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Part I

VIRUS DISEASES TRANSMITTED BY MOSQUITOES AND OTHER ARTHROPODS

HARRY D. PRATT*

The arthropod-borne viruses (arboviruses) have been defined as true viruses biologically transmissible by arthropods (primarily mosquitoes and ticks) to vertebrates (man, mammals, birds, and reptiles). At the present time about 160 arboviruses are known for the entire world, approximately 60 of which are important

to human health. Twenty-one of these arboviruses have been found in North America since 1900, and eleven of them have been found to affect man (Table 1). These viruses cause benign and inapparent infections, mild or severe sickness, including encephalitis, or death. Ten of these viruses have been found in arthropods and

TABLE 1.—Arboviruses in North America Associated with Human Illness.

Virus	Serological group	Vector	Syndromes
Eastern encephalitis	A	Mosquitoes	Encephalitis
Western encephalitis	A	Mosquitoes	Encephalitis
Venezuelan equine encephalitis	A	Mosquitoes	Encephalitis
St. Louis encephalitis	B	Mosquitoes	Encephalitis
Yellow fever	B	Mosquitoes	Fever, malaise, sometimes death
Dengue	E	Mosquitoes	Fever, malaise, joint pain
Powassan	B	Ticks	Encephalitis
Rio bravo	B	Unknown	Fever, headache, malaise, joint pain
California	California	Mosquitoes	Serologically association with encephalitis
Colorado tick fever	Ungrouped	Ticks	Fever, malaise, headache, sometimes encephalitis
Vesicular stomatitis	VSV	Mites	Fever, malaise

* Invitational Paper: American Mosquito Control Association, Annual Meeting, Chicago, Illinois, March 2, 1964. H. D. Pratt is Chief, Training and Communications Section, *Aedes aegypti* Eradication Branch, CDC, Public Health Service, US DHEW, Atlanta, Ga.

other animals, but not in man (Table 2). One of these viruses causes blue tongue disease of sheep. As investigations continue, many other viruses will be reported.

Many insect-borne diseases involve 3 living factors Man--Virus--Arthropod Vector

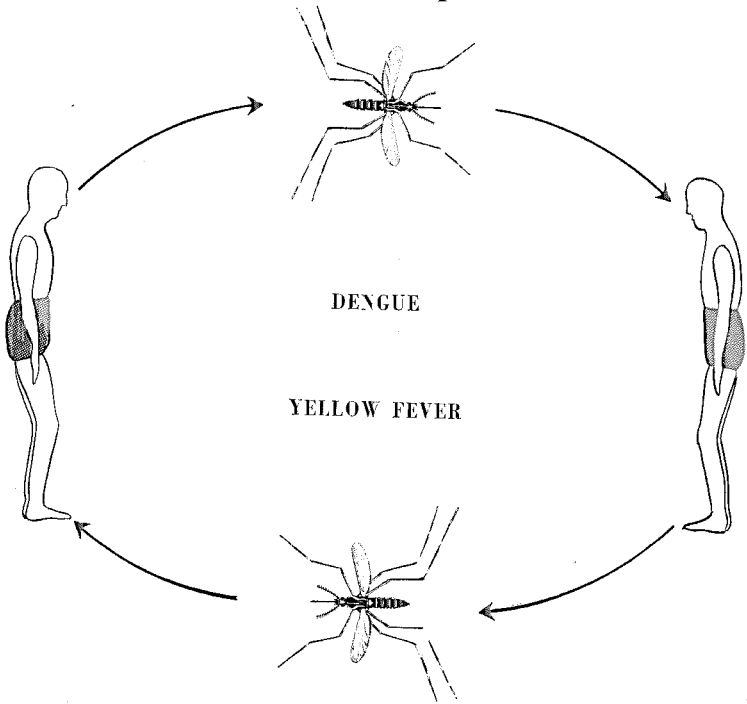


TABLE 2.—Arboviruses of North America not yet Associated with Human Illness.

Virus	Group	Vector	Location
Modoc	B	Unknown	California, Colorado
Trivittatus	California	Mosquito	North Dakota
Cache Valley	Bunyamwera	Mosquito	S. E. United States
Tensaw	Bunyamwera	Mosquito	Utah, Indiana
Turlock	Turlock-Umbre	Mosquito	California, Texas
Flanders (Hart Park-like)	Ungrouped	Mosquito	United States
Blue tongue	Ungrouped	<i>Culicoides</i>	W. United States
Hughes virus	Ungrouped	Ticks	United States
Silverwater	Ungrouped	Ticks	Canada
Epizootic Hemorrhagic Fever of Deer (EHD)	Ungrouped	Unknown	United States

