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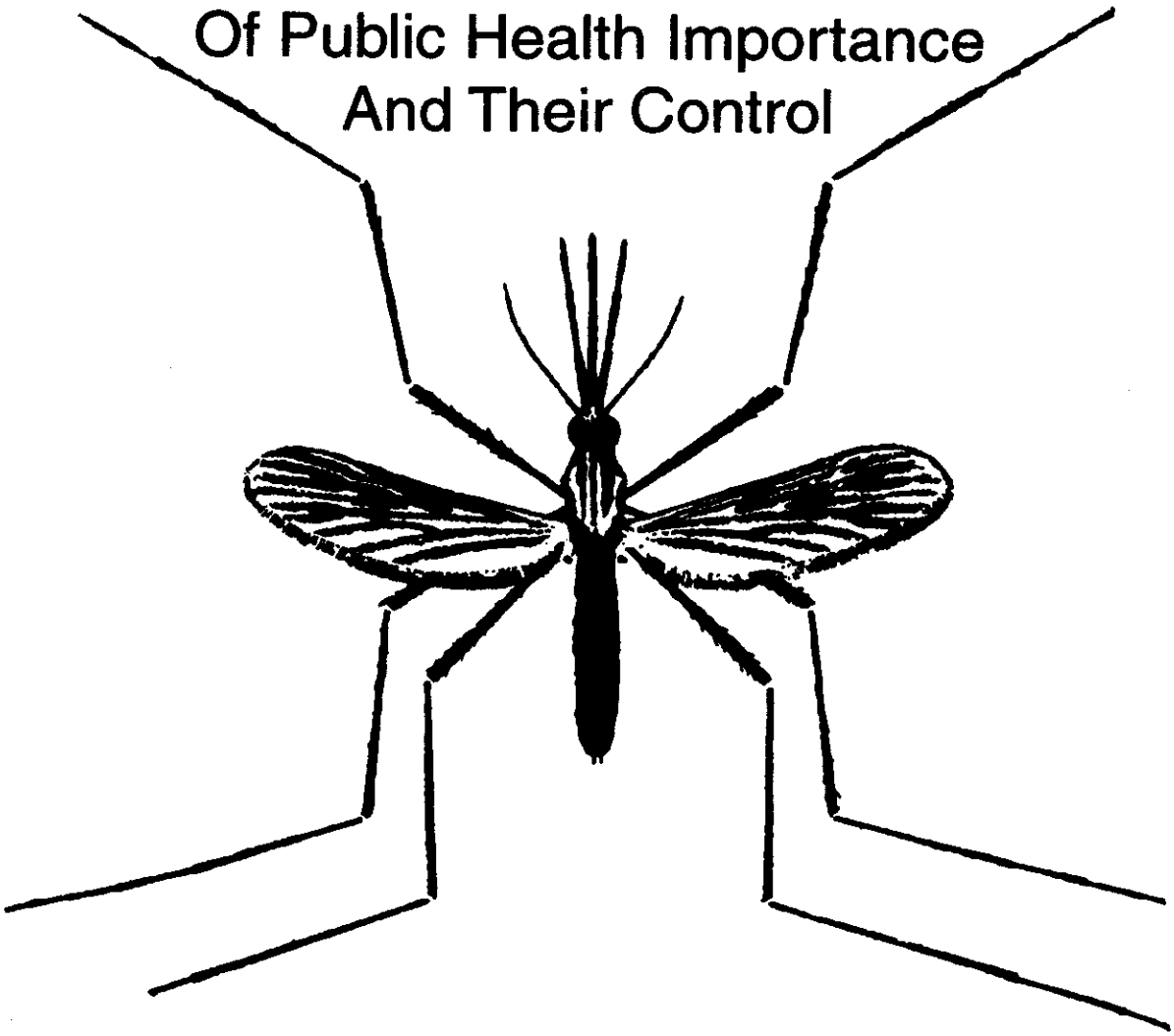
Vector-Borne Disease Control

MOSQUITOES

Of Public Health Importance And Their Control



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MOSQUITOES
Of Public Health Importance And Their Control

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CONTENTS

INTRODUCTION

Throughout history mosquitoes have occupied a position of importance as a pest of mankind, but not until the late 19th century were these arthropods identified as the agents responsible for transmission to man of some of his more devastating diseases. During subsequent years, knowledge of the relationship of mosquitoes to disease has expanded; and knowledge of methods for controlling these disease vectors has often provided us with means toward reducing or eliminating the diseases in many areas. In this manual the importance of mosquitoes to human health in the United States is considered, along with basic information on mosquito, identification, biology, and control.

MOSQUITOES AS DISEASE VECTORS

World-wide, mosquitoes are responsible for the transmission of disease to millions of people each year. These diseases include encephalitis, dengue, yellow fever, malaria, and filariasis. In the past, most of these diseases have been highly important as endemic or epidemic diseases in the United States, but presently only the arthropod-borne encephalitides continue to occur with some frequency in this country.

ENCEPHALITIS

A high proportion of the arthropod-borne viruses known to affect humans are transmitted to humans by mosquitoes. Several of these viruses are responsible for encephalitis, a disease affecting the central nervous system. The five major types of arboviral encephalitis in the United States are: Eastern equine encephalitis (EEE), Western equine encephalitis (WEE), St. Louis encephalitis (SLE), LaCrosse encephalitis (LAC) and Venezuelan equine encephalitis (VEE); each is caused by a distinctly different virus or virus complex. These viruses are normally infections of birds or small mammals. During the course of such infections the level of virus may increase (amplification), facilitating further extension of transmission. The occasional infection of human or equine hosts may result in severe illness or death. In most cases, the human or equine host is a "dead end" for the virus, with little or no possibility of subsequent transmission of the disease from these hosts because of their inability to infect mosquitoes.

