BOARD OF COUNTY COMMISSIONERS

PUBLIC WORKS DEPARTMENT

Respond to:
Mosquito Control
2300 Virginia Ave.
Ft. Pierce, FL 34982

July 14, 1992

Ms. Carol I Meeds
Florida Department of Environmental Regulation
Southeast District Office
1900 Congress Ave.
Suite A
West Palm Beach, FL 33406

Dear Ms. Meeds,

The information you requested in your letter of June 30, 1992, is provided as an attachment.

SUMMARY

MOSQUITO SPECIES AND POTENTIAL AS DISEASE VECTORS

Three species of mosquitoes have been found in the tires at Florida Tire and Recycling in the past three years. Two of those species, Aedes aegypti and Aedes albopictus, are capable of disease transmisssion. However, no evaluation of the local population at the tire recycler has been performed to determine if they are actually carrying disease. The diseases for which these species are known vectors are the following:

A. aegypti - Yellow Fever, Dengue, Dog Heartworm (and others)
A. albopictus - Yellow Fever, Dengue, Eastern Equine Encephalitis, St. Louis Encephalitis, other Flaviviruses and Bunyaviruses

CHEMICAL TREATMENTS

The tires were Ultra Low Volume adulticided and handfogged with thermally applied pesticide on repeated occasions from April through November of 1990. The tires were also larvicided with Abate 4E during the same period. Chemical treatment ceased the following year, after mosquito populations declined in abundance during the winter of 1990-91, and after the area sustained significant flooding in the following summer (which restricted vehicle operation around the tire piles).

Furthermore, at the reduced abundances observed, the mosquitoes don't seem to be exodusing far from the site, which is outside the District.

The adulticiding chemicals employed were; Scourge (resmethrin and piperonyl butoxide diluted 6:1 with oil (orchex or BVA) applied as ULV by truck or by hand-held fogger, Permanone 10-EC (diluted in water at 2.5 qts/100 g of water) and Pyrethrin thermal fog (diluted 4 oz/gal diesel).

The larviciding chemicals employed were; Abate (diluted in water at 8 oz/200 g).

Please contact the District if you require any further information.

Respectfully yours,

Frank D. S. Evans
Director Mosquito Control

cc: Public Works Administrator
file

CHEMICAL APPLICATIONS

FLORIDA TIRE RECYCLER

YEAR	MONTH	DAY	TIME	LARVICIDE	ADULTICIDE**
1990	APRIL	25	0700-0930	ABATE	
1990	APRIL	30	1300-1730	ABATE	PERMANONE
1990	MAY	1	0700-0845	ABATE	PERMANONE AND SCOURGE
1990	MAY	9	0700-0930		PYRETHRIN
1990	MAY	14	0700-0930		PYRETHRIN
1990	MAY	15	0700-1630	ABATE	PERMANONE AND PYRETHRIN
1990	MAY	31	0755-1230	ABATE	PYRETHRIN
1990	JUNE	6	0720-1000	ABATE	PERMANONE
1990	JUNE	28	1042-1730	ABATE	PERMANONE
1990	JULY	12	1300-1600	ABATE	
1990	JULY	26	1500-1700	ABATE	
1990	JULY	16	1530-1730	ABATE	
1990	AUGUST	21	0700-1330*	ABATE	
1990	SEPT.	13	0700-1200*	ABATE	

NOTE: TIMES ARE APPROXIMATE

* OTHER LOCATIONS ALSO TREATED

** HAND APPLIED THERMAL OR ULV ADULTICIDE

TYPE OF CHEMICAL SPRAYED	CONCENTRATION/DILUTION
SCOURGE ULV APPLICATION	1 PART SCOURGE/5 PARTS ORCHEX OIL
PERMANONE 10-EC	2.5 QTS PERMANONE/100 GAL WATER
ABATE 4E	8 FL. OZ. ABATE/200 GAL WATER
PYRETHRIN THERMAL FOG	4 FL. OZ. PYRETHRIN/GAL DIESEL

RECEIVED
JUL 2 4 1992
DEPT. OF ENVIRONMENTAL REG.
WEST PALM BEACH

FLORIDA TIRE RECYCLING

09-Jul-92

SPECIES FOUND IN EGG POTS

AEDES SPECIES

MONTH	AEGYPTI	ALBOPICTUS	TRISERIATUS
1990			
APRIL	(X)	(X)	
MAY	X		
JUNE	X		
JULY	X	X	X
AUGUST		X	
SEPTEMBER		X	
OCTOBER		X	
NOVEMBER		X	
DECEMBER		(X)	
1991			
JANUARY		X	
FEBRUARY	X	X	
MARCH			
APRIL			
MAY	X	X	
JUNE		X	
JULY	X	X	
AUGUST		X	
SEPTEMBER		X	
OCTOBER		X	
NOVEMBER	X	X	
DECEMBER	X	X	
1992			
JANUARY			
FEBRUARY			
MARCH			
APRIL			
MAY		X	
JUNE		X	

RECEIVED

JUL 2 4 1992

DEPT. OF ENVIRONMENTAL REG.
WEST PALM BEACH

FLORIDA TIRE RECYCLING

09-Jul-92

FOGGED USUALLY BETWEEN 08 - 10 AM

SCOURGE

1990	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER
1					X		X	
2				X	X		X	
3		X			X			
4		X	X			X		
5			X			X		X
6					X	X		
7		X	X		X	X		
8		X	X		X		X	
9				X	X			
10		X		X	X	X		
11		X		X		X		
12		X	X	X		X		
13			X	X	X	X		
14		X	X			X		
15			X		X		X	
16				X	X			
17		X		X	X			
18			X	X				
19			X	X				X
20					X			
21		X	X		X	X		
22		X			X		X	
23	X				X			
24	X	X		X	X	X		
25	X	X	X					
26	X		X	X				X
27	X		X	X	X	X		
28			X		X	X		
29			X				X	
30	X	X		X	X			
31				X				

* NOTE: No more treatments from December 1990 through June 1992.

