AEDES ALBOPICTUS AND ARBOVIRUSES: A CONCISE REVIEW OF THE LITERATURE[1, 2]

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ABSTRACT. *Aedes albopictus* is an efficient vector of the four dengue viruses, and it is also capable of transovarially transmitting these viruses. *Aedes albopictus* can also serve as a host and/or vector of several additional arboviruses, some of which are of considerable medical or veterinary importance. This review compiles previously published studies that have demonstrated an experimental or natural association between *Ae. albopictus* and specific arboviruses.

INTRODUCTION

The recent establishment of *Aedes albopictus* (Skuse) in North America (Sprenger and Wuithiranyagool 1986) is a concern for at least two reasons. Not only is it a formidable pest mosquito, but *Ae. albopictus* is a known or potential vector to man of several arboviruses. We know much about the vector competence of this species for some viruses, but there are many important gaps in our knowledge. In this review I have attempted to briefly summarize what is known of the ability of *Aedes albopictus* to serve as a host and/or vector of arboviruses. This report does *not* constitute a comprehensive review of all past arbovirus studies that have involved *Ae. albopictus*, nor does it attempt to inform in depth. The intent is instead to identify only those basic, published reports which demonstrate an experimental or natural association between particular viruses and this mosquito. Important, but as yet unpublished studies of *Ae. albopictus* will not be discussed in this review.

DISCUSSION

It is popular belief that *Aedes aegypti* (Linn.) is the sole vector of the four distinct viruses which cause the spectrum of disease symptoms that we collectively term "dengue" (including dengue fever, dengue hemorrhagic fever and dengue shock syndrome). This belief is largely due to the undisputed involvement of *Ae. aegypti* in many large epidemics of this disease. However, other *Aedes* (*Stegomyia*) species are known dengue vectors, and may be responsible for the occurrence of these viruses in locales where *Ae. aegypti* is absent (Rosen et al. 1985). Unfortunately, it has sometimes been further assumed that *Ae. aegypti* is an especially *efficient* vector of dengue viruses. This is not the case.

A comprehensive study by Rosen et al. (1985) compared the susceptibility of *Ae. albopictus* and *Ae. aegypti* to oral infection with each of the dengue viruses. In every case, a significantly larger proportion of the *Ae. albopictus* became infected when both species were fed on the same infected blood source. The relative efficiency of these species to transmit dengue viruses by bite once they are infected has not been as well-studied. Surprisingly little has been published regarding the efficiency of either *Ae. aegypti* or *Ae. albopictus* to transmit dengue viruses by bite. For technical reasons it has been impossible until recently to reliably quantitate oral transmission of dengue viruses by mosquitoes (Gubler and Rosen 1976). Jumali et al. (1979) were able to simultaneously compare Indonesian *Ae. albopictus* and *Ae. aegypti* infected with dengue-3 virus, and found that the species were equally efficient in transmission by the oral route. The best early evidence that *Ae. albopictus* could transmit dengue viruses came from studies of Simmons et al. (1930), in which human volunteers suffering from dengue were used to infect *Ae. albopictus* females that engorged upon them. These same *Ae. albopictus* mosquitoes were then able to transmit dengue to other volunteers after a suitable period of incubation.

Aedes albopictus has been repeatedly incriminated as a vector during dengue outbreaks, particularly in Southeast Asia. Notable *albopictus*-associated epidemics include those in Japan (Sabin 1952), Thailand (Gould et al. 1968), Singapore (Chan et al. 1971), Central Java (Jumali et al. 1979), and the Republic of Seychelles (Metselaar et al. 1980).

Aedes albopictus is also a potential vector of several additional arboviruses, some of which are of considerable medical importance (Table 1). For example, *Ae. albopictus* can be easily infected in the laboratory by ingestion of Chikungunya virus, which causes a dengue-like

[1] University of Florida, Institute of Food and Agricultural Sciences, Experiment Station Journal Series No. 7384.

[2] Presented at a symposium entitled "*Aedes albopictus* in the Americas" at the annual meeting of the American Mosquito Control Association, New Orleans, Louisiana on April 21, 1986.