The viruses that cause Eastern equine, Western equine, or St. Louis encephalitis are normally transmitted from bird to mosquito to bird, and less commonly from bird to mosquito to human or horse. The viruses that cause LaCrosse encephalitis and Venezuelan equine encephalitis are normally transmitted by mosquitoes among small mammals, but occasionally are transmitted to humans or, in the case of VEE, to horses. Because of the higher VEE viremias common in infected horses, these hosts may provide a source for the mosquito transmission of the virus to new hosts.

Human cases of arbovirus infections range from inapparent or mild to very severe illnesses which may permanently damage the central nervous system and, in some instances, are fatal. Similarly, horses may have mild to severe or fatal infections with EEE, WEE, or VEE viruses. SLE virus causes only asymptomatic infections in horses (they develop antibodies but not clinical illness). Birds may die of infection caused by some

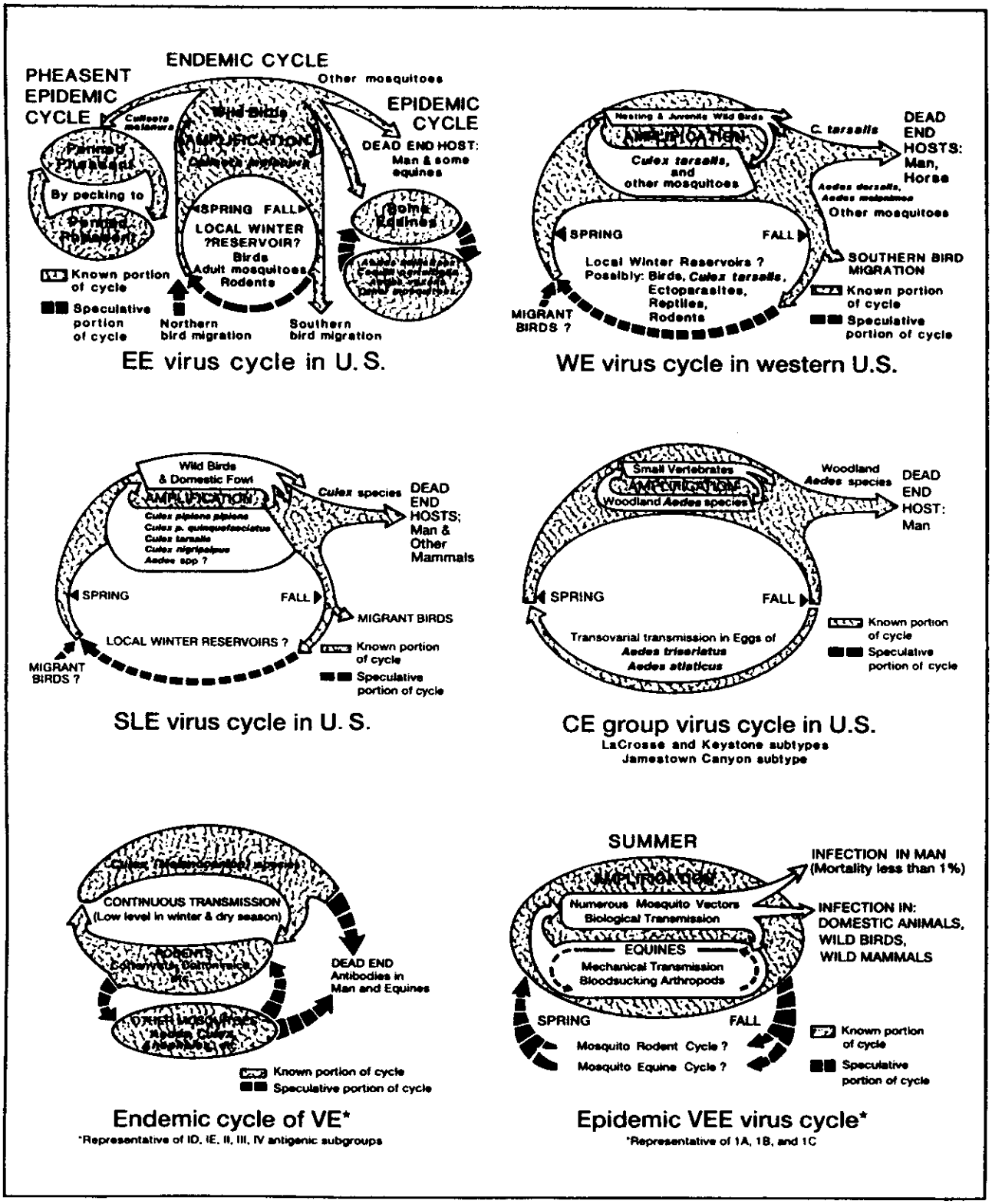

Figure 1. North American Mosquitoborne Arboviral Encephalitis Cycles

encephalitis viruses but not by others. For example, deaths due to EEE virus have been reported in red-winged blackbirds, house sparrows, whooping cranes, and pheasants. SLE virus produces no outward signs in birds.

Schematic illustrations of the basic transmission cycles of the five major types of encephalitis viruses of North America are presented in Figure 1. Portions of some of these cycles are still speculative and await further confirmation. The geographic distribution of four of these viruses is shown in Figure 2. The human cases of four major mosquito-borne viral encephalitides of North America reported by the states to the Centers for Disease Control and Prevention since 1970 are summarized in Table 1.

TABLE 1. REPORTED CASES OF ARBOVIRAL ENCEPHALITIS IN THE U. S.