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	
Arumowot	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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A Florida Mosquito Control Factsheet

The Asian Tiger Mosquito in Florida

G. F. O'Meara, Ph.D. & A. D. Gettman, Ph.D.

Prior to 1985, the distribution of *Aedes albopictus*, the Asian tiger mosquito, was confined to Asia and many islands in the Pacific Ocean, including some of the Hawaiian Islands. Yet, in recent years the range of this mosquito has greatly expanded. It is now known to occur on every continent except Antarctica.

Aedes albopictus was most likely introduced into North America through the importation of used tires from Japan or Taiwan. During the 1980s there was a large increase in the number of used tires imported into the United States from countries where *Ae. albopictus* is indigenous. Most imported used tires arrive in containerized shipments that are not adequately inspected for mosquitoes at the ports of entry. Imported tires are sent to numerous locations where they may be stored outdoors. Those that are not suitable for recapping may end up at illegal dump sites. These conditions have enabled *Ae. albopictus* to become well-established in the United States. To date, *Ae. albopictus* has been detected in 22 states including Hawaii.

In 1986, the Asian tiger mosquito was initially discovered in Florida at a tire dump site in Jacksonville (Duval County). By December 1991, this exotic mosquito had been found at one or more sites in 61 of the 67 Florida counties (Fig. 1). Currently, *Ae. albopictus* populations are very common throughout north Florida, widely but sparsely distributed in the central part of the state and rare in south Florida. *Aedes albopictus* will probably continue to expand its range down the Florida peninsula, becoming a common container-inhabiting mosquito statewide.

FACTORS CONTRIBUTING TO THE SPREAD OF *AEDES ALBOPICTUS*

- The widespread availability of suitable aquatic habitats
- The movement of containers
- The decline of *Aedes aegypti* populations
- The rapid adaptation of *Ae. albopictus* to shorter photoperiods

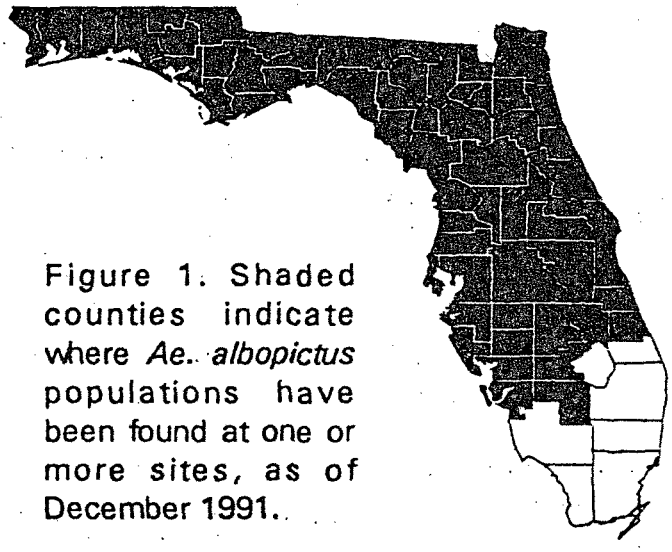

Figure 1. Shaded counties indicate where *Ae. albopictus* populations have been found at one or more sites, as of December 1991.

AQUATIC HABITATS

Although immature *Ae. albopictus* inhabit many different types of containers, scrap tires harbor this mosquito more frequently and in greater numbers than any other type of habitat. Major pest problems caused by *Ae. albopictus* are often associated with accumulations of

scrap tires. Approximately 12 million waste tires are generated yearly in Florida. To deal with this solid waste problem, the Florida Department of Environmental Regulation established new regulations concerning the storage, movement and disposal of waste tires. Authority to issue the Waste Tire Rule was provided by new state laws that became part of the Florida Statutes (§403.717, F. S.) in 1988 with amendments added in 1990. If vigorously enforced, the new waste-tire regulations will eventually diminish the availability of used tires as habitats for *Ae. albopictus* and related mosquitoes.

Other common man-made habitats for immature *Ae. albopictus* include bird baths, water bowls for pets, buckets, plates under potted plants, clogged rain gutters and flower vases. Natural containers, such as treeholes and tank bromeliads, also provide suitable habitats for immature *Ae. albopictus*. Indeed, this mosquito shows a much greater propensity for using natural containers than does *Ae. aegypti*.

