PUBLIC HEALTH MONOGRAPHS

edited and issued by Public Health Reports

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

OVETA CULP HOBBY, *Secretary*

PUBLIC HEALTH SERVICE

LEONARD A. SCHEELE, *Surgeon General*

Biological Factors In the Transmission Of American Arthropod-Borne Virus Encephalitides

A Summary

Frederick F. Ferguson, M.S. Ph.D

Public Health Monograph No. 23

The Author

Dr. Ferguson, a public health parasitologist at the Public Health Service's Communicable Disease Center, Atlanta, Ga., has been chairman of the Mosquito Control and Allied Problems Work Group of the Arkansas-White-Red River Basins Inter-Agency Committee. He has done research in mosquito insecticides, in the toxicology of insecticides, and in the morphology of platyhelminths. He has been professor of biology and parasitology at the College of William and Mary (Norfolk Division) and at the University of Washington. In 1951, he served as secretary-treasurer of the American Association of Professional Biologists and is presently engaged in schistosomiasis research at the Puerto Rico Field Station.

Public Health Service Publication No. 372

(Issued concurrently with the November 1954 issue of Public Health Reports, Vol. 69, No. 11)

(Received for publication March 1954)

Library of Congress Catalog Card No. 54–60655

UNITED STATES GOVERNMENT PRINTING OFFICE, WASHINGTON : 1954

For sale by the Superintendent of Documents, U. S. Government Printing Office
Washington 25, D. C. — Price 30 cents

Contents

not acc.

Acknowledgments

Appreciation is extended to L. D. Beadle for many helpful suggestions as to sources of data. Numerous interlibrary loans were made available through the work of Effie B. Lunsford, Communicable Disease Center Library, Atlanta, Ga. Sincere gratitude is expressed to the following persons who offered constructive criticism in the development of this summary: E. H. Arnold, Bernard Brookman, A. L. Burroughs, W. J. Clark, R. W. Chamberlain, D. E. Eyles, S. Gladness, W. McD. Hammon, A. D. Hess, D. W. Jenkins, M. A. Manzelli, W. J. Hayes, Jr., G. F. Mathews, W. C. Reeves, and G. H. Bradley.

Introduction

The aim of this paper is to present a selected bibliography with annotations, for the use and information of investigators and to furnish pertinent tabular summaries for those who are actively engaged in studies on the epidemic encephalitides in the Americas. The general goal of the workers in this field has been clarification of the problem of how the etiological agents are maintained and transmitted in nature, with the hope that practical means of dealing with these diseases may be found. This review stresses the entomological phases of encephalitis transmission.

The text of the review reflects principally the winnowing of data for inclusion in a tabular summary. Subject literature has been covered through April 1952. Some indication of the content of each bibliographic item (but not relative values) has been given. However, in a work of this magnitude, there will remain errors involving judgment, interpretation, and other variables, as well as transcription difficulties. Change of concepts in this rapidly developing field must be considered, for in many cases the ideas expressed in early works are no longer tenable and, in some instances, unpublished data may change the emphasis on, or even refute, a concept previously delineated. In this regard, the author has adhered to published materials, with but little exception.

The published data on arthropod-borne neurotropic viruses of Central and South America are limited and are briefly summarized for comparative purposes. The following types of encephalitis or encephalitis-producing viruses occur there: Argentine equine encephalomyelitis (*176*, *253*) in which the virus is identical (*329*) to that of western equine encephalomyelitis (WEE [1]); eastern equine encephalomyelitis (EEE), now distributed in Panama, Brazil, Mexico, Cuba, and the Dominican Republic; Ilheus encephalitis (*218*) from Brazil, a mosquito-borne human infection; *Anopheles* A, *Anopheles* B, and *Wyeomyia* encephalitis (*328*) from Colombia, known as mosquito-borne viruses capable of producing these diseases in laboratory animals; *Anopheles* I, Sabenthine I, and an unnamed virus from *Psorophora ferox* infective for laboratory animals, also from Colombian wilds (*64*); and Venezuelan equine encephalomyelitis (VEE) (*21*, *42*, *116*, *215*, *216*, *308*), a disease of man and equines, now established in Trinidad, Venezuela, Colombia, and Ecuador. VEE is marked by explosive outbreaks among equines and appears to be transmitted by mosquitoes in nature and possibly by dust under laboratory conditions. For published details on VEE, refer to the bibliography maintained at the Camp Detrick Technical Library, Frederick, Md. (*2*). There is a paucity of data on the vector problem related to these Central and South American virus diseases (*308*). The arthropod-carried viruses reported as being capable of causing encephalitis found in the Western Hemisphere are these: EEE, WEE, SLE, Hammon-Reeves or California,[2] Ilheus, *Anopheles* A, *Anopheles* B, *Wyeomyia*, *Anopheles* I, Sabenthine I, the unnamed virus from *P. ferox*, and VEE.[3]

There have been but few attempts to classify the confusing series of diseases manifested as encephalitis (*28*, *32*, *34*, *101*, *131*, *412*). Ayres and Feemster (*32*) in 1949 divided these diseases

[1] Symbols used in the literature: WEE (western equine encephalomyelitis); EEE (eastern equine encephalomyelitis); SLE (St. Louis encephalitis); VEE (Venezuelan equine encephalomyelitis); type A (von Economo's disease or lethargic encephalitis); type B (Japanese encephalitis); type C (St. Louis encephalitis).

[2] The California virus strain (BFS–91) was isolated from *Aedes dorsalis* from Kern County, 1943 (*147*).

[3] References on certain foreign neurotropic viruses are presented for comparative purposes: VEE (*21*, *42*, *116*, *215*, *216*, *308*); *Japanese B encephalitis* (*65*, *192*, *307*); Russian tick-borne encephalitis (*75*); Australian X disease (*76*); Australian encephalitis (*16*); Ilheus virus (*218*); West Nile virus (*292*, *375*); *Anopheles* A and B, and *Wyeomyia* viruses (*328*), Argentine equine encephalomyelitis (*253*); and louping ill (*412*).

into three groups: "Virus encephalitides caused by familiar viruses not ordinarily encephalitogenic; suspected, primary virus infections; and primary virus infections."

In the first group, the following are usually listed: herpes simplex, measles, lymphogranuloma venereum, mumps, and infectious mononucleosis. Postinfective encephalitis as an aftermath of infectious hepatitis, smallpox, vaccinia, chickenpox, influenza, mumps, and measles also belong to this group, along with postvaccinal encephalitis due to vaccination against rabies and smallpox.