	TYPE OF ENCEPHALITIS				
YEAR	EEE	SLE	WEE	LAC/CE	TOTAL
1970	2	15	4	89	110
1971	4	57	11	58	130
1972	0	13	8	46	67
1973	7	5	4	75	91
1974	4	74	2	30	110
1975	3	1815	133	160	2111
1976	0	379	1	47	427
1977	1	132	41	65	239
1978	5	26	3	109	143
1979	3	32	3	139	177
1980	8	125	0	49	182
1981	0	15	19	91	125
1982	12	34	9	130	185
1983	14	19	7	64	104
1984	5	33	2	89	129
1985	0	21	1	68	90
1986	1	43	7	64	115
1987	3	17	41	87	148
1988	1	0	0	41	1
1989	9	34	0	65	108
1990	5	240	0	62	245
1991	12	70	1	6	89
TOTALS	99	3199	297	1531	5126

Eastern Equine Encephalitis

Eastern equine encephalitis (EEE) is found along the Atlantic and Gulf Coasts and inland in limited areas in New York and the Midwest. As will be noted in Table 1, cases usually occur in small numbers in any one year. Cases generally occur in the late summer and are concentrated in the young age groups. There were localized epidemics in Massachusetts in 1956 (12 cases), and in New Jersey in 1959 (32 cases); 1968 (12 cases); 1982 (12 cases) and 1983 (14 cases). Eastern equine encephalitis is the most deadly of the North American mosquito-borne viral diseases, with 50 to 75% of the human cases ending fatally and leaving a high proportion of the survivors with severe central nervous system sequelae. The disease often occurs in horses and in game farm pheasants and may be fatal in up to 90% of these animals. The bird-to-bird cycle of the virus is typically maintained by *Culiseta melanura,* a mosquito whose larvae are found in freshwater swamps. Because this mosquito rarely bites humans or horses, other mosquitoes, such as the eastern salt marsh mosquito *Aedes sollicitans, Coquillettidia pertubans,* and *Aedes vexans,* are probably the vectors responsible for transmission of the disease to these dead end hosts.

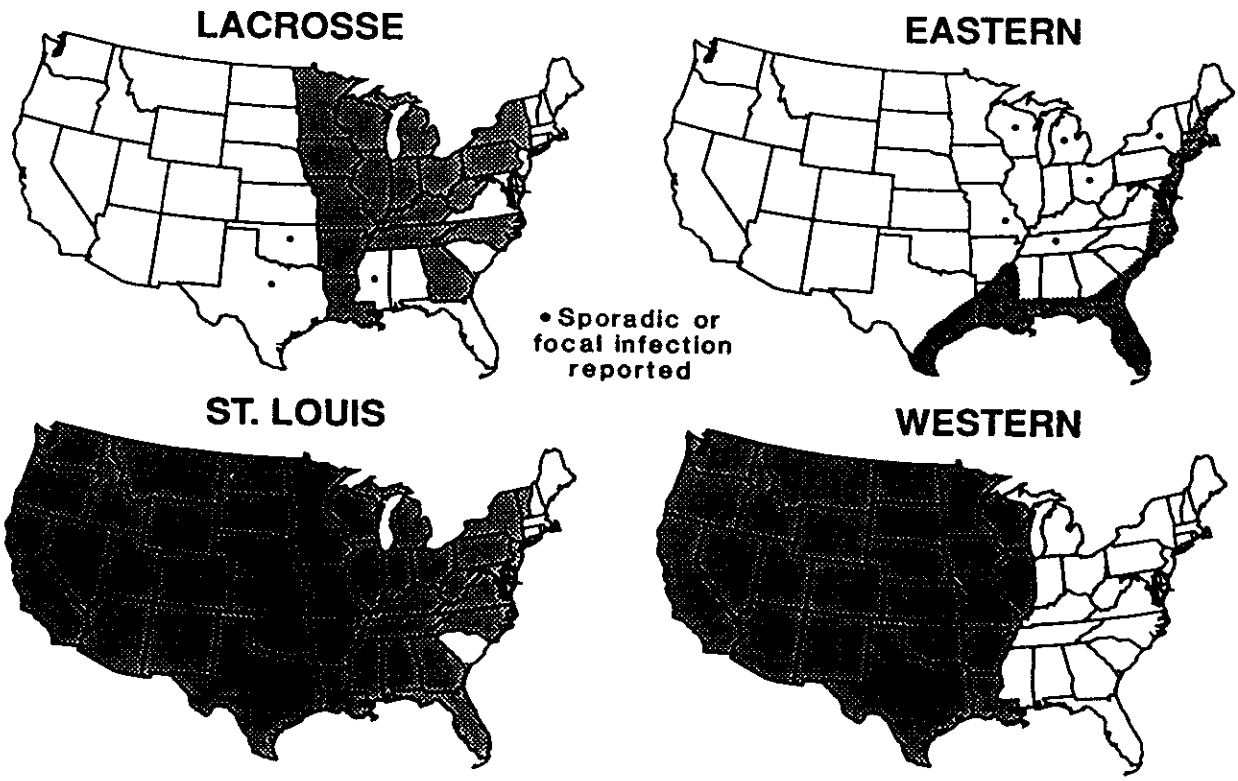

Figure 2. Distribution of 4 Types of Mosquitoborne Arboviral Encephalitis

Western Equine Encephalitis

Western equine encephalitis (WEE) is found in the states west of the Mississippi River, Wisconsin, and Illinois, as shown in Figure 2. As in Eastern equine encephalitis, cases are concentrated in the young. There were many major outbreaks in horses in the 1930's, with thousands of cases and many deaths. The largest human epidemic, probably over 3,000 cases, occurred in the western United States in 1941. Another large epidemic occurred in 1952, primarily in the Central Valley of California (Reeves 1990). There were 141 cases in 1958 and 172 cases in 1965. In 1975 133 cases were reported, primarily in the Red River Valley of Minnesota and South and North Dakota. While deaths have been reported in human cases of WEE, it is generally a much milder disease than EEE. Mortality has been reported as ranging from 1 to 5% of the cases. The death rate in horses is considerably higher.