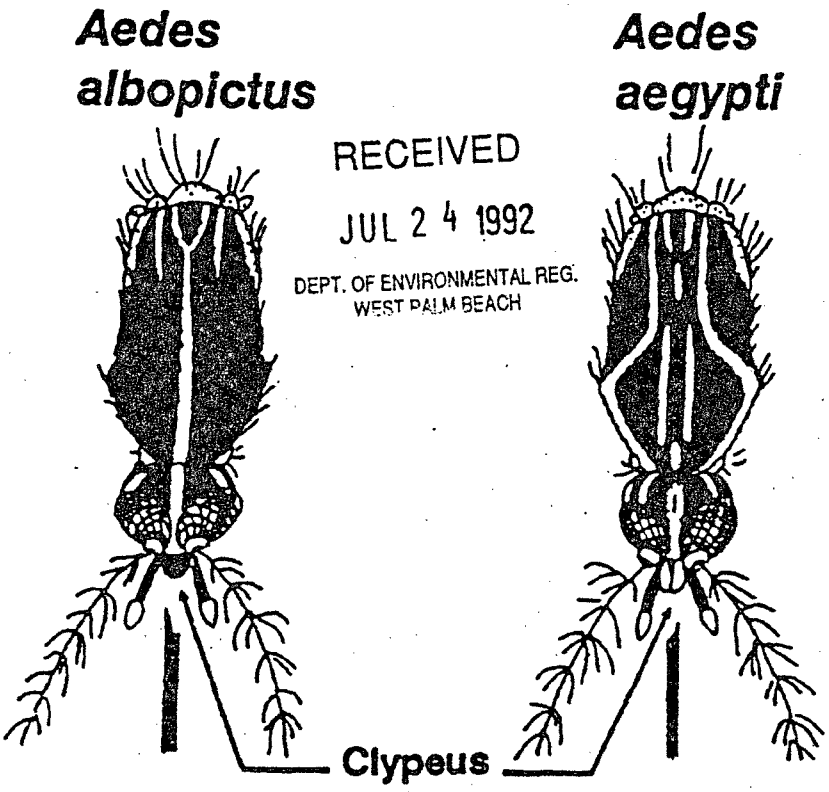

Figure 2.

MOVEMENT OF CONTAINERS

The movement of waste tires to disposal and recycling sites has enhanced the spread of *Ae. albopictus*. Along the east coast of Florida, the southern-most population of *Ae. albopictus* occurs at a site where waste tires are brought from all parts of the state for recycling. Unfortunately, waste tires have been accumulating at this site faster than they are being shredded. Similar problems have occurred at some county landfills, thereby allowing these sites to serve as focal points for the spread of *Ae. albopictus*.

In more than a dozen Florida counties, the initial discovery of *Ae. albopictus* was made at cemeteries. Plastic floral baskets with fresh-cut flowers are often placed at a grave site at the time of burial. After a few days, the wilted flowers are discarded, but the baskets are usually recycled. They may return to the same or a different cemetery holding either fresh or silk flowers. *Aedes* eggs laid while the basket is in one cemetery may hatch in another. This invasion route may explain why *Ae. albopictus* has become well-established in some cemeteries before appearing in nearby accumulations of waste tires.

DECLINE IN *AEDES AEGYPTI* POPULATIONS

The spread of *Ae. albopictus*, especially in north Florida, seems to be occurring at the expense of resident *Ae. aegypti* populations. The abundance of *Ae. aegypti* in several Florida cities has declined drastically and rapidly with the introduction of the Asian tiger mosquito. Several theories have been proposed to account for the apparent displacement of *Ae. aegypti* by *Ae. albopictus*. However, additional field studies are needed to assess the role *Ae. albopictus* populations may play in bringing about these changes.

ADAPTATIONS TO SHORTER PHOTOPERIODS

Populations of *Ae. albopictus* in North America exhibit photoperiod-induced egg diapause, and this is one of several factors which suggests this mosquito has invaded the continental United States from the temperate region of Asia. Probably due to its temperate origin, *Ae. albopictus* quickly spread in the temperate zone of central and eastern United States.

By contrast, this mosquito was relatively slow in spreading to lower latitudes. Short daylength (<13.5 hours) triggers egg diapause. For *Ae. albopictus* populations to remain active in south and central Florida during a significant portion of the year requires a lowering of the daylength threshold or the total elimination of the photoperiod response, because in this region daylengths for much of the year are less than 13.5 hours. Rapid selection for both of these features has been detected in *Ae. albopictus* populations along the southern border of its Florida distribution.

DISTINGUISHING *AEDES ALBOPICTUS* FROM *AEDES AEGYPTI*

Adult *Ae. albopictus* and *Ae. aegypti* are day-active mosquitoes which usually can be distinguished with ease in the field by the distinctive scaling patterns on the top of the thorax (its back) (Fig. 2). These scales may be rubbed off, especially in specimens taken in light or suction traps. In addition, fortunately, the clypeus, a structure located on the head between the palpi (Fig. 2), is covered with white scales in female *Ae. aegypti* and with only black scales in female *Ae. albopictus*. The scales on the clypeus are seldom absent.

Larvae of the two species can be separated by differences in the shape of the comb scales (CS), the size of the thoracic spines and the number of branches in seta 7-C on the head (Fig.3). These structures are easily seen with a stereomicroscope at 40 to 50X using a good illuminator.

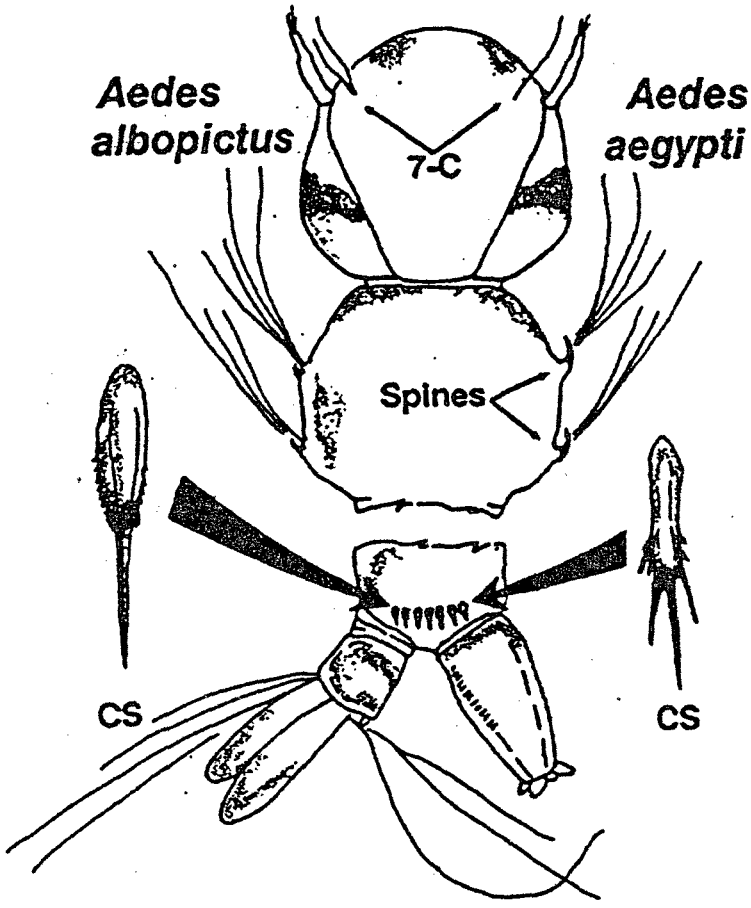

Figure 3.