The diseases caused by the arthropod-borne viruses (arboviruses) can be divided into two main epidemiological types. The first type involves only three living factors: man, the virus, and the arthropod-vector (Fig. 1). Urban yellow fever and dengue

are classic examples of this first type in which man is the vertebrate host, a virus is the parasite or causative agent of the disease, and the yellow fever mosquito (*Aedes aegypti*) is the insect carrier or vector. Great progress has been made in

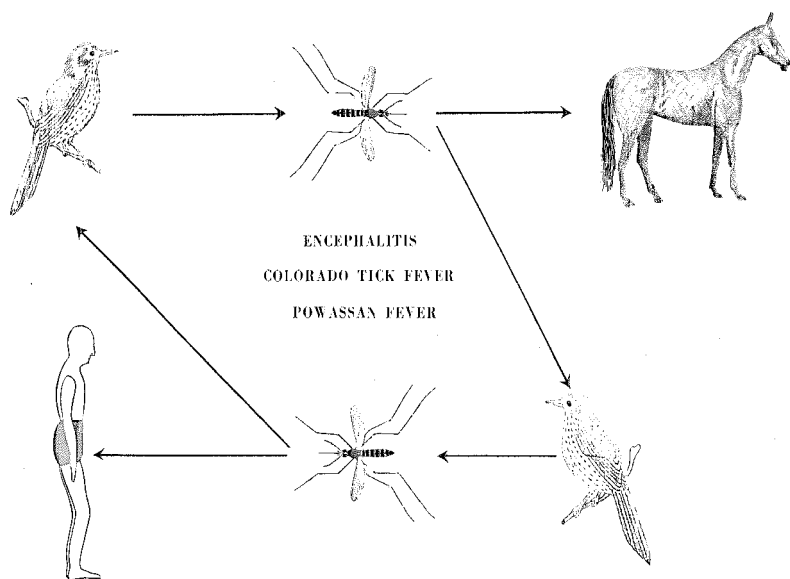
campaigns against these diseases through a combination of public health measures including mosquito control, international quarantine, vaccination, and recently, a program of *Ae. aegypti* eradication in the Western Hemisphere.

The second epidemiological type involves four living factors: man, the virus, the arthropod vector, and a reservoir of infection in wild vertebrate animals. The

against Eastern and Western encephalitis have been produced and used to protect horses, but have been used only with limited groups of human beings. At the present time vector control offers the greatest promise in preventing or controlling epidemics, but it needs careful evaluation with regard to surveillance techniques and the actual size and type of operations.

Many arthropod-borne diseases involve 4 living factors.

Man--Virus--Vector--Reservoir

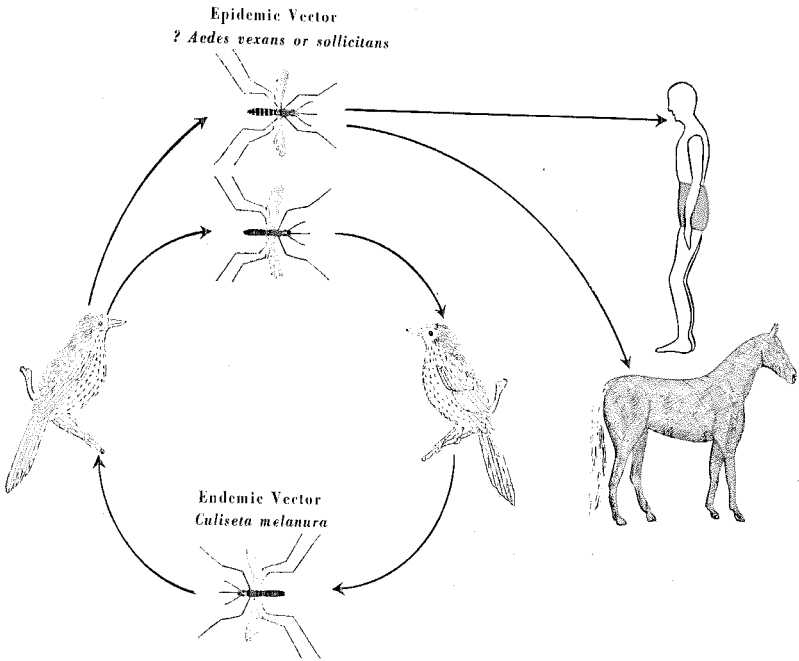


diseases of this second group are often termed zoonoses, that is, diseases of animals transmissible to man. They include jungle forms of yellow fever and dengue, the mosquito-borne encephalitides, and certain tick-borne diseases such as Colorado tick fever and Powassan virus fever.