In the second group—suspected primary virus infections—belong types in which antibodies have been found in the blood of man and the viruses are shown to be neurotropic in laboratory induced disease. In this category are the following virus diseases: West Nile (*375*), Bwamba fever (*376*), Semliki Forest (*373*), Bunyamwera (*377*), Ilheus (*218*) and California (*147*, *149*) in which the syndrome in man has not been adequately described. Von Economo's disease (encephalitis lethargica) and acute disseminated encephalomyelitis also are suspected of being caused by primary neurotropic viruses. Due to the gradual disappearance of von Economo's disease in the United States between 1920 and 1930, it is not at present considered important epidemiologically.

In the third group belong the primary virus encephalitides, which may be divided into the summer, or epidemic, forms: St. Louis encephalitis; eastern, western, and Venezuelan equine encephalomyelitis (*216*); Japanese B encephalitis (*412*); Russian Far East (tick-borne, or spring-summer) encephalitis (*75*); Australian X disease (*76*, *287*); and into the endemic viruses: acute hemorrhagic meningoencephalomyelitis, or leukoencephalitis; lymphocytic choriomeningitis; pseudolymphocytic choriomeningitis; swineherd's disease (*91*); louping ill (*412*); acute encephalitis due to rabies; and Sabin B virus. The chief medical problem in North America appears to lie with the epidemic encephalitides (EEE, WEE, and SLE), in which the mortality ranges from 5 to 60 percent.

The references listed constitute a bibliography in the sense that, in the author's opinion, all important papers relating entomology to encephalitis published prior to 1952 have been included. The literature is vast and widely scattered in many fields of science. The *Quarterly Cumulative Index Medicus* (American Medical Association) lists thousands of references, garnered from international sources before 1930, which pertain to the purely human clinical manifestations of alleged encephalitis. Since 1930, references to the particular diseases WEE, EEE, and SLE may be found generally concentrated in the sources discussed.

Information is scattered throughout the standard abstracting journals and reference indexes (*Biological Abstracts*, *Review of Applied Entomology—Series B*, and *Bibliography of Agriculture*, *Quarterly Cumulative Index Medicus*, and *Index of American Economic Entomology* by Banks, Colcord, and Hawes). The standard texts and reference books, such as those of Rivers (*327*), Van Rooyen and Rhodes (*412*), Bates (*36*), Smith (*363*), and Neal (*280*), are cited.

The various State agricultural bulletins contain data generally designated for lay consumption and pertain mostly to the care of horses suffering with these diseases. The annual reports of the U. S. Bureau of Animal Industry constitute a source from which data on the prevalence of WEE and EEE in the United States may be obtained. Veterinary journals (*American Journal of Veterinary Research*, *North American Veterinarian*, and *Journal of American Veterinary Medical Association*) inform the veterinary profession of new developments in the field and contain many important reviews.

With respect to encephalitis in man, some reviews are to be found in the *New England Journal of Medicine*. Technical studies on clinical cases, epidemiological summaries, and editorials designed to clarify a confused picture of disease for the practicing physician are to be found in the *Journal of the American Medical Association*. Various State medical journals contain clinical case reports and invitation-type papers which are written for uninformed practicing members of the profession. Technical studies on the viruses are to be found in the *Journal of Immunology*, *Archives of Pathology*, and similar publications. Experiments involving laboratory transmission of these diseases have been described most often in the *Journal of Experimental Medicine*, *Proceedings of the Society of Experimental Biology and*

Medicine, *Journal of Infectious Diseases*, and *American Journal of Tropical Medicine*. In general, epidemiological studies have been published in the *Canadian Journal of Comparative Medicine*, *Canadian Journal of Public Health*, *Public Health Reports*, *Public Health Service Bulletins*, *American Journal of Hygiene*, *American Journal of Public Health*, and *Proceedings of the California Mosquito Control Association*. Numerous preliminary reports are to be found in *Science*. The Matheson Commission of New York has accumulated and distributed data on the epidemiology of human encephalitis, and the Hooper Foundation of California has made extensive contributions in the general field of epidemiology of the diseases (principally WEE and SLE) in the United States.

Useful general reviews are the following: Ayres and Feemster, 1949 (*32*); Casey, 1942 (*69*); Cockburn et al., 1951 (*79*); Davis, 1940 (*87*); Dingle, 1941 (*89*); Feemster and Getting, 1941 (*99*); Fothergill, 1940 (*101*); Geiger et al., 1922 (*108*); Giltner and Shahan, 1942 and 1936 (*115*, *114*); Hammon et al., 1948, 1948, 1945, 1945, 1945, 1942 (*141*, *142*, *160*, *138*, *147*, *152*, respectively); Manzelli, 1948 (*235*); Matheson Commission, 1939 (*426*); Meyer, 1933 and 1933 (*249*, *250*); Milzer, 1950 (*259*); Neal, 1942, 1934, 1928 (*280*, *279*, *277*, respectively); Price, 1950 (*296*); Reeves, 1941 (*305*); Schoening, 1940 (*337*); Shahan and Giltner, 1945 (*348*); Sulkin, 1949 (*382*); Udall, 1913 (*409*); Webster, 1941 (*417*); Wright, 1927 (*429*), and Zinsser, 1928 (*432*). Certain papers are given minor consideration in this review because either they do not contain results of original tests or the information is of a generalized nature (*7*, *10*, *14*, *17*, *19*, *43–45*, *54*, *55*, *60*, *63*, *67*, *96*, *130*, *134*, *140*, *143*, *201*, *230*, *237*, *244*, *274*, *295–297*, *303*, *306*, *309*, *325*, *326*, *334*, *339*, *347*, *378*, *387*, *400*, *404*, *427*). In the literature, the term "encephalitis" is used somewhat loosely to cover both encephalitis involving virus infection in the brain proper and encephalomyelitis which is centered in the brain and spinal cord. Encephalitis is popularly known as sleeping sickness or brain fever.

Historical Notations

Four types of encephalitis (literally, brain inflammation) have appeared in epidemic form in the United States within the last 35 years:[4] von Economo's disease (encephalitis lethargica or Japanese A encephalitis), eastern and western equine encephalomyelitis, and St. Louis encephalitis.[5] All of these forms affect the central nervous system.

Encephalitis Lethargica

Although von Economo's encephalitis is an ancient disease (*232*, *362*, *414*), in modern times it has been best known from about 1915, when it was studied in France by Cruchet et al. (*85*). It is clinically manifested by somnolence, occulomotor palsy, pyrexia, and signs of cerebromeningeal disturbance. This disease of unknown etiology was established in epidemic form in New York, N. Y., and in Winnipeg, Canada, in 1919–20, but by 1926 it had begun to subside in North America and the world over. Since then, the disease has become relatively rare (*173*, *276–280*), and possibly the cases seen now are of different etiology. Arthropods have not been suspected of transmitting this essentially winter-season disease.