STATUS OF *AEDES ALBOPICTUS* AS A PEST AND DISEASE VECTOR

Aedes albopictus has quickly become a serious pest species in many north Florida communities where the annoyance level generated by populations of this mosquito is considerably greater than that caused previously by *Ae. aegypti* populations. The wider range of habitats occupied by *Ae. albopictus* tend to make it more common than *Ae. aegypti*. Moreover, Florida populations of *Ae. albopictus* were most likely derived from temperate zone stock, thus they may be better adapted to survive and thrive in north Florida than *Ae. aegypti*, which is primarily a tropical and subtropical mosquito.

In Florida, the Asian tiger mosquito has not been implicated in the transmission of any

human pathogens. However, it is a major vector of dengue and yellow fever elsewhere. Results of laboratory tests indicate that *Ae. albopictus* is capable of serving as a vector for several other viruses that are pathogenic to man or animals.

CONTROL

The best approach for controlling *Ae. albopictus* (and *Ae. aegypti*) is by eliminating larval habitats. Educational programs, which inform citizens about the important role they can play to prevent mosquito production from containers around their homes, need to be expanded and offered on a continuing basis. By eliminating unneeded containers and by frequently emptying the water in other containers (e.g., bird baths and pet-watering dishes) around their homes, residents can complement the control efforts of the local mosquito control program. Current requirements for the storage of waste tires are grossly inadequate for mosquito abatement. The Waste Tire Rule should be revised to require that all tires be stored under cover. With improved legislation and better informed citizens, it should be possible to greatly reduce the abundance of container-inhabiting mosquitoes throughout Florida.

Achieving permanent mosquito control in natural containers, such as tank bromeliads, may be more difficult. Therefore, homeowners should consider limiting the number of these plants that they place in their yards. Chemical and microbial larvicides may provide temporary control, but generally they are not cost effective. Mosquitoes and plant debris can be flushed out of tank bromeliads with a garden hose equipped with an appropriate nozzle. It is unlikely that plants flushed twice a week would produce many adult mosquitoes. Growing bromeliads in plastic pots makes it easier to flush out the mosquitoes.

NEED MORE INFORMATION . . .

. . . on the distribution of the Asian tiger mosquito in your county, contact the mosquito control program in that county, or **Dr. Charlie Morris, Extension Medical Entomologist, Florida Medical Entomology Laboratory, IFAS-University of Florida, 200 9th Street Southeast, Vero Beach, FL 32962, 407/778-7200.**

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PUBLIC HEALTH IMPORTANCE OF *AEDES AEGYPTI*

Introduction

Aedes aegypti is a highly domestic mosquito, characteristically breeding in artificial containers in and around human habitation. The species is widely distributed around the world, generally within the limits of 45° north and 35° south latitude. While it has been known to thrive outside these limits, such populations are probably introduced during the warm seasons but do not survive the winters. *Aedes aegypti* is thought to be a tropical old-world species which was introduced into the Western Hemisphere during the early European explorations and colonizations. In the Western Hemisphere, *Ae. aegypti* is currently known to exist or to have existed in all countries and territories except Canada. In the United States, the species is widely distributed throughout the Southeast, being found in at least 10 states and in Puerto Rico and the U.S. Virgin Islands.

The role of *Ae. aegypti* as a vector of human disease was first demonstrated 1900-1901, when the U.S. Army Yellow Fever Commission in Cuba definitely incriminated the species as a vector of yellow fever. In 1906 in Australia, *Ae. aegypti* was suggested as a vector of dengue, another important viral disease. It was proven to transmit dengue during an epidemic in that country in 1916. The species has also been implicated in the transmission of several additional viral infections (e.g., Chikungunya and Zika viruses) and has been demonstrated as an important vector of the dog heartworm, *Dirofilaria immitis*.

Because of its habit of breeding and feeding in and around human habitations, populations of *Ae. aegypti* may at times reach proportions sufficient to become a serious pest mosquito in some U.S. mainland areas; however, only in the U.S. territories in the Caribbean has the species been involved in the transmission of human disease during the past several decades. Because of the recent dengue outbreaks in Puerto Rico and the Virgin Islands, active *Ae. aegypti* surveillance and control programs exist in these areas. However, in the southeastern United States such programs are relatively rare, existing in only a few urban situations with active mosquito abatement programs which include specific efforts directed at the control of this potential vector.

The control of *Ae. aegypti* has been of importance in the Western Hemisphere since it was demonstrated that the species was the principal vector of yellow fever. At that time, considerable enthusiasm was generated in favor of the concept that yellow fever could be eradicated by vector control. This concept was developed on limited knowledge of the natural history of yellow fever and a conviction that the control of *Ae. aegypti* would prevent the maintenance of the virus in nature. Later information showed that the natural cycle of yellow fever virus involved monkeys and mosquito species other than *Ae. aegypti* in a jungle environment and, therefore, the natural cycle would remain unaffected by efforts to eradicate yellow fever from the urban areas.