With the second epidemiological type, the outlook for control is less hopeful because of the vast reservoir of infection in vertebrates and the tremendous number of infected vectors, primarily mosquitoes and ticks. In the United States, vaccines

YELLOW FEVER. Yellow fever probably was brought to the New World in the days of the slave trade and became firmly established in major port cities in the seventeenth and eighteenth centuries. Rigorous campaigns concentrating on control of the yellow fever mosquito (*Ae. aegypti*) wiped out the urban form of yellow fever from major foci in Havana in 1900-1901, in New Orleans in 1905, in Guayaquil, Ecuador in 1918-1919. By 1928 many authorities felt that urban yellow fever was almost eradicated from the Americas. Then a wave of yellow fever

EASTERN ENCEPHALITIS



swept to the suburbs of Rio de Janeiro in Brazil, and a huge urban epidemic occurred as the result of transmission to the susceptible human population by *Ae. aegypti* mosquitoes in the city. More important, some 4700 miles of coast line were reinfected with yellow fever—from Buenos Aires, Argentina, to Manaus, Brazil 2000 miles up the Amazon River (Yellow Fever Conference, 1955; Soper, 1963).

At this time Dr. Fred Soper and the Brazilian authorities, extending Gorgas' 1909 concept of controlling yellow fever by eradicating *Ae. aegypti* from urban areas, began work toward eradicating urban yellow fever and dengue from the Americas by the complete elimination of the yellow fever mosquito from the New World. By 1954 most of South America was free of *Ae. aegypti* mosquitoes (Yellow Fever Conference, 1955), and additional progress has been made since. As of

December 1963, the Pan American Health Organization (PAHO) considered Mexico, the seven countries of Central America, Brazil, Argentina, and six other countries of South America as free of *Ae. aegypti*. Infestations remain in small areas of Venezuela, and Colombia, and in the Guianas, southeastern United States, and the Antilles. (see Fig. 3).

Following the 1928 resurgence of yellow fever in South America, public health workers became increasingly aware of two epidemiological types of this disease, urban and jungle yellow fever, both caused by the same virus. In urban yellow fever the virus is transmitted from man to man by *Ae. aegypti*. In jungle yellow fever the same virus is transmitted by *Haemagogus* or other wild mosquitoes in a cycle ordinarily limited to monkeys and other jungle-canopy animals but occasionally including man. If a man becomes infected in the jungle and returns to cities where

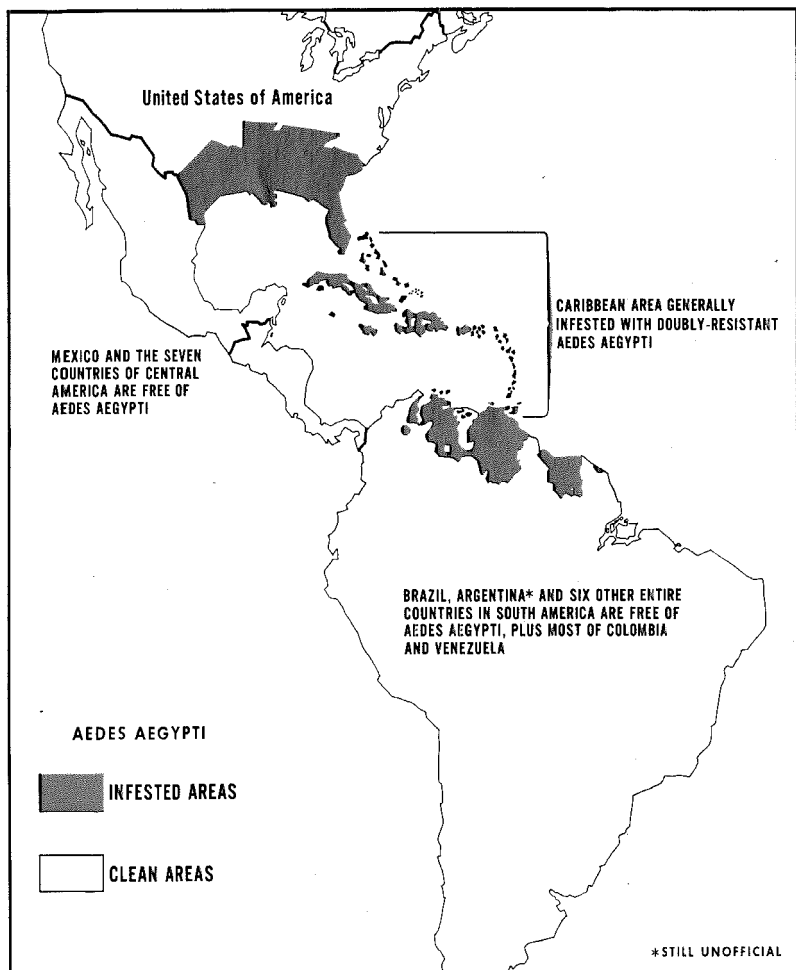
Ae. aegypti is present, he can become the source of urban epidemics.