Culex tarsalis and *Aedes melanimon* are the important mosquito vectors of Western equine encephalitis. Isolations of WEE virus have been made from many species of mosquitoes and birds. (Reisen and Monath, 1988).

St. Louis Encephalitis

St. Louis encephalitis (SLE) was first recognized during an epidemic in the St. Louis, Mo., area in 1933, although retrospectively the first known outbreak of the disease was found to have occurred in 1932 in Paris, Illinois. Since that time, human cases of SLE have been reported from all of the contiguous states, with the exception of the New England area and South Carolina (Fig. 2). (Monath, 1980; Chamberlain, 1987; Tsai and Mitchell, 1988).

The largest number of cases of SLE on record for a single year occurred in 1975 when 1815 cases were reported from 30 states, the majority of them in the Ohio and Mississippi valleys. Human cases of SLE typically occur in late summer and fall. During the Florida epidemic of 1990, in order to prevent infection from night-biting mosquitoes, people were advised to stay indoors after dark and many football games were rescheduled for afternoon playing.

The following major outbreaks have occurred:

Location	Date	Cases	Deaths
St. Louis, MO	1933	1,097	221
St. Louis, MO	1937	431	107
Hidalgo Co., TX	1954	373	10
Louisville, KY	1956	110	12
Cameron Co., TX	1957	114	3
Tampa Bay area, FL	1962	222	43
Houston, TX	1964	243	27
Dallas, TX	1966	172	22
Florida	1990	226	10

St. Louis encephalitis is considered to be a more serious disease than Western equine encephalitis but less so than Eastern equine encephalitis. The disease is more commonly seen and is more severe in older people. Case fatality rates for most outbreaks of SLE have varied from 2 to 20%, and neurologic sequelae have been reported in a small percentage of cases. However, most infections of SLE virus in humans do not result in illness, and many mild cases show aseptic meningitis or fever only.

St. Louis encephalitis virus exists primarily as an infection of birds transmitted by mosquitoes. It may be enzootic in some areas but occasionally epizootics may occur in which a large percentage of birds in the area become infected. Birds develop the levels of viremia needed to infect mosquitoes but do not suffer ill effects from the disease. Humans and horses acquire SLE infections from mosquitoes which have previously fed on infected birds, but apparently do not develop a sufficient amount of virus to infect mosquitoes that feed

on them.

Members of the *Culex pipiens* complex, *Culex pipiens pipiens* (the northern house mosquito) and *Culex pipiens quinquefasciatus* (the southern house mosquito) are the main urban vectors of St. Louis encephalitis. *Culex tarsalis* is the chief vector in rural areas in western states. *Culex nigripalpus* is the main vector in Florida.

Culex salinarius and *Culex restuans* are potential vectors in some localities, but this observation requires confirmation.

LaCrosse Encephalitis/California Encephalitis

Transmission of LaCrosse (LAC) and other California serogroup viruses including California encephalitis (CE) and Jamestown Canyon (JC) to humans is rather constant. As shown in Table 1, there are about 75 reported cases nationally (range 30 to 160) each year. Routine testing for and reporting of California serogroup encephalitis was initiated widely in the United States beginning in 1963. "Between 1963 and 1984, 1611 cases of CNS (central nervous system) illness, serologically confirmed as CE, were reported to CDC...., the vast majority of these cases undoubtedly resulted from infection with LAC virus." (Grimstad, 1988). The greatest number of cases of LaCrosse encephalitis have been reported in the midwestern states of Ohio, Indiana, and Wisconsin, in August and September, usually in children under 16 years of age, and more often in males than in females. As shown in Table 1, each year from 1970 to 1991 more cases of LAC/CE were reported than cases of EEE or WEE, and often than cases of SLE. Fortunately infections with LAC virus cause a less severe illness than is commonly found with other mosquito-borne encephalitides. The mortality rate is probably in the order of 4 per 1,000.

Human cases of California encephalitis (CE) have been reported from California where the CE virus has been isolated from *Aedes melanimon* mosquitoes. Human cases of Jamestown Canyon (JC) encephalitis have been reported from Michigan, New York, and Ontario. *Aedes stimulans* may be the principal vector of JC virus. Other viruses in the California encephalitis serogroup include Keystone and Trivittatus--usually with *Aedes* mosquito vectors.

LaCrosse encephalitis differs from Eastern equine, Western equine, and St. Louis encephalitis in that it has mammalian, rather than an avian, vertebrate host system. The major mammalian hosts are the eastern chipmunk, tree squirrels, and foxes. The principal vector is the eastern treehole mosquito, *Aedes triseriatus*. The virus has been shown to overwinter in the eggs of the treehole mosquito and has been passed through the eggs to larvae, pupae, females and males (Watts *et al.* 1973). The virus is maintained in woodland habitats and even within small, isolated woodlots, which are quite numerous in some north central states. There is a strong association between the occurrence of human LAC encephalitis cases and the presence of *Ae. triseritus* in artificial containers, such as tires, on patients' premises. Detailed accounts of the California encephalitis group, including data on human cases and isolations of the various subtypes from many species of mosquito and vertebrate hosts, has been published (Sudia *et al.* 1971 and Calisher and Thompson 1983).