In the early 1920's some antimosquito programs in South America succeeded in eliminating *Ae. aegypti* from defined areas. Such success was often cited in support of a growing concept of species eradication for the control of yellow fever. In 1947, the member nations of the Pan American Health Organization (PAHO) resolved to eradicate *Ae. aegypti* from the Western Hemisphere. Nearly all of the involved countries and smaller political entities subscribed to this concept, and during the last three decades, at one time or another, have planned or conducted a program with *Ae. aegypti* eradication as the objective. Based on standard criteria developed by PAHO, by

1965 17 countries had eradication confirmed. In 1977 only seven countries and the Panama Canal Zone were shown by PAHO to have completed eradication and were under a surveillance program. Many countries have now experienced the discouragement of administrative, technical, or financial problems, and regardless of the level of control effort, few areas, if any, have remained permanently free of resurgent *Ae. aegypti* populations.

Yellow Fever

Yellow fever is a severe, mosquito-borne hemorrhagic viral disease of the tropics characterized by high fever, generalized pain, hemorrhage, jaundice and prostration. Inapparent, subclinical yellow fever infections do occur and many overt cases are mild, but the usual clinical course is the sudden onset of fever (often greater than 40°C), headache, and generalized pain. Other early signs are conjunctivitis, photophobia, low white blood cell count, and low platelet count. Early involvement of the liver results in an enlarged liver and jaundice. The heart and kidneys may be involved and renal shutdown may occur. Hemorrhagic manifestations follow which may vary from bleeding gums to massive vomiting of blood, the so-called "black vomit." Prostration, coma, and death may ensue. Although in some epidemics more than half of those with yellow fever have died, the mortality rate is usually 5 to 10%.

There is no specific treatment for yellow fever. Supportive measures such as maintenance of proper hydration and procedures to decrease the high fever are all that are available. Infection elicits an immune response that protects an individual for life. Reinfection is not known to occur.

Diagnosis: Yellow fever should be suspected in any patient exposed in an area where yellow fever transmission is occurring, who has high fever and prostration, followed by heptomegaly, jaundice, hemorrhagic manifestations and renal problems. Laboratory confirmation should be immediately sought. Inapparent and mild cases do occur.

Serum samples collected acutely and then 10-14 days later will show a rise in titer by neutralization (N), hemagglutination-inhibition (HI) or complement fixation (CF) tests. Because the yellow fever virus is a flavivirus (group B arbovirus), serologic cross reactions occur to related viruses such as dengue, Japanese B encephalitis, and St. Louis encephalitis. Therefore, for the diagnosis to be established the virus must be isolated, or serologic titers and neutralization indices must be greater for yellow fever than for the other flaviviruses.

Yellow fever virus can be isolated from blood collected 2-3 days after onset of illness and inoculated into suckling mice or mosquito cell culture. The virus can also be isolated from liver material obtained at postmortem examination.

Yellow fever virus may be isolated from *Ae. aegypti* mosquitoes or from monkeys, but such isolation does not help in the clinical diagnosis of a patient since it merely confirms the presence of virus activity in the area.

Epidemiology: *Aedes aegypti* is the major vector of urban yellow fever and *Haemogogus* or *Sabethes* spp. are the major vectors responsible for sylvatic transmission in the New World.

The mosquito becomes infectious 1-2 weeks (depending on the ambient temperature) after biting an infected person. At that time, spherical viral particles can be found in the vector's salivary gland. The mosquito remains infectious for life and infects the primate host during the ingestion of blood. Clinical symptoms

appear within 3-6 days, and the virus is present in the blood for at least 3 days after the onset of symptoms. Although severity of the disease may vary according to age, in a nonimmune population all age groups are involved.

Human infection with yellow fever results from two different cycles of virus transmission, urban and sylvatic. The urban cycle is the simple transmission from person to person by *Ae. aegypti*, usually occurring in epidemic form. The sylvatic or jungle yellow fever cycle may vary according to the ecologic situation, available primate reservoirs, and vector populations. Some species of monkeys have a high case fatality rate, but others rarely succumb to sylvatic yellow fever. The principal sylvatic primate hosts include several species of monkeys; in the Western Hemisphere the virus is transmitted among these hosts by mosquitoes of the genera *Haemogogus* and *Sabethes*, which typically inhabit the treetops or forest canopy. Humans become involved in the sylvatic cycle incidentally in two ways: either human invasion of the forest or human-directed change in the terrain brings people into contact with the otherwise remote treetop mosquitoes of the same areas, or infected primates approach a semi-sylvan village, are bitten by peridomestic mosquitoes (such as *Ae. simpsoni* in Africa), and then these mosquitoes transmit the disease to humans. The initiation of an urban epidemic outbreak may then ensue, providing an urban vector such as *Ae. aegypti* is present.

For centuries, yellow fever was a serious scourge in the tropical Americas and Africa, extending to temperate areas in violent epidemics during the summers, chiefly in seaport and river cities. In the United States, devastating epidemics occurred during the period from 1668 (New York) to 1905 (New Orleans), striking cities from Texas to New England. Philadelphia suffered 20 epidemics, New York 15, Boston 8, and Baltimore 7. The 1793 Philadelphia epidemic was most severe, with 4,041 deaths from August to November in a city of only 40,000. The explosive nature of the outbreaks is illustrated by the 1878 epidemic in Memphis, Tenn., where approximately 4,000 people died, and by the 1898 epidemic in New Orleans, La., which produced 13,817 cases and 3,894 deaths. The last epidemic in the United States (1905), with 8,399 cases and 908 deaths, struck most heavily in New Orleans which reported 3,384 cases and 443 deaths. The fact that the 1905 epidemic was much less extensive in New Orleans than that of 1898 was attributed largely to a concerted drive against *Ae. aegypti*, the sole urban vector of yellow fever.

