and integrated collection and taxonomic evaluation of the ceratopogonid fauna of the continent will not occur in the foreseeable future and the deposit of reference materials of both parasite and vector is an essential interim measure. A potential for confusion exists also in the case of *Mansonella ozzardi*, especially in view of the possible difference in identity between the *Culicoides*- and *Simulium*-transmitted forms. However, specimens are being deposited from the Amazon region (e.g., Shelley et al. 1980, Tidwell et al. 1980) and this foresight will greatly facilitate future work.

Acknowledgments. The permission of Allen Press, Inc., to reproduce Fig. 5 is gratefully acknowledged. We thank G.F. O'Meara and J.H. Frank for critical readings of the manuscript.

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ADDENDUM

Very shortly after this review went to press, 3 papers were published that contain important observations on *Mansonella ozzardi.* The 1st paper, by Orihel & Eberhard (1982, Am. J. Trop. Med. Hyg. **31:** 1142–47), provides a redescription of Haitian strain *M. ozzardi* taken from experimentally infected Patas Monkeys. From comparative studies the authors concluded, also, that the Haitian and Colombian strains of *M. ozzardi* are morphologically identical. Further comparisons indicated that *M. ozzardi* and the 2 African filariae of man, referred to in this review as *Dipetalonema perstans* and *D. streptocerca,* are congeneric. Thus, in accordance with taxonomic priority, the African species should now be designated *M. perstans* and *M. streptocerca.*

The 2nd paper is by Tidwell & Tidwell (1982, Am. J. Trop. Med. Hyg. **31:** 1137–41) and describes the successful development, to the infective stage, of *M. ozzardi* in experimentally infected *Simulium amazonicum, S. argentiscutum* Shelley & Luna Dias, and *Culicoides insinuatus* Ortiz & Leon. A 3rd paper, by Kozek et al. (1982, Am. J. Trop. Med. Hyg. **31:** 1131–36) provides extensive data on the prevalence of *M. ozzardi* in teenage and adult populations from 16 villages on or near the Colombian bank of the Amazon.