Venezuelan Equine Encephalitis

Venezuelan equine encephalitis (VEE) is caused by a number of subtypes of virus. The endemic Everglades virus (EV) in Florida is of low virulence and is transmitted by *Culex (Melanoconion)* mosquitoes from wild rodents. Three clinical human cases have been reported in southern Florida (Ventura *et al.* 1974). In

addition, antibodies to Everglades virus have been found in Seminole Indians, hunters, fishermen, and campers in localized areas in southern Florida. The virulent epidemic type 1A and 1B viruses have been involved in extensive epizootics in horses and epidemics in humans in tropical America since 1935.

In 1971 an outbreak of the 1B type of VEE occurred in Texas, in association with a similar outbreak in Mexico, and more than 1500 equines were fatally infected. Although only 19 human cases of VEE were reported to the Center for Disease Control during 1971, one authority places the number of cases in Texas at 110, with no deaths (Bowen *et al.* 1976). *Psorophora columbiae, Ps. discolor, Aedes sollicitans,* and *Ae. thelcter* were the primary vectors in South Texas during the 1971 epidemic. Other species of *Culex, Anopheles, Mansonia* and *Deinocerites* were also found infected in Texas in 1971, and may be important in other countries. The Texas epidemic was controlled by a three-fold program involving (a) vaccination of equines with live, attenuated vaccine to protect horses and to preclude them as a source of virus for mosquito vectors; (b) quarantine of equines in Texas and adjoining states; and (c) ultra-low volume aerial application of technical malathion to some 13.5 million acres of mosquito-producing area along the Mexico-Texas border and the Gulf Coast of Texas and Louisiana (Sudia and Newhouse 1975). No further Venezuelan equine encephalitis activity has been reported in equines or humans in the United States during the period of 1972 to 1975.

DENGUE

Dengue is a viral disease transmitted from person to person by mosquitoes. It may occur in epidemic form in almost any part of the tropics or subtropics. It is usually an acute, nonfatal disease, characterized by sudden onset of fever, headache, backache, joint pains, nausea, and vomiting. There are four virus serotypes (DEN 1-4). Three of the four serotypes (DEN-1, DEN-2, and DEN-4) have been circulating in the Americas since 1981. Although endemic transmission of DEN-3 has not occurred in the region since 1977, it could be reintroduced. While most infections result in relatively mild illness, some may cause the severe forms of the disease, dengue hemorrhagic fever (DHF) characterized by severe rash, purpura, mild gum bleeding, nosebleeds, gastrointestinal bleeding and circulatory failure resulting in dengue shock syndrome (DSS) and even death.

In the Western Hemisphere the principal and perhaps the only mosquito responsible for transmission of dengue in human populations has been *Aedes aegypti*. *Aedes albopictus* has been a vector in Hawaii, the Philippines, and Southeast Asia and may become one in the New World. The mosquitoes obtain the virus from the blood of infected persons during the period from the day before the initial fever through the third or fourth day of illness. The virus multiplies in the mosquito, which becomes infective to man 8 to 14 days after the infective blood meal. The mosquitoes may remain infective for the rest of their lives with the ability to transmit the disease-causing virus during subsequent blood feedings on humans. (Gubler, 1988).

Dr. Benjamin Rush reported one of the earliest outbreaks of dengue in the United States from Philadelphia in 1780. In 1922 there was a large epidemic from Florida to Texas with an estimated 2 million cases, and with perhaps a million cases in Texas alone. Cases of dengue were reported in Louisiana in 1945. Dengue epidemics spread from Jamaica in 1977, to Honduras in 1978, to Mexico in 1979, and to Texas in 1980 where 27 cases were reported in people who apparently had not been out of the country. Cases of dengue contracted overseas but occurring in the

United States have been reported for many years, as 102 cases in 1990 (CDC, 1991).

Epidemics of dengue, some with cases of dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) have occurred in the Americas. The estimated cost of the DEN-2 and DEN-3 epidemic in Puerto Rico in 1977 was between $6 and $16 million (U.S.) in medical costs, lost work, and control measures. The 1981 DHF epidemic in Cuba had an even greater economic impact with an estimated cost of over $100 million (U.S.) in control measures and medical costs during a 4-month period from July through October. Gubler (1988) reported 502,026 cases in 1977 in the Americas and 362,398 in 1981 with 116,143 persons hospitalized and 158 deaths, with most of the 1981 cases from Cuba. Some authorities have theorized that the severe hemorrhagic forms of the disease (DHF and DSS) occur only in persons experiencing a second infection with a different (heterologous) dengue serotype. For example, the 1981 Cuban epidemic of DHF caused by DEN-2 virus was preceded by a large DEN-1 epidemic in 1977.

Dengue is endemic in Puerto Rico with 11 outbreaks since 1963. Starting in 1986, from 6,000 to 11,000 cases of dengue-like illness have been reported each year in Puerto Rico.

The most recent major epidemic of dengue in the Americas occurred in Peru in 1990, with 76,000 cases reported. The most recent outbreak of dengue hemorrhagic fever (DHF) occurred in Venezuela in 1989-90 involving 3,108 cases of severe dengue and 73 deaths (CDC, 1991).

YELLOW FEVER

Yellow fever is a viral disease which is transmitted to humans by mosquitoes. Illness from this infection may be acute and fatal or so mild as to be inapparent. There are two distinct epidemiologic types of the disease in the Americas: Urban yellow fever and jungle yellow fever. In both the virus is the same, and humans are protected from each by the same vaccine; however, the mosquito vectors and normal vertebrate hosts differ.