Unlike the United States, the countries of Central and South America continued to experience urban epidemics of yellow fever. In 1928 and 1929, the disease reappeared in Rio de Janeiro, Brazil, after an absence of 20 years, with 435 recorded deaths. During the years 1932 to 1954, urban yellow fever occurred in one or more municipalities in Bolivia, Brazil, Colombia, Paraguay, and Trinidad. In addition, outbreaks of jungle yellow fever have continued to occur in all of the Central and South American countries with the exception of El Salvador, Uruguay, and Chile.

In areas of South America where the mosquito-nonhuman primate cycle continues the natural maintenance of yellow fever transmission, hundreds of cases of human sylvatic yellow fever have been reported. Cases and deaths were reported from regions of Paraguay, Brazil, and Argentina in 1966. Fatal cases are reported almost annually from Peru, Colombia, and Venezuela. A resurgence of sylvatic yellow fever in Ecuador was noted in 1975 due to the opening of the eastern regions of the country to oil exploration, and has periodically occurred in Boltvia and Venezuela. In 1948 there was an outbreak of sylvatic yellow fever in Panama, and during the next decade it appeared to spread northward through Central America as far as southern Mexico. Sylvatic yellow fever reappeared in eastern Panama in 1974, but no northward spread was apparent following this outbreak. In 1978-1979 sylvatic yellow fever was reported from Trinidad. Despite the relatively frequent occurrence of human cases of yellow fever acquired in the sylvatic cycle, no significant epidemics of urban yellow fever have been reported in the Americas since 1954.

Sylvatic yellow fever is common in recognized areas of sub-Sahara Africa and has given rise to epidemics in Eastern Nigeria, the Sudan, Ethiopia, and Senegal. The most recent epidemics have been reported from the Gambia in 1978-1979.

Yellow fever has never invaded Asia despite the widespread distribution of man-biting *Ae. aegypti*. The reason is unknown.

In theory, the possibility of the recurrence of epidemics of yellow fever persists wherever there are populations of *Ae. aegypti*. However, epidemics have not occurred within the United States since 1905, and such epidemics in North America or in the Antilles would seem unlikely to occur in the future, even with the presence of *Ae. aegypti* populations, because of the absence of nearby jungle areas where the sylvan cycle of yellow fever persists. Current opinion is that, should a yellow fever outbreak occur in this area, it could be quickly brought under control with the use of vaccines and modern mosquito control methods.

Prevention and control: Effective vaccines are available which offer protection against yellow fever. No case of yellow fever has been reported in a properly vaccinated individual. Toxic reactions are minimal; however, persons with known allergies to eggs or egg products should be vaccinated with caution. Current recommendations suggest revaccination at 10-year intervals.

Epidemic yellow fever can be prevented by control of the vector, *Ae. aegypti*. Elimination of sylvatic yellow fever in the jungles of Africa and South America is presently inconceivable because of the multiplicity of mosquito vectors, the unknown range and identity of vertebrate reservoirs, and the vast areas involved.

In the Americas, the principal vectors are members of the genera *Haemagogus, Aedes, and Sabethes*. In Africa, jungle yellow fever is essentially a disease of monkeys. *Aedes africanus* and *Ae. luteocephalus* appear to be the active vectors of the monkey population, whereas *Ae. simpsoni*, a semidomestic mosquito, transmits the virus from monkey to man and, at least in some areas, joins *Ae. aegypti* in transmission from person to person.

Dengue

Dengue is a mosquito-borne viral infection of the tropics and subtropics, classically characterized by fever and severe eye, joint, muscle, and bone pain—hence the name "breakbone fever." Skin rashes may occur. Though generally considered an acute, nonfatal disease, in some areas of the world dengue infection commonly results in severe, frequently fatal diseases known as "dengue hemorrhagic fever" and "dengue shock syndrome." *Aedes aegypti* is the most important vector.

Infection with any of the 4 known dengue serotypes may be asymptomatic, may cause nonspecific febrile or respiratory illness, or may cause "classical" dengue fever. In some patients a reticular, morbilliform, or macular rash develops on the 2nd to 5th day, occurring first on the trunk but later spreading to the arms, legs, and face. Itching of the palms and soles and generalized lymphadenopathy may also occur. A later petechial rash on the extremities, axillae, or in mucuous membranes may occur on the last day of fever in 20-70% of patients. The uncomplicated case rarely, if ever, fatal but the patient may suffer from continued fatigue and weakness for several weeks.

The syndromes "dengue hemorrhagic fever" (DHF) and "dengue shock syndrome" (DSS), occur primarily in children 2-6 days after the onset of fever and are manifest by vomiting, shortness of breath, enlarged liver, and bleeding in the skin, i the intestines, and from the gums. These hemorrhagic manifestations may progress to shock and death. It is likely that the DHF and DSS are related, with DSS repr senting the severe consequences of the hemorrhagic disease. The cause of these m

severe manifestations of dengue is unclear. The viral agents appear to be the same as those responsible for the classical nonfatal disease; however, further studies are required.

Diagnosis: The dengue viruses are classified as flaviviruses (group B arboviruses) and demonstrate considerable serologic cross-reactivity with other flaviviruses such as yellow fever, Japanese B encephalitis, and St. Louis encephalitis. There are 4 distinct serologic types of dengue virus (dengue 1, 2, 3, and 4). Infection with one serotype does not induce lasting immunity to others.