St. Louis Encephalitis

Briefly, the history of SLE is as follows: The first cases of the disease to become known as St. Louis encephalitis occurred in Paris, Ill., in 1932, although there is evidence that it has long been endemic in the area (*79*). Cases

[4] See table 7, p. 17.

[5] In addition to SLE, WEE, and EEE viruses, there is the California virus named by Hammon and Reeves which has not been studied in man (*147*, *149*).

occurred in St. Louis County, Mo., and in surrounding areas in a wide radius during the summer and fall of 1933 in epidemic fashion (*79*, *171*, *294*). A minor outbreak occurred in Toledo, Ohio, in 1934, and neutralizing antibodies were later recorded for the residents of New York and Pennsylvania. The disease subsided with no true cases being reported for 1935–36. However, it reappeared in epidemic form in the summer of 1937 in St. Louis County, Mo. (*61*, *69–70*, *121*). Several hundred cases developed, and the disease has since become established endemically in the United States, having occurred notably in California and Washington. Ten cases in 1938 failed to show SLE antibodies (*366*), but about one-third of 66 cases during 1939–44 gave serologic evidence of SLE antibodies (*50*). Results from a serum survey in 1943–44 (*371*) indicate that there were fewer persons with SLE antibodies who came to California and Washington after 1937 as compared with persons with SLE antibodies resident there during 1933–37. Some of the details of the continental distribution of SLE in man may be found in the following papers: Arizona, (*238*); California (*159*, *179*, *184*); Colorado (*22*, *293*); Minnesota, 1941 (*92*); Missouri, 1933 (*272*); Nebraska, 1941 (*431*); New York (*57*, *419*); North Dakota (*56*); Oklahoma (*315*); Utah (*26*); and Washington (*129*, *144*). There is evidence that SLE occurs as a human disease in Africa (*372*, *374*, *375*). Some of the reports of virus isolations from man are cited (*51*, *61*, *225*, *238*, *273*).

St. Louis encephalitis is not generally thought of as being "equine" in nature, although it does occur in horses (*1*, *11*, *84*, *144*, *153*, *293*) as a subclinical infection, and under laboratory conditions it may induce equine deaths (*351*). The susceptibility of mules to SLE virus infection also has been confirmed (*18*, *84*).

Western Equine Encephalomyelitis

Zwick and Seifried (*433*), working on European equine encephalomyelitis in 1924, determined that the etiological agent of that disease was a filtrable virus. In 1932, Meyer (*248*) recorded the possibility of human infection following infection of horses with WEE virus and subsequently described the first cases in man. Howitt (*178*) isolated WEE virus from the brain of man in 1938 and from human serum in 1939 (*180*). Numerous human cases have been reported from California. The disease has been found in Minnesota, 1938; Arizona, 1941; North Dakota, 1938 and 1941; South Dakota, 1941; Nebraska, 1941; Montana, 1947; Texas, 1944; Manitoba, Alberta, and Saskatchewan, Canada, 1941; and Manitoba, Canada, 1947. The virus of Argentine equine encephalomyelitis appears to be identical with that of WEE (*275*, *353*). A few isolations of WEE virus have been made from human materials (*67*, *82*, *128*, *178*, *223*, *239*).

Eastern Equine Encephalomyelitis

Human infection with EEE virus first was recognized as a nosologic entity with the isolation of the agent by Fothergill et al. (*103*) and Webster and Wright (*422*) in Massachusetts in 1938. This development followed the original isolation of EEE virus from infected horses by Giltner and Shahan in 1933 (*112*). In man the disease followed a fulminating course resulting in fatality of 74 percent of the 34 persons affected in the first outbreak (*33*, *425*) and produced somewhat similar results in a later Louisiana outbreak (*257*, *282*). There has been only scattered evidence of human infection since 1938: Indiana, 1939; Louisiana, 1947 (*169*, *257*); Tennessee, 1947; and Texas, 1941–42 (*52*, *117*, *136*). A few virus isolations from man have been reported (*103*, *257*, *282*, *338*). The history and present public health significance of EEE has been summarized recently by Beadle[6] who noted that EEE virus had been demonstrated in warm-blooded forms (man, horses, and birds) in all of the States bordering the Atlantic Ocean and the Gulf of Mexico plus Michigan, Missouri, Arkansas, and Tennessee.

---

[6] Beadle, L. D.: Eastern equine encephalitis in the United States. Mosq. News 12 (2): 102–107 (1952).

Laboratory and Field Techniques and Results

The difficulties encountered in inducing virus infection in warm-blooded animals or in transmitting these viruses by means of arthropod vectors oftentimes have been overcome through sheer ingenuity. Of special interest are the arthropod-vector techniques employed by early experimenters such as Meyer et al. (*252*), Simmons et al. (*355*), Knowlton and Rowe (*213*), and Kelser (*202*). Many descriptions of the presently used methods are available (*6*, *71*, *72*, *155*, *157*, *341*, *421*).

Arthropods of many species have been ground up and injected into laboratory animals in an effort to isolate the viruses WEE, EEE, and SLE from nature (*18*, *105*, *114*, *124*, *212*, *316*, *368*). The feces of mice (*24*) and assassin bugs (*124*) have likewise been used. Mitamura et al. (*260*) used groundup mosquito eggs in an effort to show transovarial passage of Japanese B virus. The fresh brain tissue of man, taken at autopsy, has been ground and injected into horses (*338*), producing either fatal or mild cases of encephalitis. Brain tissue has also been injected into mice (*418*) with positive results.

Reeves et al. have presented special methods employed in the attempt to transmit SLE virus directly by the bite of *Culex pipiens* (*314*). The viremia of such agents is short lived, relatively speaking, and chance has been an important factor in working with them. In Trinidad, Gilyard (*116*), working with one of the longer-lived viremias, noticed that *Mansonia titillans* was predominant among the mosquitoes taken. Gilyard trapped six female mosquitoes feeding on the back of an encephalitic donkey, and by placing them on a normal-appearing donkey he obtained direct evidence of virus transmission by arthropod biting in that epidemic of Venezuelan equine encephalomyelitis.

Intranasal instillation of virus suspension has been commonly used, especially in early tests, in trying to demonstrate droplet infection via the upper respiratory tract of test animals (*175*, *302*, *388*, *411*, *413*). Several authors have resorted to the use of the embryonic chorio-allantoic membrane in order to show arthropod transmission in laboratory animals (*342*, *367*, *368*, *370*, *371*).