An episode of jungle yellow fever in Central America, which began in the late 40's and lasted more than a decade, made the United States increasingly aware that so long as *Ae. aegypti* remains in this country the threat of yellow fever also remains. Beginning in Panama in 1948, a wave of jungle yellow fever started spread-

ing in the direction of this country. It swept across Panama that year, reached Costa Rica in 1951, Nicaragua in 1952, Honduras in 1953, Guatemala in 1955, and the Guatemala-Mexico border in 1957. About this time two outbreaks of yellow fever occurred on the island of Trinidad, British West Indies, also, in 1954 and in 1959.

Because of this dual threat to the United

GENERAL STATUS OF THE AEDES AEGYPTI ERADICATION CAMPAIGN IN THE AMERICAS AS OF DECEMBER 1963



States, one via Central America and the other via the Antilles, the Public Health Service took action for preparedness. In cooperation with the State health departments concerned, surveys were made in the Southeast to clearly define the boundaries of the area still infested with *Ae. aegypti* and to assess the present degree of infestation (Tinker and Hayes, 1959). Between 1957 and 1961, the CDC conducted a pilot eradication project at Pensacola, Fla., to study eradication methods used elsewhere and to determine the most practical methods applicable to conditions in this country.

As countries in Central and South America completed their *Ae. aegypti* eradication programs, some of them expressed fears that their territories would be reinfested with yellow fever mosquitoes from the United States, Puerto Rico, or the Virgin Islands. In order that this country can meet its responsibilities to the community of American nations, in October 1963, Congress appropriated three million dollars to begin an *Ae. aegypti* eradication program in this country. A new *Aedes aegypti* Eradication Branch has been organized at the Communicable Disease Center, Public Health Service, DHEW, with headquarters in Atlanta, Ga. D. J. Schliessman is the Chief of this new Branch with the following members of the American Mosquito Control Association on his staff: Russell Fontaine, G. R. Hayes, John W. Kilpatrick, Harvey B. Morlan, Louis J. Ogden, Harry D. Pratt, and Milton S. Tinker. In the first half of 1964 this eradication campaign will be started in Puerto Rico, the Virgin Islands, southern Florida, and southern Texas. The program will be based primarily on improved environmental sanitation to eliminate breeding places of the yellow fever mosquito, and selective residual spraying of actual or potential breeding places.

DENGUE. Dengue is an acute but rarely fatal disease caused by a number of strains of arboviruses in Group B of Casals and Clarke of the Rockefeller Foundation. The transmission cycle of this disease is

similar to that of urban yellow fever, that is, from person to person, generally by *Ae. aegypti*. In a typical cycle the dengue virus is injected into man and for the first few days produces only vague symptoms. Then the virus develops rapidly in the human host, courses through his peripheral blood, and causes his temperature to rise. From the day before fever develops until the third or fourth day of the disease, mosquitoes that feed on the patient's blood can obtain the virus and subsequently transmit it. At about the end of the first week, the patient's temperature begins to drop and the body develops antibodies to the dengue virus. Mosquitoes that feed on his blood thereafter do not become infected. After a period of convalescence, the patient recovers and develops short- or long-term resistance, or immunity, associated with the continued presence of protective antibodies or with the body's ability to develop such antibodies if it receives new injections of the virus.

Five outbreaks of dengue in the past 42 years have been of concern to the Public Health Service. They are:

- 1922-1923 Florida to Texas—perhaps 2,000,000 cases
- 1934 Florida and Georgia, estimated 15,000 cases
- 1943 Hawaii—estimated 1,400 cases
- 1945 Louisiana—several hundred cases
- 1963-1964 Caribbean—about 30,000 cases

The present epidemic in the Caribbean may have originated in Santo Domingo, Haiti, or Cuba and then spread to Jamaica and Puerto Rico. Between August 1963 and February 1964 over fifteen hundred cases of dengue were reported from Jamaica and approximately 27 thousand from Puerto Rico (Morbidity and Mortality Weekly Reports, 1963-1964). The epidemic may be still expanding because in December 1963 and early January 1964, 300 cases were reported from the island of Antigua, B.W.I. which is some 300 miles east of Puerto Rico (Morbidity and Mortality Weekly Reports, 1964). During this time 18 apparently healthy people had returned from the Caribbean to the United

States and then came down with dengue (Morbidity and Mortality Weekly Reports, 1963). There have been no secondary cases of dengue reported as yet in the United States in 1963 or 1964.