In individuals without prior flavivirus experience, the diagnosis of dengue can be established by the demonstration of seroconversion or a 4-fold rise in titer between acute and convalescent serum by hemagglutination inhibition (HI), complement fixation (CF), or plaque reduction neutralization tests. If 2 different serologic tests show a primary antibody response to a single dengue serotype, that serotype is probably the cause of the disease.

Subsequent infections with flaviviruses result in broadly cross-reactive results and are more difficult to interpret. Strict proof of causation demands virus isolation.

Epidemiology: Dengue virus has been reported most frequently between latitudes 25°N and 25°S. The disease is transmitted by mosquitoes of the subgenus *Stegomyia* with *Ae. aegypti* being the principal species involved in transmission. There is no significant reservoir amplifying bird or animal host although several species of monkeys are known to be susceptible to dengue. The endemic/epidemic cycle is human-mosquito-human.

After a person is bitten by an infected mosquito, the virus can be found in the blood (viremia) after about 5-6 days (prepatent period) at about the same time that the initial symptoms of the disease develop (incubation period). At this time, and for the next 4-5 days, the person is infective for the vector mosquitoes which may take a blood meal. After an incubation period of 2-15 days, generally 8-11 days, the female *Ae. aegypti* which acquires dengue virus by feeding on a viremic human is infective for life with the potential of transmitting the infection each time she feeds on a new human subject.

Historically, dengue has been seen in sweeping epidemics in many areas of the world. One of the earliest accounts of such an epidemic was that of Dr. Benjamin Rush who described a severe outbreak in Philadelphia during the summer and fall of 1780. Since the 18th century, numerous epidemics have occurred in tropical and neotropical areas throughout the world. Notable among these have been epidemics in the southern United States in 1922, with perhaps as many as 2 million cases, and in Greece in 1927-1928 with approximately 1 million cases. Although the last continental U.S. epidemic occurred in Louisiana in 1946, there have been epidemics in Puerto Rico in 1963, 1969, 1975, 1977, and 1978. Dengue has persisted as an endemic disease in Puerto Rico and other areas in and adjacent to the Caribbean, with occasional epidemics occurring throughout this region. The largest recent epidemic occurred in Colombia in 1972, with an estimated half-million cases. In Puerto Rico there have been estimates as high as 224,000 clinical cases (1,358 confirmed) in 1977 and 450,000 clinical cases (2,602 confirmed) in 1978.

Dengue continues to be endemic and epidemic in wide areas of south and southeast Asia and in the southern and western Pacific. In these regions all four serotypes are found and hemorrhagic manifestations of the disease are relatively common.

Until 1977, only dengue virus types 2 and 3 had been isolated in the Western Hemisphere, although there had been serologic evidence of dengue type 1 in older

individuals. However, since 1977 epidemics of dengue type 1 have been reported fro Puerto Rico, Jamaica, Virgin Islands, Belize, Guatemala, El Salvador, Honduras, Colombia, and Mexico. Of concern was the report in Puerto Rico in late 1975 of sev eral cases with more severe hemorrhagic manifestations than usual for this area and which were possibly definable as dengue hemorrhagic fever (DHF), the severe form o the disease which had not been previously reported from this area. Although borde line cases of DHF have been observed in Curacao, other cases have not been found i the Western Hemisphere.

Dengue is an ever present threat in any area within the range of the vector. The potential for the establishment of an epidemic from an imported case is obvious given the 5- to 6-day incubation period during which time an infected person may be completely asymptomatic, the rapid air transport now available, and the wide distri bution of the principal vector, *Ae. aegypti*. The recent rapid spread and propagati of dengue 1 epidemics in the Caribbean is testimony to the epidemic potential exist ing with the combination of a nonimmune population, a receptive vector, and importe cases. In 1978 alone, 89 confirmed cases of dengue were diagnosed in persons arri ing in the United States from the Caribbean area. In spite of this importation of cases, no introduction of transmission resulting in secondary cases was seen. Neve theless, with the probable continuing epidemic outbreaks in nearby areas, the risk introduction of dengue into the southeastern United States where *Ae. aegypti* popul tions are widespread, remains a significant potential health problem. Dengue also continues to be endemic and epidemic in wide areas of south and southeast Asia and the southern and western Pacific. In these areas *Ae. aegypti* is joined by several other *Aedes* species which may serve as efficient vectors. While importation of ca into the United States from these areas seems less likely, there were at least six confirmed cases imported into Guam from Vietnam in 1975 during the admission of refugees from that country; there was no transmission of the infection reported in Guam as a result of the case importation. However, it does seem likely that the occasional dengue epidemics in island situations in the southern Pacific area resu from the importation of a case or cases during the incubation period and subsequen infection of local mosquitoes.

Prevention and control: Because no practical immunization or therapeutic mea ures exist for preventing or treating dengue, control of this disease relies entir on the control of the mosquito vector, on the early identification of cases and, i possible, on the isolation of cases from contact with vector mosquitoes. At prese there is no vaccine available for dengue, although experimental attempts to develo a vaccine are being carried out.

While *Ae. aegypti* is recognized as the vector of dengue in the Western Hemi sphere, in other areas other mosquito species can transmit dengue virus. *Aedes albopictus* can be an efficient vector of dengue in all areas where both the mosqui and the disease appear together. This species breeds commonly in tree and rock ho as well as artificial containers and is widely distributed in south and southeast Asia and in many Pacific islands.

Aedes scutellaris consists of a complex of about 17 distinct but very closely related species known as the *scutellaris* group. Because *scutellaris* appears in large numbers and attacks humans readily and because other vectors are absent, it i regarded as a vector of dengue in New Hebrides and northern New Guinea. Members of the complex will breed in almost any small, well-shaded collection of clear rain water, both in ground pools and artificial containers.