Isolation of Virus

The technique of Hammon et al. (*155*) for virus isolation from field-caught arthropods has become a standard; by this technique, specimens are identified under chloroform anesthesia, hermetically sealed by flame in shell vials, packed in dry ice, and held for later thawing and grinding of the specimens preparatory to injection into test animals, for which mice are generally suitable. After the mice have manifested encephalitic symptoms, their brains are removed aseptically, triturated, and passed to other mice. Upon establishment of a strain of virus, it is identified by using the neutralization test.

Many specimens of the same species of arthropod (especially mosquitoes) taken from the same place are often pooled for a single injection. Obviously, this technique demands proper speciation when possible. One notes that in preliminary studies Semliki Forest virus (*373*) was recovered from a taxonomically confused group of *Aedes* sp., and that Bunyamwera virus (*377*) was isolated from 15 specimens of a pool of 4,114 mosquitoes belonging to 14 species of *Aedes*. In this country, isolations of virus have been made from pools of arthropods containing different species (*187*, *316*). Arthropod collecting and handling under field conditions have been detailed in the following works: *115*, *213*, *240*, *293*, and *312*.

All of the life-cycle stages of certain candidate arthropod vectors (especially ticks and mites) have been utilized in attempts to show transovarial transmission of these viruses (*368*, *369*, *390*, *393*). Old and young viremic mice and infected ticks have been successfully manipulated to show transmission of SLE (*47*).

Brodie (*58*), working with SLE, was unable to infect normal mice orally with contaminated food or by intragastric injection, using laparotomy to rule out intraperitoneal infection. Details on an unusual laboratory method of virus transmission by one mouse eating another have been given by Harford et al. (*164*, *165*). Hogs were shown to be resistant to the feeding of several pints of WEE-virus-laden chick embryo materials (*241*).

Davis (*87*) made comparative studies on the ability of various mosquitoes to infect birds with EEE by biting. He showed that in a 25-day life span of an individual *Aedes triseriatus*, this mosquito succeeded in transmitting the virus to three different guinea pigs and to a cowbird by a bloodless puncture. TenBroeck and Merrill used the unusual approach of raising the "titer" of WEE-infected *Aedes aegypti* by continued serial feedings of normal mosquitoes on crushed infected mosquitoes (*398*). Growth of WEE has been accomplished by using the tissues and organs of *A. aegypti* in vitro; and it was found that the midgut of full-grown larvae and the ovaries of newly emerged adults best supported virus growth (*405*). Virus is thought to be uniformly distributed in the parts of infective *A. aegypti* (*245*). Initial feeding of arthropods on artifically infected animal hosts (via virus injection) or on virus suspensions of various kinds has, of necessity, been the rule in many laboratory tests. (References *131*, *132*, *145*, *146*, *245*, *260*, *379*, *399* depict the use of virus in blood, in brain suspensions, or in other media as a source of infective agent.)

Arthropods have been test-fed on a large number of warm-blooded candidate virus reservoirs (table 6). Of all the common routes by which infection may be given to candidate hosts, the intracranial or intracerebral injection and the dermal route have been most widely utilized. Comparative immunological studies on domestic encephalitis viruses (EEE, WEE, and SLE) and similar studies on those of foreign origin are to be found in these papers: *176*, *177*, *215*, *217*, *228*, *253*, *288*, *302*, *346*, *365*, *416*, *420*. An analysis of routine laboratory methods has been prepared by Milzer (*259*).

Laboratory Infections

Laboratory virus infections in man induced by accident or by other means serve as a source of valuable information when the cases can be adequately studied. Sulkin and Pike have furnished general review up to 1949 of viral infections contracted in the laboratory (*384*). Casals et al. summarized two such infections with Venezuelan equine encephalomyelitis which were notable for the isolation of that virus from the nasopharynx and blood of these nonfatal cases (*68*). Lennette and Koprowski (*227*) were able to study eight patients with the same disease, which apparently was dustborne. Two of the four accidental WEE cases in technicians have resulted in death (*104*, *119*, *170*). The case of WEE followed by Gold and Hampil (*119*) is especially interesting in the description of the successful use of antiencephalomyelitic immune horse serum administered in large volumes and at frequent intervals. This method is without value after CNS symptoms occur. One worker who handled EEE for 6 years and enjoyed apparent good health developed neutralizing antibodies in his serum (*284*). To date there seem to have been no cases of laboratory infection with SLE.

Epidemiology

Man is not considered to be a reservoir of encephalitis virus and may be but an accidental host (*141*). Furthermore, although devastating epizootics have occurred in the United States and in Canada among the equine species, these animals are not considered as important sources of virus agent. Disease in horses and in humans (*21*) serves as an epidemiological indicator of the presence of infection in certain areas such as California (*150*) and Washington (*144*). It is sometimes stated that the reporting of human cases of epidemic encephalitides has been more carefully done in the Western States than in the States east of the

Mississippi River. Since 1930, a total of 14,119 human cases has been reported [7] in the 22 Western States, the lowest number (226 cases) in 1948 and the highest number (3,156 cases) in 1941. The year 1950 had the highest number (554 cases) since 1941, and of these infections 333 were from California. The National Office of Vital Statistics reports a total of 27,748 human cases for the period 1926–50 in all States (the lowest number, 702, in 1932; the highest, 3,516, in 1941; and 1,135 cases in 1950).

Equine Encephalomyelitis

The term "equine encephalomyelitis" was first applied to this disease entity by C. H. Stange in November 1912 when he spoke before the Iowa State Veterinary Medical Association (*12*). At this time the disease in horses was poorly understood and was variously known as horse plague, Borna's disease, botulism, forage poisoning, and sleeping sickness (*220*). Zwick and Seifried, working on European materials, in 1924 (*433*) disclosed that the equine disease was caused by a filtrable virus; this preceded the classic work of a similar nature by Meyer and co-workers in the United States (*252*). Naturally, the horse is the animal which has been the most studied clinically. Although all of the papers cited (*17*, *86*, *154*, *240*, *275*) have interesting descriptions of equine disease, the early works of Records and Vawter (*299–303*) are noteworthy for the inclusion of a series of photographs of animals with equine encephalomyelitis in progressive stages. Motion pictures were taken of horses with symptoms of the disease in the Montana outbreak of 1936 (*275*).