Urban Yellow Fever

In the classical urban type of yellow fever, epidemics are the result of human-to-human transmission of the virus by *Aedes aegypti,* commonly called the yellow fever mosquito. Although no epidemics have occurred in the United States since the outbreak of 1905 in New Orleans, epidemics were reported with some frequency during the 18th and 19th centuries from most of the larger seaports in the eastern United States, even as far north as Philadelphia, New York, and Boston. These epidemics probably originated from the importation of cases from infected areas of Central and South America. Although *Aedes aegypti* is commonly found in many areas of the United States, importation of cases leading to the establishment of epidemics in this country is a theoretical but highly remote possibility because of vaccine usage and the absence of urban yellow fever elsewhere in the Americas.

Jungle Yellow Fever

Jungle yellow fever, also called sylvan or sylvatic yellow fever, is normally a disease of monkeys and perhaps other wild animals, transmitted most frequently by sylvan, treetop-frequenting mosquitoes in the genera *Haemagogus, Sabethes* and *Aedes*. Enzootic and epizootic yellow fever is maintained in the jungle primates by these vectors. Humans usually become involved when they invade jungle habitats for occupational or other purposes and are bitten by the sylvan vectors. Epizootics continue to occur in jungle areas. The most recent extension of activity toward the United States occurred in 1956 (Trapido

and Galindo 1956) reaching as far north as Mexico. The yellow fever virus may be highly fatal to some of the species of monkeys involved in the jungle cycle, while others develop viremias with little or no apparent illness. The potential for initiation of urban transmission of the disease depends on the return of a human who has become infected in the forested area to an area where *Aedes aegypti* is present to initiate and maintain human-to-human transmission. This has occurred only rarely in the Americas and has in no case resulted in an epidemic. Establishment of the urban cycle may occur with some frequency in Sudan, Ethiopia and other African areas (Monath 1988).

MALARIA

Although malaria had disappeared as a significant problem within the United States by the early 1950's, it is still one of the most important communicable diseases on a worldwide basis. Malaria in humans is an acute or chronic disease caused by any one of four species of microscopic protozoan parasites in the genus *Plasmodium (P. vivax, P. falciparum, P. malariae* and *P. ovale)*. The parasite is transmitted from person to person by the bite of *Anopheles* mosquitoes. Although there are 17 species of *Anopheles* in the United States, only three served as significant vectors of the disease: *An. quadrimaculatus* east of the Rocky Mountains, *An. freeborni* and *An. hermsi* in the West.

Malaria was the number one insect-borne disease in the United States from colonial days until the late 1940's. As shown in Figure 3 malaria control has been a continuing battle from 1930 to the present time. During the Great Depression of the 1930's over 100,000 cases were reported each year, but the number was probably grossly underreported. In the late 1930's the TVA, WPA, and other governmental agencies began area-wide malaria control programs and reduced the number of cases to about 60,000 a year at the beginning of World War II. During World War II, a cooperative program of PHS and state and local health departments, the Malaria Control In War Areas Program (MCWA), helped control this disease, but the number rose at the end of the war due to cases contracted overseas in the China-Burma-India and Mediterranean theaters of war. As the result of a Malaria Eradication Program from 1945-1952, the number of cases dropped dramatically, from over 60,000 cases in 1945 to 2,184 in 1950 (Fig. 3).

As Veterans returned from the Korean conflict, many came down with malaria. The number of cases rose to 5,600 in 1951 and peaked at 7,023 in 1952, of which approximately 50 were transmitted in the United States. As the number of veterans returning from malarious areas decreased, the number of cases of malaria decreased, reaching a low of 132 in 1957. Beginning in 1965 soldiers returning from Vietnam came down with malaria in this country and the number of cases of malaria rose to a peak of over 4,000 cases in 1970. Following President Carter's decision to admit up to 14,000 Asian refugees a month, the number of cases rose to 1,864 in 1980, due in part to infected Indochinese refugees. Since 1980 about a thousand cases of malaria have been reported each year, almost all of them contracted overseas. However, migrant agricultural workers from Mexico (some suffering from malaria) came across the border and slept outdoors in California where they were bitten by malaria mosquitoes which later transmitted malaria to other migrants and residents who had never been out of the country. In San Diego county, California 27 malaria cases were reported in 1986 and 30 cases in 1988.