In order to prevent epidemics of dengue from developing in the United States, it is important that people entering continental United States from infected areas, such as Puerto Rico and Jamaica, be aware of the symptoms of dengue. In the event that they come down with the disease, then they should be zealously protected from exposure to *Ae. aegypti*, at least during the febrile period or the first week of the disease. This is particularly important in southern United States.

Two factors serve to limit the possibility of dengue epidemics in the United States: the distribution of the most likely vector, *Ae. aegypti*, and the fact that the virus would not develop in the mosquito host during cold weather. Certain travelers came down with dengue during the month of September 1963 in Connecticut, New York, and Minnesota after visiting the Caribbean. There should be no epidemic resulting from these cases since there are no wild *Ae. aegypti* north of the Mason-Dixon line and since the cases occurred in September or later when it is too cold for the dengue virus to develop in alternate species of mosquitoes.

On the other hand, there is a great need for rigid *Ae. aegypti* control and ultimate eradication in Florida and other southern States to prevent contact of yellow fever mosquitoes with dengue patients.

ENCEPHALITIS. A vast amount of research has been published concerning the arthropod-borne encephalitides. About 160 "arboviruses" are known at the present time. A list of 140 of these, which were being studied in 1961 and 1962 at the Rockefeller Foundation Virus Laboratories in New York, was published by Mettler, Casals, and Shope (1963).

Three of these arboviruses are of great importance in the United States and Canada: Eastern, Western, and St. Louis encephalitis. Eastern encephalitis occurs

along the Atlantic and Gulf Coasts and in localized areas inland. Western encephalitis occurs in epidemic form in human beings and horses west of the Mississippi and in Wisconsin and Illinois, and in birds, rodents, and mosquitoes in the eastern part of the United States. St. Louis encephalitis occurs west of the Mississippi, in the Ohio Valley, and in Florida. The absence of Western and St. Louis encephalitis in human beings in northeastern United States may be correlated with the scarcity or absence of *Culex tarsalis*, the good vector of these diseases in western United States and Canada. Surveys by Hayes (1962) and other workers indicate that western encephalitis virus occurs in mosquitoes such as *Culiseta melanura* and in rodents in Massachusetts and New Jersey.

Eastern and Western encephalitis affect both man and equines, while St. Louis encephalitis causes clinical symptoms in man but normally not in horses. This information is often used by epidemiologists studying outbreaks of diseases affecting the central nervous system in central and western United States. If an outbreak of a disease is reported affecting the central nervous system of both human beings and equines, Western encephalitis is often suspected; but, if a similar outbreak affects only human beings, public health workers frequently think of St. Louis encephalitis or poliomyelitis.

The data available suggest that Eastern is the most virulent of these diseases. Human mortality rates range from 60 to 74 percent in Eastern encephalitis, from 10 to 24 percent in St. Louis encephalitis, and from 2 to 10 percent in Western encephalitis. Antibody surveys indicate subclinical inapparent infections occur with all three types.

These encephalitides show differences as to the age groups they attack. Eastern and Western encephalitis seem to affect the younger and the older age groups, while St. Louis encephalitis is found most frequently in the older age groups, particularly those over 50. For example, Feemster (1938) reported that 26 of 34 cases (76

percent) of Eastern encephalitis in the 1938 Massachusetts outbreak occurred in the youngest age group, under 10 years old. In summarizing a 10-year study in Kern County, California, Hammon, Reeves, and Longshore (1962) reported that Western encephalitis "showed a marked tendency to produce clinical disease in younger people, almost one-fourth of patients being infants less than 1 year old and more than one-half being under 10 years. During the entire study only one case of St. Louis encephalitis occurred in an infant, and less than one-third of those infected were under 10 years of age. About half were adults 20 years of age or older." In the 1959, 1961, and 1962 epidemics of St. Louis encephalitis in Florida the disease also affected the older age group. Waters, *et al.* (1963) reported that in the 1961 epidemic the average age was 56 years in 25 cases studied.

The vectors of the three main types of encephalitis differ considerably (Hess and Holden, 1958). *C. tarsalis* is probably the most important vector of Western encephalitis virus to birds, horses, and man. This same species is considered the important vector of St. Louis encephalitis virus in western United States. However, in the Kansas City-St. Louis-Ohio Valley area, members of the *Culex pipiens* complex are probably involved in epidemics of St. Louis encephalitis; in the Rio Grande valley, *Culex quinquefasciatus* may be of importance; and there is good evidence that *Culex nigripalpus* is the vector of St. Louis encephalitis in the Tampa Bay area of Florida. Authorities differ, often markedly, as to the probable vector of Eastern encephalitis (Hess and Holden, 1958). Some believe that *Culiseta melanura* is the endemic vector of Eastern encephalitis virus from bird to bird, and might even be responsible for the occasional infections which have occurred in man. Others feel that the majority of human and horse cases have occurred in areas teeming with *Aedes vexans* and *Aedes sollicitans*. They point out that almost all the human cases occur close to the Atlantic or Gulf Coasts, within