Equine epidemics (presumably involving EEE and WEE) have been recorded for the United States in 1847, 1850, 1867, 1882, 1897, 1912, 1919, 1920, 1921, 1923–28, and 1930–31 (*220*, *249*, *267*). Morbidity and mortality data for these early epidemics are relatively scarce (*249*). The 1912 epidemic seems to have been rather widely distributed, covering 17 States, with an estimated loss of 35,000 horses and mules in Kansas and Nebraska (*249*) alone. Meyer early thought of these first epidemics as being cyclic in nature since the peaks seemed to come at 10- to 15-year intervals. Since 1936, yearly reports by Miller (*255*, *256*), Mohler (*263*, *265*, *266*, *268–271*) and Simms (*356–361*) from the Bureau of Animal Industry (U. S. Department of Agriculture) have presented a continuous picture of the disease in horses. The data are accumulated through the cooperation of local veterinarians, livestock officials, and Bureau inspectors and are often based on questionnaires. A 15-year summary (*361*) up to 1951 shows the following peak years, with numbers of reported cases: 1935 (23,512); 1937 (173,889); 1938 (184,662, the highest number in 15 years); 1940 (16,941); 1941 (36,872); and 1944 (19,590). Since 1944, incidence has gradually dropped to the lowest recorded figure of 762 for 1951.

The number of deaths from equine encephalomyelitis during the period 1935–51 ranged from 274 (1951) to 8,210 (1941), with a total of 33,563 equine deaths recorded for the 13-year period (1939–51). Equine deaths are not recorded for the years 1935 through 1938 in the May 1951 report by Simms (*361*). The mortality per 100 affected animals has ranged from 21 in 1938 (the peak year of the period 1935–50) to 60 in 1949 (with only 4,037 cases), with an average mortality of 33.2 since 1938.[8] On the basis of cursory analysis, there is apparent indication of a possible spread and shifting of the disease (lumping the incidence of WEE and EEE) if one considers the number and location of the States affected either epidemically or endemically, as indicated by the Bureau of Animal Industry reports, noting cases in 17 States in 1912, and 28, 17, 30, 39, 42, 38, 33, 35, 34, 33, 33, 37, 33, 35, 37, 33, and 28 States involved for the years 1935 through 1951, respectively.

Geographic Distribution

There is no adequate evidence that these diseases have spread geographically from a central focus in North America. As an example of the distribution, 96 percent of all cases were

[7] It should be pointed out that this rather large number of cases primarily represents the reporting of "infectious encephalitis" and not "arthropod-borne virus encephalitides" alone, for the number of laboratory confirmations of the latter cases is a small proportion of the total figure.

[8] Combination of mortality rates for WEE and EEE in the horse is unfortunate since it is sometimes stated that the rate for WEE usually averages 20–40 percent, whereas the rate for EEE averages 80–90 percent.

located west of the Mississippi River in 22 States in 1940; whereas in 1950, cases were rather evenly and lightly distributed north of the Ohio River and west of the Mississippi River, with the highest concentrations (5 or more cases per 1,000 equines) in the extreme southern sectors of Oregon and in parts of California. In general, California, especially the central regions, appears to have been the 1950 focal point of disease distribution. During the epizootic period of 1951, California, Nebraska, and Missouri had the highest indexes. The disease has remained rather statically distributed since the peak year of 1938, while the lowering of the index has been gradual. This has been coupled with a decreasing equine population in the United States—from 15,245,000 (1938) and 9,130,000 (1948) to a figure of 7,463,000 in 1951. The range in the number of States affected by the disease has been 17 (1936) to 41 (1939); in the latter case, all States except Mississippi, Tennessee, South Carolina, West Virginia, Pennsylvania, New York, and New Hampshire had reported the equine disease. At this writing, equine disease has not been reported from Tennessee, West Virginia, and Pennsylvania. The following Provinces in Canada have reported equine infections: Ontario, Manitoba, and Saskatchewan, where the first case was noted in 1935 (*107*). Actual virus isolations of WEE and EEE from equines of 29 States up to 1951 are presented in table 5.

These equine diseases probably are widely distributed in many foreign countries, as evidenced by the following references: EEE and WEE in Canada (*341, 348*); EEE in Panama (*95, 196, 199, 380*), in Brazil (*95, 226*), in Mexico (*95, 348*), in Cuba (*95*), in the Dominican Republic (*3, 94, 95*), and in the Philippine Islands (*231*); WEE in Argentina (*253, 176*); while equine encephalomyelitis is assumed to have been reported for both Hawaii and Puerto Rico (*361*).

Many data representing the various facets of epidemiology have been gathered in numerous surveys of these human and animal diseases in North America, of which the following list represents the major efforts for each: SLE (*18, 53, 69, 129, 137, 144, 149, 150, 152, 154, 156, 159, 179, 182, 184, 221–224, 229, 242, 382*); WEE (*66, 77, 83, 90, 92, 106, 137, 149, 150, 152, 154, 156, 158, 160, 167, 179, 181, 184, 291, 299, 315, 430*); EEE (*87, 94, 98, 99, 110, 112, 158, 189, 206*). With respect to field methods, the questionnaires designed for use in the famous Missouri SLE outbreak and the questionnaire supplied observers in equine outbreaks (*249, 263*) are of interest. Probably the most complete study of arthropod vectors of EEE outbreaks was that of Feemster and Getting (*99*), in which the data of an extensive survey have been presented. Analysis of the recent (1948–49) epidemic of EEE in the Dominican Republic, by Eklund and co-workers (*94, 95*), was made by use of the most advanced techniques.

Climate and Seasonal Appearance

Climate and the seasonal appearance of these diseases (WEE, EEE, and SLE) have received considerable attention from epidemiologists. Since all but one (Russian tick-borne encephalitis) of the epidemic encephalitides are held to be mosquito-borne, and since they have occurred epidemically principally in temperate regions, the distribution has been primarily in summer and fall months. The Bureau of Animal Industry reports on horses have selected three seasonal periods (pre-epizootic, epizootic, and postepizootic) as useful indicators. As an example, these periods, with respective months and number of cases per month, are presented for 1950 (*361*): preepizootic—January (7), February (6), March (3), April (11), and May (41); epizootic—June (94), July (153), August (218), September (303), October (141); postepizootic—November (39) and December (7).

Many of the equine outbreaks in the United States have appeared under hot, dry conditions. Such was the case in the San Joaquin Valley in 1931, where the number of cases rose when the temperature ranged between 90° and 100° F. At that time it was tentatively thought that this condition brought out inapparent cases (*249*). High humidity was recorded for the disastrous horse epidemic of 1912 in Kansas and in more recent outbreaks in Saskatchewan, Canada. Records and Vawter (*300*) reported three infected horses in Nevada in November. These animals had been moved in from a high mountain ranch to another corral to join a herd of horses in which there had been no apparent infection for 15 months, and within 8 days, each of the three animals developed a fulminating viremia. The authors felt that, in general, disease transmission via mosquitoes was unlikely since several "localized outbreaks with a high mortality have occurred in the winter months."