The malaria parasite inhabits the human red

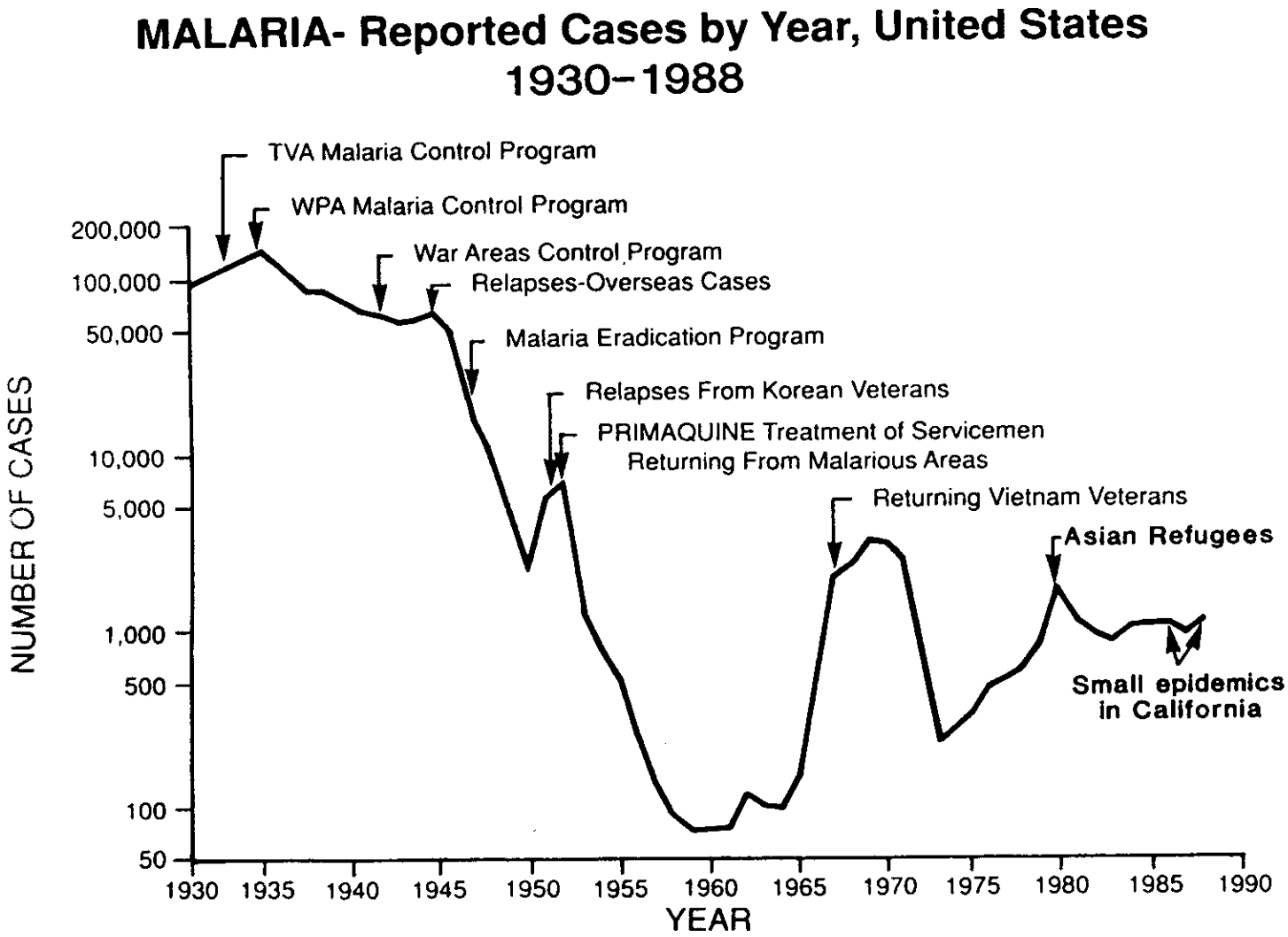

Figure 3. Malaria -- Reported Cases by Year, United States. 1930-1988

blood cells, where it multiplies asexually over a period of about 48 or 72 hours, depending on the species. After reaching maturity it bursts from the red cell, releasing a large number (usually 8 to 20) new parasites which enter new red cells and reinitiate the cycle. Typical malaria symptoms, chills and fever, are associated with the rupturing of infected red cells and release of the parasites. This process is often synchronous, which accounts for the intermittent nature of the symptoms, often occurring at about 48- or 72-hour intervals (the so-called "tertian" or "quartan" cycles). In addition to this asexual cycle in humans, some of the parasites develop into sexual forms, the male and female gametocytes. Infection of the mosquito takes place when a receptive anopheline feeds on an infected person who is carrying the gametocyte stages. The parasite then undergoes an asexual cycle in the mosquito which requires 7 to 20 days and culminates with the invasion of the mosquito salivary gland with numerous microscopic, spindle-shaped forms known as sporozoites. The human infection is initiated when these forms are injected during the bite of the infected mosquito. Before the asexual cycle in the red cells is established, the parasite must complete a 5- to 10-day period of multiplication in cells of the liver.

Clinically, the malaria infection varies from a moderately severe to a highly fatal illness, depending on the species of parasite and the host's condition. In the absence of prompt treatment, the so-called "malignant tertian" malaria, caused by *P. falciparum,* is particularly severe and often fatal in infants and young children and in those adults who possess no partial immunity due to previous malaria experience. The benign tertian parasite, *P. vivax,* generally causes a less severe illness and a lesser rate of mortality. There appears to be no true "immunity" conferred by a malaria attack; persons in a highly endemic area may be infected over and over again, usually developing a tolerance for the parasite which prevents severe clinical consequences, but which does not prevent the continuation of a chronic, often debilitating infection.

If insufficiently treated, a malaria infection may persist in humans for many months or years and have a continuing or periodically renewed ability to infect mosquitoes, often in the absence of symptoms or with a less severe clinical attack. For two of the species, *P. vivax,* and *P. ovale,* the continuation of parasites in the liver cells for prolonged periods may give rise to relapses by reinvasion of the red blood cells from this source at intervals for several years after the initial infection. There is probably no such true relapse mechanism for *P. falciparum* or *P. malariae*. However, in the case of *P. malariae* low-grade parasitemias may continue to be present in persons who have developed a tolerance for the infection. These parasitemias are characteristically asymptomatic or with infrequent mild symptoms and are less likely to be treated with a specific drug for malaria than would be the primary symptomatic attack and, therefore, from time to time may provide an unsuspected source of infection to mosquitoes. The remaining hazard of malaria transmission within the United States stems from the possible importation of such cases, or of cases recently acquired in endemic areas but not yet clinically apparent, into receptive areas of the country.

Despite the widespread presence of anopheline mosquitoes throughout the United States, a highly susceptible human population, and the importation of thousands of cases of malaria acquired overseas during World War II and the conflicts in Korea and Vietnam, there have been reported between 1964 and 1990 a total of 79 cases in 16 episodes of introduced malaria, i.e., malaria due to local transmission from an infected individual who

came into the United States. All cases of malaria in the United States which are suspected as introduced cases are carefully investigated by state and/or federal health authorities to determine the exact origin. In the event of introduced malaria, immediate steps are necessary to prevent further spread, and these include detection and treatment of cases as well as mosquito surveillance and, if warranted, localized mosquito control measures.