50 or 60 miles of the seashore, which is within the 50- to 60-mile or more flight range of the salt marsh mosquito (*Ae. sollicitans*). There is the reasonable possibility that *C. melanura* is an endemic or enzootic vector of Eastern encephalitis from bird to bird, and that *Ae. sollicitans*, *Ae. vexans*, or other vicious *Aedes* species may be epidemic vectors of Eastern encephalitis virus from wild birds to man and horse (Fig. 4).

Considerable evidence now indicates that these three encephalitides are normally virus infections of birds, and that human and horse cases are accidental dead-end cases not in the main cycle of transmission. For this reason many public health workers prefer to use the shorter, simpler terms "Eastern," "Western," and "St. Louis encephalitis," rather than "Eastern equine or Western equine encephalitis or encephalomyelitis." Research of many investigators, including Stamm (1963) and Stamm, Chamberlain, and Sudia (1962) of the CDC laboratories, indicate that small birds are a more important source of virus than the larger birds.

The methods of dissemination and survival of the three major encephalitis viruses are being studied intensively by many research groups. Much time and money has been spent studying bird mites, ticks, and *Triatoma* bugs as vectors and reservoirs of these infections but with little success. At the present time, two hypotheses have been advanced to explain the overwintering mechanism of these viruses: (1) a long-lived vector which maintains the virus from fall to the following spring; and (2) a vertebrate host with long-term viremia or a relapsing, chronic, latent infection (Reeves, 1962). While mosquitoes may serve as long-lived reservoirs and vectors of these arboviruses in southern United States from Florida to California, there is much evidence suggesting that in northern United States and Canada these insects feed on flower nectar in late summer or fall and do not take a blood meal before hibernating (Bennington *et al.*, 1958). In this huge area, therefore, they may not

play a significant role in carrying the viruses through the winter to the following spring. For this reason, the second hypothesis, that a vertebrate host may play a significant role in overwintering, is receiving increasing attention. A great deal of research is currently being conducted with regard to (1) the importance of other vertebrates such as snakes, rodents, and birds as reservoirs of the various types of encephalitis viruses, and (2) the possibility that blackflies, sand flies (*Phlebotomus*), fleas, or other ectoparasites may serve as vectors from rodents with viremia during wintertime. Since many of the mosquitoes in which the encephalitis viruses have been isolated (such as *C. tarsalis*, *C. melanura*, and *C. pipiens*) feed readily on birds, much attention is being given to both migrating and resident birds as overwintering sources of the arboviruses.

Another field in which intensive studies have been carried out recently deals with the relationship of weather to actual epidemics of the encephalitides. Studies of Hess *et al.* (1963) have revealed that most of the outbreaks of Western encephalitis have occurred at or north of the 70° F. June isotherm, whereas epidemics of St. Louis encephalitis generally have occurred at or south of this isotherm.

The importance of mosquito control activities during an actual epidemic of encephalitis needs additional and careful evaluation. Members of the American Mosquito Control Association in this audience have conducted control operations during encephalitis outbreaks—in California in 1952, in the Rio Grande valley of Texas in 1954, at Calvert City, Ky., in 1955, in Louisville, Ky., in 1956, in Massachusetts in 1938, 1955 and 1956, in Utah in 1958, in New Jersey in 1959, and in Florida in 1961 and 1962. Recently, studies have been made in Texas and California in an effort to determine the size of the area in which it is necessary to control infected mosquitoes in order to prevent transmission of the encephalitis viruses in populated areas. These studies indicate that it is necessary to have a high level of mos-

quito control for many miles outside the populated area in order to reduce the number of infected mosquitoes flying or drifting into the control zone and significantly lower the level of transmission. (Reeves, 1964, unpublished data; Bailey, 1964, unpublished data; Hess, 1964, unpublished data.) In a number of the epidemics listed above, the true importance of control operations will never be known. In some of them it is probable that control operations began after the peak of the outbreak and that, as has happened with epidemiologists the world over, the control workers "rode to glory down the descending curve of the epidemic."

However, mosquito control may actually have played a significant role in limiting the outbreak of encephalitis in the Tampa Bay area in Florida in 1962. Outbreaks of St. Louis encephalitis occurred in this same area during the months of September, October, November, and December in 1959 and 1961. In 1962 another large epidemic began in the Tampa Bay area in August. It is possible that intensive mosquito control work in this area, including fogging to kill the infected mosquitoes, may have played a significant role in stopping this outbreak in September, several months earlier than the previous epidemics had ended (Fernald, 1963).