Lambert et al. (*219*) found that young horses were more susceptible to WEE than older ones, but that sex differences did not show in a statistical analysis of a Miles City, Mont., outbreak in 1939. Low indexes of clinical cases in animals over 2 years old may in large part be due to immunity developed by clinical or, more probably, nonclinical infection at an early age. At present, most infections appear in horses under 2 years of age. The fact that mice (*283*) and chickens (*262*, *385*) are more susceptible to viruses when young forms the basis for several laboratory procedures. Young birds have been studied as sources of these viruses in nature (*379*). Chicks may serve as a short-term source of WEE virus for mosquitoes (*148*).

Certain unusual findings in equine epidemics are included here for consideration. Fulton (*107*) noted that Saskatchewan outbreaks of WEE occurred in areas known to be very dry for months and to be without mosquitoes. Meyer also commented on the same thing early in the history of the disease (*251*). Early studies on horse botulism showed the disease (which included "sleeping sickness") to be most prevalent in the fall and winter, which is a seasonal distribution not generally found at present in WEE and EEE. In a notable virus isolation of EEE from a Florida horse during the middle of January 1939, the wintertime occurrence was tentatively attributed to the probable presence of mosquitoes (*15*, *285*). One horse case of WEE occurred in the center of an area not completely treated for the insect (*163*).

Human Encephalitis

An interesting human case of WEE (*324*) occurred in the center of Chicago, and allegedly careful checking showed no known contact with arthropods. One wonders at the western distribution of SLE virus in the United States and its apparent absence in Canada, as reflected in reporting. Certain manifestations of these viruses which appear unusual at present have been reported, such as the development of a virus which caused lymphocytic choriomeningitis in monkeys and mice upon the injection of a virus suspension taken from a patient dying with SLE (*31*); the suggested relationship existing between SLE and epidemic keratoconjunctivitis (*74*, *332*); certain changes in viruses (*406*), especially the isolation of a mixture of viruses from which both SLE and WEE were obtained (*162*); and the discovery of an agent in normal mouse urine which neutralized large amounts of SLE virus (*286*).

In recent years SLE has become increasingly more important in California. At present, two established endemic foci of WEE are Weld County, Colo., and Kern County, Calif. Moreover, new viruses have been discovered, such as the California virus (*159*).

The age distribution of human cases of virus encephalitis has been considered, especially the distribution of SLE (*50*). In general, age distribution varies with the immunity and type of exposure of a population. SLE was shown to have a higher case index among older groups but no preference for sex or color.

Transmission of Virus

Kelser's researches (*193*) on the direct transmission of WEE virus through the bloodsucking activity of an arthropod are classic in the history of the epidemic encephalitides. Kelser used *A. aegypti* of Philippine Islands origin and WEE virus from the brain of a North Dakota horse in the first laboratory transmission of encephalomyelitis. He injected virus suspensions into three guinea pigs. Mosquitoes were initially fed on the three guinea pigs at 48, 72, 96, 120, and 144 hours following injection, a period which covers the febrile stages of the disease in guinea pigs. Each lot of *Aedes* was then test fed on normal guinea pigs 6 days from the time of initial feeding, an interval thought of as the incubation period. All guinea pigs died from encephalitis from each of the mosquito test groups except the last one (144 hours). Some of the mosquitoes were infective up to 18 days after initial feeding. A horse which also was infected by the above method of direct transmission by arthropod bite died with encephalomyelitis. TenBroeck and Merrill learned later (*399*) that *Aedes* sp. must

Table 1. Results of attempted virus isolations from nature (arthropods)

Arthropods studied	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis
Aedes aegypti	−315	−315	
Aedes campestris	−151	−151	
Aedes cinereus	−151, 156	−151, 156	
Aedes dorsalis	+147, 149, 159; −151, 156, 424	+147, 149; −151, 156	
Aedes increpitus	−151, 156	−151, 156	
Aedes lateralis (sticticus)	−151, 156	−151	
Aedes nigromaculis	−137, 149, 156, 160, 315, 424	−137, 315	
Aedes scapularis	−158	−158	−94, 158
Aedes sollicitans	−158	−158	−158
Aedes taeniorhynchus	−158	−158	−94, 158
Aedes triseriatus	−160		
Aedes trivittatus	−160		
Aedes vexans	−124, 137, 149, 151, 156, 158, 160, 315, 424	−137, 149, 151, 158, 315	−158
Anopheles albimanus			−94
Anopheles crucians	−158	−158	−94, 158
Anopheles freeborni	+137, 147; −156, 151	−137, 151, 156	
Anopheles pseudopunctipennis	−158	−158	−158
Anopheles punctipennis	−137, 156, 160	−137	
Anopheles quadrimaculatus	−158	−158	−158
Culex bahamensis			−94
Culex coronator	−158	−158	−158
Culex erraticus	−158, 315	−158, 315	−158
Culex pipiens	+137, 147; −151, 156, 160	+137; −151, 156	−98
Culex quinquefasciatus	−147, 149, 158, 315	−149, 158, 315	−158
Culex restuans (territans)	+281; −124, 160		
Culex salinarius	−158, 160, 315	−158, 315	−158
Culex stigmatosoma	+147, 159; −149	−147, 149, 159	
Culex tarsalis	+4, 8, 23, 137, 147, 149, 151, 156, 159, 160, 211, 424; −124, 158, 159, 311, 315	+132, 137, 151, 156; −158, 315	−158
Culex thriambus	−149	−149	
Culiseta incidens	−137, 151, 156	−137, 151, 156	
Culiseta inornata	+137, 147; −149, 151, 156, 315	−137, 149, 151, 156, 315	
Culiseta melanura			+26, 73
Deinocerites cancer			−94
Deinocerites spanius	−158	−158	−158
Mansonia perturbans			+188, 206

be fed on virus suspensions (or on animals injected with virus suspensions) of high titer in order to secure uniform results.

Very few isolations of EEE have been made from arthropods in nature (table 1), although mosquitoes (as well as horses) were suspected as factors in disease transmission in early epidemics (*425*). Chamberlain and co-workers made an isolation from the mosquito *Culiseta melanura* in Louisiana (*73*). Before this, the virus had been detected in Tennessee in chicken mites and chicken lice (*187*), and in Georgia in a mosquito, *Mansonia perturbans* (*188*, *206*).