BIOLOGY AND HABITS OF *AEDES AEGYPTI*

Aedes aegypti is a highly domestic mosquito. In the Western Hemisphere the species is closely associated with humans. Artificial containers, so abundantly provided by modern industrial society, are by far its most important breeding place and are essential to the production and maintenance of large populations of *Ae. aegypti*. Although treeholes and possibly other naturally occurring containers produce *Ae. aegypti*, the overwhelming majority come from auto tires, buckets, pet watering pans, tin cans, vases, jars, clogged roof gutters, and, in fact, from almost any man-made object that retains water and has walls other than soil.

Some containers are more attractive to the mosquitoes than others. Female *Ae. aegypti* are attracted to dark-colored containers with wide openings, especially when they are located in shaded areas. Dark-colored water and the presence of decaying leaves stimulate oviposition but odorous and highly polluted receptacles will be avoided.

Oviposition occurs mainly in the afternoon. If the walls of the container are very smooth (e.g., glass), the eggs may be scattered on the water surface but usually they are attached to the sides of the container at or near the water line. The eggs are less than 1 mm in length and are white at first, but within 2 hours they darken to an almost black color. At oviposition the embryos within the eggs are not yet ready to hatch. A period of 2-3 days at the high humidity near the water line is necessary for their full development to the larval stage. Should the eggs become dry during this developmental period, they will collapse and the embryos will die. By the time larvae are fully formed the eggs are resistant to dessication and can survive for periods of several months to more than a year. Under dry conditions, the dormant larvae within the eggs remain capable of hatching whenever the eggs are submerged by a rising water level and the consequent decreased oxygen supply furnishes the necessary hatch stimulus. Not all eggs hatch the first time they are flooded.

The larva which emerges from the ruptured egg shell is the first of 4 larval stages, each larger than the one preceding (Appendix I illustrates the characters used in identification of *Ae. aegypti* larvae and includes a pictorial larval key). During the course of its development the larva increases from about 1 mm to 6 or 7 mm in length. Passage from one larval stage (instar) to the next is achieved by the process of molting, during which the insect sheds its old exoskeleton (body covering). At molting, a fluid is secreted that allows separation of the exoskeleton from the newly developed body covering underneath. The head capsule and thorax of the exoskeleton split open and the larva emerges with a complete new body covering which allows for increase in size.

The larva spends most of its time feeding, using its fan-like mouth brushes to sweep microorganisms and particulate matter out of the water, browsing on submerged objects and on any organic material which accumulates on the sides and bottom of the container. Larvae of *Ae. aegypti* can be recognized by their characteristic sinuous swimming movements, avoidance of light, and the relatively blunt air tube which is their connection with the atmosphere.

Larval development normally requires 5-7 days and ends when the fourth stage larva molts to the nonfeeding pupal stage. Adverse conditions can greatly extend this time. Inadequate food supply also increases development time and results in undersized pupae and adults. Overcrowding of larvae, if severe, has a similar effect. Transformation from the larval to the adult form is completed during the 2- to 3-day pupal period.

CONTROL OF *AEDES AEGYPTI*

Approaches to Control

Because of the habitats of *Ae. aegypti*, methods for control of this mosquito vary from those used against other mosquitoes, especially in the pre-adult stages. Since most breeding occurs near or inside human dwellings, conducting effective control measures is both difficult and expensive and for these reasons there must be very strong justification before any control measures are begun. Such justification may be based on reduction of nuisance where *Ae. aegypti* are overly abundant, prevention of disease, or control of disease outbreaks.

Nuisance reduction: In most areas of its distribution, *Ae. aegypti* is not considered to be a major pest mosquito. However, where rainfall is frequent and artificial containers are abundant, populations occasionally become large enough to require local temporary control measures. Standard mosquito adulticiding methods such as use of thermal fogs or ultra-low volume (ULV) aerosols may be sufficient. Occasionally, it may be necessary to apply larvicides to breeding containers, especially to large accumulations such as discarded tires at service stations or tire-recapping plants. Education of property owners and/or enforcement of sanitation laws can be used effectively to eliminate any large accumulation of breeding containers. In residential areas where excessive numbers of containers are found on many of the premises, it may be necessary to conduct neighborhood source reduction campaigns.

Prevention of disease transmission: When there is reason to believe that dengue or yellow fever viruses are likely to be introduced via travelers from areas where outbreaks are occurring, efforts should be made to reduce *Ae. aegypti* populations to levels that are unlikely to support disease transmission. A premises infestation rate of 5% (larval infestation) has been said to be adequate to support transmission. However, factors such as quality of housing, number of residents, longevity of adult mosquitoes, and distance between houses could influence the likelihood of an area to support transmission if dengue or yellow fever were introduced. To have a margin of protection, larval infestations of the premises in a given area should be reduced to considerably less than 5%.

Education of the public in source reduction through the news media, civic organizations, and public schools may be adequate to achieve safe *Ae. aegypti* infestation levels in some areas. In others it may be necessary to implement active source reduction or larviciding programs.

During peak periods of *Ae. aegypti* production in the summer and fall months or until other control efforts have reduced larval infestations to safe levels, it may be necessary to use adulticiding methods to temporarily reduce the adult mosquito populations.

Control during disease outbreaks: Once the virus of dengue or yellow fever has been introduced and disease transmission is occurring, control efforts must be concentrated on adulticiding. With dengue, if cases are not numerous, spraying small areas surrounding each case using truck-mounted equipment may be sufficient. If cases are too numerous for localized treatment, use of aerial spray applications may be necessary to bring the outbreak under control. Should urban yellow fever occur, the entire urban area should be sprayed at least once every 3-4 days until vaccination against the virus is accomplished or until cases are no longer reported.