Epidemiologists have noted that in the United States as a whole, there is a tendency for major encephalitis outbreaks to occur about every two or three years. There were major outbreaks of some type of encephalitis in this country in 1952, 1954, 1956, 1959, and 1962. In 1963 two epidemics of Western encephalitis and one epizootic of Eastern encephalitis were noteworthy. In Hale County, Texas, about 87 reported cases have been studied. Out of 61 paired sera, approximately 30 showed indication of infection with Western encephalitis (Sciple, 1964). In Canada unofficial reports indicated a large outbreak of Western encephalitis in the Prairie Provinces of Saskatchewan, Manitoba, and Alberta, with both human and equine cases. In Saskatchewan there were nearly 100

human cases, approximately one-third of which were confirmed as due to Western encephalitis (McLintock, 1964, unpublished data). Late in the summer of 1963 an epizootic of Eastern encephalitis was investigated in southern Georgia and Eastern encephalitis infection demonstrated in many horses and mosquitoes, but no human cases occurred (Chamberlain, 1964, unpublished data). Otherwise, 1963 can be considered a "low year" for encephalitis in North America. Members of this audience, therefore, will be interested in watching to see if 1964 will be a "peak year" for encephalitis—if somewhere in the United States there is a major epidemic of this dread disease.

COLORADO TICK FEVER. Colorado tick fever was the first tick-transmitted virus disease of man to be recognized in the Western Hemisphere (Eklund, Kohls, and Brennan, 1955). The disease in man is now known to occur in at least ten western states: California, Colorado, Idaho, Montana, Nevada, Oregon, South Dakota, Utah, Washington, and Wyoming. Well over 1500 cases are known, the greatest number from Colorado where a peak of 242 cases in 1960 was reported by the Colorado State Health Department. The Rocky Mountain wood tick (*Dermacentor andersoni*) is the only known vector, although isolations of the virus have been reported from *Dermacentor variabilis*, *D. parumapertus*, *D. occidentalis*, *D. albipictus*, and *Otobius lagophilus*. Studies by Burgdorfer and Eklund (1959, 1960) indicated that the distribution of the virus in parts of western Montana could be correlated with the presence or absence of the golden-mantled ground squirrel (*Citellus lateralis*). Other mammals from which the virus has been isolated include the Columbian ground squirrel (*Citellus columbianus*), a chipmunk (*Eutamias amoenus*), pine squirrel (*Tamiasciurus hudsonicus richardsoni*), a deer mouse (*Peromyscus maniculatus*), and the porcupine (*Erethizon dorsatum*).

Colorado tick fever virus has been isolated by Eklund, Kennedy, and Casey

(1961) from the blood of 552 patients who lived in the Rocky Mountain area or visited there prior to their illness. These observers reported that in these cases the onset of illness occurred between March 1 and October 14 and was most common during May and June. There were about three times as many cases in males (421) as in females (131), probably because boys and men engage in activities that bring them in contact with the ticks: ranching, lumbering, sheepherding, hiking, and fishing. Usually Colorado tick fever is considered a mild, dengue-like disease, but the studies of Eklund, Kennedy, and Casey (1961) indicate that there were nine reports of serious illness and one case was fatal. Four children had serious bleeding and hemorrhagic symptoms. Four children and one woman had severe symptoms of the central nervous system suggesting acute encephalitis.

The tick-borne virus diseases as a group differ epidemiologically from those transmitted by mosquitoes. In many tick-borne diseases, such as Rocky Mountain spotted fever, the pathogen can pass from infected adult ticks through the egg to the larvae, nymphs and adults, a phenomenon known as transovarial and transstadial infection of the vector, whereas in mosquitoes, the adult is typically the only stage infected with viruses causing human diseases. Florio *et al.* (1950) has reported that Colorado tick fever virus was passed from infected adult ticks through the egg to larvae, nymphs, and adults.

Eklund and his co-workers (1961), however, questioned the occurrence of transovarian passage of Colorado tick virus to the progenies of infected tick females. They felt, and laboratory observations indicated, that this phenomenon played no significant role in the maintenance of Colorado tick virus in nature. They postulated that the cycle of infection was limited between immature ticks and their small-animal hosts. The virus is transmitted to adult ticks, the stage that feeds on man and causes human infections. Experimental vaccines have been developed by

several workers including Thomas, Ek-lund, Philip, and Casey (1963).

POWASSAN VIRUS DISEASE. Powassan virus disease was first diagnosed in a fatal case of encephalitis in a child in Powassan, Canada. The virus has been found in *D. andersoni* ticks in Colorado, and antibodies have been found in human blood in people from the Mexican province of Sonora (McLean, *et al.*, 1960; Thomas *et al.*, 1960; Public Health Reports, 1961).