The present record on WEE virus isolations (as based on initial dates) from the important vector species *Culex tarsalis* in the United States is as follows: Washington, 1941; California, 1943; Nebraska, 1943; Montana, 1944; and North Dakota, 1948. Through 1950, 144 isolations of WEE and 14 isolations of SLE virus have been made from this important mosquito. Thompson and co-workers have recently reported on the isolation of WEE virus from *A. dorsalis* of midwestern United States for the first time.[9]

One of the main purposes of this survey is to present tabulations on intermediate and definitive hosts. Studies are summarized in tables 1 through 6, which are complete through 1951. In the use of these tabulations it must be re-

[9] Thompson, G. A., Howitt, B. F., Gorrie, R., and Cockburn, T. A.: Encephalitis in Midwest. VI. Western equine encephalomyelitis virus isolated from *Aedes dorsalis* Meigen. Proc. Soc. Exper. Biol. & Med. 78: 289–290 (1951).

Table 1. Results of attempted virus isolations from nature (arthropods)—Continued

Arthropods studied	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis
Mansonia titillans	−158	−158	−158
Psorophora ciliata	−158, 160, 315	−160, 315	−158
Psorophora confinnis	−315	−315	
Psorophora cyanescens	−158, 315	−158, 315	−158
Psorophora discolor	−158	−158	−158
Psorophora pygmaea			−94
Psorophora signipennis	−158, 160, 315	−160, 315	−158
Bdella sp	−424		
Dermanyssus americanus	+254, 316;[1] −311	−316	
Dermanyssus gallinae	+381; −311, 315, 424	+367, 368; −315	+187
Bdellonyssus bursa	+382		
Bdellonyssus sylviarum [2]	+162, 316, 424; −311	+4, 162	
Pteronyssus sp	−424		
Argas persicus	−149	−149	
Dermacentor americanus	−316		
Dermacentor andersoni	−125, 127	−149	
Haemaphysalis leporis	−149	−149	
Ixodes sp	−156	−156	
Ornithodoros turicata	−149	−149	
Ground squirrel fleas	−149	−149	
Rabbit fleas	−149	−149	
Assassin bugs	−127		
Triatoma sanguisuga	+124, 209		
Cimex lectularius	−160, 156	−156	
Menopon pallidum *Eomenacanthus stramineus*			+187
Ground squirrel lice	−149	−149	
Ceratopogoniids	−156	−156	
Haematobia serrata	−315	−315	
Hippelates pusio	−156	−156	
Siphona irritans	−156	−156	
Musca domestica	−156	−156	
Simulium sp	−156	−156	
Stomoxys calcitrans	−124, 156	−156	
Stomoxys sp	−127		
Tabanus sp	−156	−156	

[1] Original collection was a mixture of *D. americanus* and *B. sylviarum*. [2] Also listed as *Fonsecaonyssus sylviarum*.

NOTE: + or − preceding bibliographic number indicates positive or negative results during a particular trial. (?) indicates indefinite statement in paper reviewed.

membered that in many instances more than one reference relates to a single test. Both cold- and warm-blooded animals are listed as they were named in the literature, without regard to modern taxonomic placement. While many arthropod species have been tested for the presence of naturally acquired virus infections, deductions based on epidemiological factors have compelled workers to seek virus isolations principally from mosquitoes, ticks, and mites.

In these tables, demonstration of the presence or absence of virus during a particular trial is denoted by a plus or minus symbol with the appropriate bibliographic number. These symbols are qualitative and imply only that one or more tests were negative or positive as the case may be. The few parenthetic question marks are those placed by this author and indicate indefinite statements in the paper reviewed. Comparisons may be made with these tabulations and those given previously by other authors, of which the following are particularly informative: Davis (*87*), Cockburn et al. (*77*), Hammon et al. (*152*), Manzelli (*235*), and Syverton and Berry (*392*). The summaries of Manzelli are especially useful (*235*).

Table 1 indicates that mosquitoes have been used mostly in attempting to isolate viruses, of which the most numerous tests have been with WEE.

Table 2 lists certain arthropods (*A. aegypti*, *Aedes cantator*, *Aedes sollicitans*, *Aedes taeniorhynchus*, *Aedes vexans*, *C. tarsalis*, *Dermacentor andersoni*, and *Dermacentor variabilis*) which demonstrate rather uniform ability to transmit viruses directly by biting. In

Table 2. Results of attempted direct[1] virus transmission by bite of arthropod

Arthropods studied	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis
Aedes aegypti	+114, 172, 193, 198(?), 246, 398.	+45, 354; −18, 146	+87, 114, 345, 399
Aedes albopictus	+355		
Aedes atropalpus		+261	+87, 99
Aedes cantator	+247(?)		+87, 99, 247, 399
Aedes cinereus			−87
Aedes dorsalis	+233, 234; −152, 172, 212		
Aedes togoi		+260(?)	
Aedes nigromaculis	+233		
Aedes sollicitans			+87, 99, 247
Aedes taeniorhynchus	+195, 197		+99; −195, 197, 399
Aedes triseriatus			+87, 99
Aedes varipalpus	−305		
Aedes vexans	+193(?), 194		+87, 99, 399
Anopheles freeborni	−137, 152, 172		
Anopheles punctipennis		−146	−87
Anopheles quadrimaculatus	−247	−18, 423	−87, 399
Armigeres obturbans [2]		+261	
Culex apicalis			−87
Culex erraticus		−146	
Culex pipiens		+137; −18, 105	−87, 399
Culex pipiens pallens		+260, 261	
Culex quinquefasciatus			−24, 25
Culex salinarius			−87
Culex tarsalis	+145	+146	
Culex territans (restuans)			−87
Culex tritaeniorhynchus		+261	
Culiseta inornata	+137		
Culiseta melanura			−87
Mansonia perturbans			−87
Psorophora ciliata	−145		
Psorophora confinnis	−145	−146	
Psorophora cyanescens	−152		
Uranotaenia sapphirina			−87
Wyeomyia smithii			−87
Dermanyssus gallinae		+369	+24
Bdellonyssus sylviarum	−23	−23	
Dermacentor andersoni	+389, 390, 393		
Dermacentor variabilis	−124	+47, 48, 49	
Diamanus montanus	−152		
Echidnophaga gallinacea	−163		
Ctenocephalides felis	−152		
Triatoma sanguisuga	+124		
Siphona irritans	−172		
Simulium vittatum	−211		
Stomoxys calcitrans	−152		
Tabanus punctifer	−172		

[1] Direct transmission by bite is inferred, i. e., the arthropod became infected by biting a test animal which had been infected by various means. [2] Probably *A. subalbatus*.