Table 1. Susceptibility of *Aedes albopictus* to oral infection with arboviruses, and its ability to transmit them by bite.

Virus	Oral infection?	Oral transmission?	Infected in nature?	References[a]
Chikungunya	+	+		1
Dengue (−1,−2,−3,−4)	+	+	+	2–6
Japanese encephalitis	+	+	+	7–9
Nodamura	+	?		10
Orungo	+	+		11
Ross River	+	+		12
San Angelo	+	+		13–15
St. Louis encephalitis	+	+		16
West Nile	+	+		17
Western equine encephalomyelitis	+	+		18
Yellow fever	+	+		19

[a] (1) Mangiafico 1971, (2) Chan et al. 1971, (3) Rosen et al. 1985, (4) Rudnick 1966, (5) Simmons et al. 1930, (6) Smith et al. 1971, (7) Huang 1957, (8) Rosen et al. 1978, (9) Wu and Wu 1957, (10) Tesh 1980b, (11) Tomori and Aitken 1978, (12) Kay et al. 1982, (13) Shroyer 1986, (14) Tesh 1980a, (15) Tesh and Shroyer 1980, (16) Mitamura et al. 1940, cited in Ferguson 1954, (17) Akhter et al. 1982, (18) Simmons et al. 1936, (19) Dinger et al. 1929, cited in Warren 1951.

disease in man (Mangiafico 1971, Yaminishi et al. 1983). *Aedes albopictus* infected by the oral route are also capable of efficient transmission of Chikungunya virus by bite (Mangiafico 1971).

Table 1 also shows that *Ae. albopictus* is capable of transmitting Japanese encephalitis virus by bite, and that this virus has reportedly been isolated from field-collected mosquitoes (Huang 1957, Wu and Wu 1957). The latter observation suggests that *Ae. albopictus* virus may serve as a vector of Japanese encephalitis in some areas, although it is not presently believed to be a major vector.

Laboratory studies have shown that *Ae. albopictus* is susceptible to oral infection with the remaining viruses listed in Table 1 and that (with the exception of Nodamura virus) these viruses can be transmitted by bite. In some cases the demonstrated susceptibility and transmissibility is only marginal. In other cases (eg., Ross River virus) it is clear that the vector competence of the species is very high. It is noteworthy that Table 1 includes St. Louis encephalitis, yellow fever, West Nile, Ross River, Western equine encephalomyelitis and Orungo viruses, which are all capable of causing serious disease in man. Additional, more extensive study of the potential of *Ae. albopictus* as a vector of these viruses is urgently needed to evaluate the significance of these preliminary studies.

Feeding on a viremic vertebrate is not the only method by which a mosquito can become infected with an arbovirus. Infected female mosquitoes can directly transfer arbovirus infections to their offspring, a process called "transovarial transmission." For at least some arboviruses, it is clear that transovarially transmitted virus is essential to the virus life cycle, while vertebrate infections may be largely incidental "dead-ends" (DeFoliart 1983, Patrican et al. 1985). All four dengue viruses are transovarially transmissible by *Ae. albopictus* (Rosen et al. 1983), as are the other viruses shown in Table 2. Note that the list of transovarially transmissible viruses includes St. Louis and Japanese encephalitis viruses, as well as three of the California encephalitis group

Table 2. Arboviruses that can be transovarially transmitted by *Aedes albopictus*.

Virus	References[a]
Flaviviruses	
Banzi	1
Bussuquara	2
Dengue–1	3
Dengue–2	3
Dengue–3	3
Dengue–4	3
Ilheus	2
Kokobera	2
Kunjin[b]	2
Japanese encephalitis	4
St. Louis encephalitis	5
Uganda S	2
Bunyaviruses	
Keystone	2
La Crosse	2
San Angelo	2

[a] (1) Tesh et al. 1979, (2) Tesh 1980a, (3) Rosen et al. 1983, (4) Rosen et al. 1978, (5) Hardy et al. 1980.
[b] Venereally transmissible by infected males (Tesh 1981).

viruses (Table 2). We probably know more about the mechanism of transovarial transmission of arboviruses in *Ae. albopictus* than in any other mosquito, yet we generally have only a vague impression of the efficiency of transovarial transmission of any particular virus. This is due to an incomplete understanding of the conditions and factors which influence the proportion of *Ae. albopictus* females that transovarially transmit, and the proportion of transovarially infected offspring found within individual families. It is not presently known whether transovarial transmission by *Ae. albopictus* plays an important role in the natural history of any of these viruses.