Since Powassan is just south of Lake Nipissing, the remnant of glacial Lake Nipissing that once covered much of the Great Lakes region, one can wonder how long the Powassan virus has been in Powassan. Much of this area was buried a mile or more under the huge continental glacier only five to ten thousand years ago—a part of the last glacier which extended south only 25,000 years ago approximately to the level of the Ohio and Missouri rivers (Flint, 1957).

Serological evidence indicates that Powassan virus is related to other tick-borne diseases such as Russian spring-summer encephalitis of Siberia, Kyasanur Forest disease of India, Omsk hemorrhagic fever of Russia, and louping ill of Scotland. One can speculate that many thousand years ago there was an original tick-borne virus occurring in small mammals from Great Britain across Siberia to North America when much of the present oceans was bound up in glacial ice. At that time the level of the ocean was as much as 300 feet lower than at present and there was a land bridge connecting Alaska and Siberia which allowed an interchange of the fauna and flora between the present two continents. As the glaciers melted and retreated northward, the oceans rose; and the original stem tick-borne virus became established over a vast area in different vertebrate hosts with different tick vectors in which it evolved into distinct serological strains through a process of selection and geographical isolation. As the ice sheet receded, the forest, the vertebrate hosts, and the tick vectors also migrated north-

ward and became established in their present areas.

An alternate hypothesis is suggested by the research of Hoogstraal and his co-workers (1961) in Africa who have found ticks on migrating birds from northern Europe and Asia. It is possible that migrating birds could have carried ticks with Powassan-like virus to many parts of the North American continent.

Many of you in the American Mosquito Control Association have worked for years on the control of mosquito-borne diseases or pest mosquitoes. You have demonstrated tremendous ability to work with the public and have developed fine organizations for long-term mosquito control programs. In some areas, because of your acknowledged competency in leadership in mosquito control, you have been asked to expand into broader programs including fly control, sand fly or biting-fly control, even area control of ticks or chiggers. As you continue either in mosquito control programs or programs of broader arthropod control, it will be well to consider carefully these continuing needs involved in the control of the arbovirus diseases, so well discussed by Dr. A. W. Donaldson, Deputy Chief of our Communicable Disease Center in 1958. These needs are:

1. Determination of true public health importance of arboviruses;
2. Comprehensive understanding of the epidemiology of these diseases;
3. Improved diagnostic tools, procedures, and resources; and
4. Development of practical and effective methods of control.

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THE COLONIZATION OF TEMPERATE NORTH AMERICA BY MOSQUITOES AND MAN *

HERBERT H. ROSS

Illinois Natural History Survey, Urbana, Ill.

In attempting to understand the relationships between organisms occurring together in the same habitat, it is frequently helpful to know how the particular mixture of species came into existence. This is true of mosquitoes and man in the temperate part of North America. In this area (as in all other parts of the world permanently inhabited by man) man lives in many kinds of ecological communities and in each one of these communities man and a particular assemblage of mosquito species form an intimately related part of the ecosystem. One way of looking at this North American mosquito-man segment of the ecosystem is to try to determine when the various species reached this area, where they came from, and what has happened to them since. Information on these questions would explain one facet of the relationship between man and mosquitoes on this continent.

First let us consider the mosquitoes. There are slightly less than 150 species known from North America north of Mexico, out of the 2500 species known for the entire world. The questions we seek to answer about these 150 are: (1) where did they come from, (2) when did they reach this region? We can obtain reliable clues about these problems in only one way. First we need to know the family tree of all these mosquitoes; in other

words, determine as reliably as possible how the different genera and species evolved. This is done by comparing detailed characters of all of them, determining which characters probably are primitive and from this deducing the order in which different character combinations evolved. This comparison should include all living and all fossil species. With mosquitoes, the fossils are too poorly preserved to show the minute characters that we need to see for such a study. After the family trees are worked out to the best of our ability, we can then superimpose geographic distribution on these and, in some cases at least, arrive at plausible explanations concerning the dispersal of each group. Using these methods, the following account outlines the results obtained by treating the North American mosquitoes genus by genus.

THE GENUS *Deinocerites*

Two of our most unusual mosquitoes are *Deinocerites cancer* and *mathesoni* that normally breed in the holes of land crabs. The genus *Deinocerites* is entirely American, restricted for the most part to the American Mediterranean region (Fig. 1). It is evident that the evolution of this genus has occurred primarily in the tropics. In each of two main lineages, however, a species became adapted to live in subtropical and even slightly temperate conditions. One of these is the species

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