FILARIASIS

The World Health Organization estimates that at least 250 million people are infected with the filarial nematode parasites *Wuchereria bancrofti* and *Brugia malayi,* transmitted by mosquitoes (WHO 1974). The adult worms live in various parts of the human lymphatic system, causing the diseases known as Bancroftian and Brugian filariasis. Persons may harbor the parasites with no apparent symptoms, or the filarial worms may cause inflammation and other complications. In some persons who have had prolonged and repeated infections, there may be extreme enlargement of the external genitalia, breasts, or legs, hence the clinical term elephantiasis for pronounced enlargement of parts of the body, often with a thickened rough skin.

The young filarial worms are transmitted from person to person by various species of mosquitoes. These nematodes undergo developmental changes in the mosquito which is an essential link in the cycle of transmission. The immature worms, called microfilariae, occur in the human bloodstream. Here they are picked up by mosquitoes as they feed. A minimum period of 10 to 11 days is required for the developmental stages in the mosquito thorax before infective forms migrate to the mosquito proboscis, from which point they reach the new host at the next feeding. The infective filariae are not injected into the new host by the mosquito but actively penetrate the skin, perhaps at the site where the mosquito punctured the skin. Important vectors of *Wuchereria bancrofti* include species of the genera *Culex, Aedes,* and *Anopheles*. The generally accepted vectors of *Brugia malayi* are mosquitoes in the genus *Mansonia*.

Filariasis is widespread in many tropical and subtropical regions throughout the world. The Bancroftian type of filariasis is apparently an increasing public health problem in many of the larger cities in southeastern Asia. In the Western Hemisphere it occurs in the West Indies, Venezuela, Panama, and the coastal portions of the Guianas and Brazil. Filariasis control programs are based on control of the mosquito vectors and treatment of the human cases with diethylcarbamazine (Hetrazan).

A small endemic center existed for many years near Charleston, SC, but this has now disappeared. In many parts of the United States, people who have recently left the tropics may have the immature worms circulating in their blood. However the disease is not known to be naturally acquired in the United States at the present time.

DOG HEARTWORM

The dog heartworm *(Dirofilaria immitis)* is a common mosquito-transmitted filarial parasite of dogs which causes serious disease in these animals along the Atlantic and Gulf Coasts from Massachusetts to Texas and inland at least to Illinois and Minnesota. This nematode is also an occasional parasite of man; during the past 15 years at least 35 human cases have been reported in the United States (Gershwin *et al.* 1974). The parasite in man is usually located in the lung and less often in the heart. Other than as a lesion which may simulate other diseases in medical diagnosis, its clinical significance in man has not been fully determined.

The mosquito vectors of dog heartworm are

not definitely known, but a list of infected mosquitoes has been published (Ludlam *et al.* 1970), and includes *Culex salinarius, Aedes aegypti* and *Ae. taeniorhynchus. Aedes canadensis* may be an important vector in the northeastern United States. *Aedes sierrensis* is a vector in California and Utah. *Aedes albopictus* is a competent vector of *D. immitis.*

GENERAL CHARACTERISTICS AND LIFE CYCLE OF MOSQUITOES

Mosquitoes are small, long-legged, two-winged insects belonging to the Order Diptera and the Family Culicidae. The adults differ from other flies in having three characters in combination: long, many-segmented antennae; an elongate proboscis; and scales on the wing veins and wing margin. This is a very large group containing over 3000 species. There are approximately 165 species and subspecies in North America north of Mexico belonging to 13 genera distributed among 3 subfamilies according to Darsie and Ward (1981). Their general classification of the mosquitoes occurring in this area is outlined below:

Order Diptera (flies and mosquitoes)
Family Culicidae (mosquitoes)
Subfamily Anophelinae (anophelines)
Genus *Anopheles*-17 species
Subfamily Culicinae (culicines)
Genus *Aedes-79* species and subspecies
Genus *Coquillettidia* (formerly *Mansonia*) 1 species
Genus *Culex-29* species and subspecies
Genus *Culiseta*-8 species
Genus *Deinocerites*-3 species
Genus *Haemagogus*-1 species
Genus *Mansonia*-2 species
Genus *Orthopodomyia*-3 species
Genus *Psorophora*-15 species
Genus *Uranotaenia*-3 species and subspecies
Genus *Wyeomyia*-4 species
Subfamily Toxorhynchitinae
Genus *Toxorhynchites* (formerly *Megarhinus*)-2 subspecies

LIFE HISTORY

Mosquitoes have four distinct stages in their life history, the egg, larva, pupa, and adult (Fig. 4). The first three stages occur in water, but the adult is an active flying insect that feeds upon the blood of man and animals or upon plant juices.

Eggs

Eggs are white when first deposited, becoming dark within an hour or two. In general, mosquito eggs fall into three distinct groups: (a) those laid singly on the water surface: (b) those laid together to form rafts which float on the water surface; and (c) those laid singly on damp soil or vegetation. These differences are reflected in the structure of the egg.

Anopheline eggs are laid singly on the water surface. They are elongate oval, usually pointed at one end and provided with a pair of lateral floats (Fig. 4). They average about one-half millimeter in length. Hatching usually takes place within 2 or 3 days. The eggs of *Toxorhynchites* are also laid singly on the water surface where they are kept afloat by means of air bubbles which form among the spines on the egg shell. The eggs of *Culex, Culiseta, Coquillettidia, Mansonia* and *Uranotaenia* are laid side by side to form a raft often containing 100 or more eggs. They remain afloat on the surface of the water until