In the laboratory it is relatively easy to isolate families of *Ae. albopictus* which can very efficiently transmit San Angelo virus (California encephalitis group) by the transovarial route (Tesh and Shroyer 1980, Shroyer 1986). In fact, 38 consecutive generations of transovarial transmission of San Angelo virus have been monitored in *Aedes albopictus* (Shroyer 1986). These mosquitoes were *never* exposed to an infected vertebrate, yet most mosquitoes were infected. Essentially all females that are transovarially infected with San Angelo virus produce at least one infected offspring, but there is considerable variation between mothers in the proportion of infected offspring produced. We need to know whether the mechanism of transovarial transmission of other America arboviruses in *Ae. albopictus* is similar to that of San Angelo virus.

Finally, *Aedes albopictus* is capable of supporting growth of several arboviruses for which there is no information regarding their ability to orally infect, or to be transmitted by bite (Table 3). We know only that these viruses will replicate when injected into the body of *Ae. albopictus*. Some of these viruses induce a lethal paralysis in *Ae. albopictus* which is expressed only if the infected mosquito is exposed briefly to a high concentration of carbon dioxide gas (Rosen 1980). The previously mentioned San Angelo virus also induces such carbon dioxide sensitivity (D. A. Shroyer, unpublished data).

Aedes albopictus is not known to be a natural host of any of the viruses listed in Table 3, and there may be a tendency for such information to be regarded as having little relevance. However, until 1985, the distribution of *Ae. albopictus* and San Angelo virus were not known to overlap, San Angelo virus being known only from Texas, Arizona, New Mexico, and Colorado (Calisher 1983). Studies of San Angelo virus in *Ae. albopictus*, which previously served only as an experimental model, might now take on new significance with the discovery that this virus and mosquito have overlapping distributions. Clearly, medical entomology does not deal with an unchanging world.

Table 3. Additional arboviruses known to replicate following inoculation of *Aedes albopictus*.

Virus	References[a]
Alphaviruses	
Sindbis	1
Phleboviruses	
Arumowat	2
Bujaru	2
Chilibre	2
Icoaraci	2
Itaporanga	2
Karimabad	2
Pacui	2
Salehabad	2
Rhabdoviruses	
Chandipura[b]	3
Cocal	4
Gray Lodge[b]	3
Joinjakaka[b]	3
Piry[b]	3
Sigma[b]	3
Vesicular stomatitis–NJ[b]	3
Unclassified	
Matsu[b]	5

[a] (1) Stollar and Hardy 1984, (2) Tesh 1975, (3) Rosen 1980, (4) Hurlbut and Thomas 1969, (5) Rosen and Shroyer 1981.
[b] Induce lethal sensitivity to carbon dioxide.

CONCLUSIONS

I have tried to briefly convey some notion of the known and potential capacity of *Ae. albopictus* to serve as arbovirus host and vector. It is clear that for some of these viruses, the species could have the capacity to serve as a vector under appropriate environmental circumstances. We know that *Ae. albopictus* is an excellent vector of dengue viruses. Yet there are many unanswered questions that require the urgent attention of the scientific community. We need to know whether North American populations of *Ae. albopictus* are sufficiently susceptible to infection with the other important American arboviruses and whether transmission by bite is efficient enough to permit the species to serve as a natural vector.

Should *Ae. albopictus* spread into Central America and the Caribbean, will the severity of dengue disease in these areas be affected? It has been hypothesized that *Ae. aegypti*-transmitted dengue epidemics tend to result in more severe forms of disease, such as fatal hemorrhagic fever (Rosen et al. 1985). It is thought that the relatively low susceptibility of

Ae. aegypti to infection assures that only virus strains which can grow to very high titers in man will be transmitted, and that these same strains are likely to be most pathogenic. *Aedes albopictus* transmission between humans might instead lead to the selection of dengue virus strains having very different pathogenicity.

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