NOTE: + or − preceding bibliographic number indicates positive or negative results during a particular trial. (?) indicates indefinite statement in paper reviewed.

table 3 a remarkably high number of records appear indicative of positive transmission by various methods. It is obvious that the main attention has been upon tests involving mosquitoes in contrast with other arthropods. As indicated in table 4, little work has been published on the detection of neutralizing antibodies of EEE in animals. Additional data on the detection of neutralizing antibodies in the serums of a wide range of Colorado small mammals are detailed in the paper by Hutson et al.[10] in which only 6 out of the 300 tests were positive (5 positive WEE and 1 positive SLE tests). As shown in table 5, very few isolations of viruses have been reported from animals other than horses. Virus has been isolated from

[10] Hutson, G. A., Howitt, B. F., and Cockburn, T. A.: Encephalitis in Midwest. VII. Neutralizing antibodies in sera of small wild animals—Colorado, 1950. Proc. Soc. Exper. Biol. & Med. 78: 290–293 (1951).

the blood of wild birds (*13*, *206*, *207*, *379*), the first isolation being that by Kissling and co-workers (*207*) working with purple grackles, and has been studied in the blood of laboratory chickens (*148*) and experimentally infected wild birds [11] by Hammon and co-workers. These authors infer that wild birds (species of sparrows, finches, and blackbirds) are probably more important than domestic ones as sources of WEE and SLE virus infection for mosquitoes in Kern County, Calif. Cox and co-workers isolated WEE virus from a prairie chicken taken in a sick condition in North Dakota (*83*). In table 6 the entries merely indicate those animals which have been used in laboratory tests with particular viruses.

[11] Hammon, W. M., Reeves, W. C., and Sather, G. E.: Western equine encephalomyelitis and St. Louis encephalitis virus in the blood of experimentally infected wild birds and epidemiological implications of findings. J. Immunol. 67: 357–367 (1951).

None of the previous tests on transmission with all of the manipulation of viruses and host animals have the apparent directness of Gilyard's method (*116*) on the passage of Venezuelan virus from a naturally infected animal to a normal animal by the bite of an arthropod. Yet proof was not given that the test animal was not already infected with virus. The ability of an arthropod to transmit these diseases under laboratory conditions and under natural conditions may not be the same. Obviously, to show that an outbreak of human illness follows (after a proper arthropod virus incubation period) the positive isolation of a virus from animals with apparent or inapparent viremia in the same neighborhood would be of epidemiological interest; to isolate the virus from the arthropod vector at the same time would be relatively significant. The situation in Yakima Valley, Wash., in 1942 should be considered from this viewpoint (*137*, *152*).

Table 3. Results of attempted indirect [1] transmission of virus by laboratory methods (arthropods)

Arthropods studied	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis
Aedes aegypti	+247	−18	+114; −245, 399
Aedes albopictus		+261	
Aedes campestris	+151		
Aedes cinereus		−151	
Aedes dorsalis	−212, 234	−151	
Aedes increpitus		−151	
Aedes lateralis (*sticticus*)		+146, 147, 157; −151	
Aedes nigromaculis	−234	+146, 147; −151	
Aedes sollicitans			+247
Aedes taeniorhynchus		+146, 147, 158	
Aedes vexans		+146, 147, 151	
Anopheles freeborni	−145	−146, 151	
Anopheles punctipennis	−145	−146	
Anopheles quadrimaculatus		−18	
Armigeres obturbans (*subalbatus*)	+261	−261	−261
Culex coronator		+132, 146, 158	
Culex pipiens	−145, 247	+105, 131, 132, 146, 314; −18, 151	
Culex pipiens pallens	+261	+261	+261
Culex quinquefasciatus		+158; −146	
Culex stigmatosoma [2]	−145(?)	−146	
Culex tarsalis	+145, 157	+132, 146, 157, 158	
Culex tritaeniorhynchus	−261	+261	+261
Culiseta incidens	+145	+146, 147, 151	
Culiseta inornata	+145	+146, 147; −151	
Psorophora ciliata	−145	−146	
Psorophora confinnis	−145, 158		
Dermanyssus gallinae	+382(?)	+369, 370, 371	
Amblyomma maculatum	−263		
Simulium vittatum	−212		
Stomoxys calcitrans	−263		

[1] Indirect transmission is inferred; i. e., the arthropod became infected by the use of various, somewhat artificial techniques.

[2] Reference 190 states that *C. stigmatosoma* is capable of experimental transmission of WEE. Source of data not given.

NOTE: + or − preceding bibliographic number indicates positive or negative results during a particular trial. (?) indicates indefinite statement in paper reviewed.

300402—54——3

Table 4. Results of tests of naturally infected warm-blooded hosts for neutralizing antibodies in serums

Hosts studied [1]	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis
Birds:			
Birds, wild	+4, 22, 62		+4, 22, 62
Blackbird, Brewer's	+311; −154, 315	+311; −154	
Blackbird, red-winged	−154, 311	+311; −154	
Chicken	+4, 154, 158, 159, 311, 315; −154, 158, 160, 311, 315	+154, 159, 311; −154, 158	−158
Coot	−154, 315	+154; −154	
Crow	−315	+315; −315	
Dove	−154	+154; −154	
Dove, mourning	+315; −158, 311, 315	+311; −158, 315	−158
Duck	+154; −154, 158	+154; −154, 158, 311	−158
Egret, snowy			+25
Flicker	+154	+154; −154	
Goose	+154; −154	+154; −154	
Guinea fowl	−158	−158	−158
Heron, night			+4
House finch	−311	−311	
Ibis, white			+4, 25
Jackdaw	−158	−158	−158
Killdeer	+154, 311; −154	+311; −154	
Kingbird, eastern	−311	−311	
Kingfisher	−311	−311	
Magpie	−311	+311	
Meadowlark	+315; −311, 315	+311, 315; −315	
Nighthawk	−311	−311	
Owl	+154; −154	+154; −154	
Pheasant	+154; −154, 160, 311	+154, 160, 311; −154	
Pigeon	+154; −154, 311	+154; −154, 311	
Quail (bobwhite, etc.)	+311; −315	+311	
Robin	+311	+311	
Shrike	−311	+311	
Snipe, Wilson	−311	+311	
Sparrow, English	−311	+311	
Sparrow, song	−311	−311	
Swallow, bank	−311	−311	
Swallow, barn	−311	−311	
Turkey	+154; −154, 158	+154; −154, 158	−158
Woodpecker, hairy	−315	+315; −315	
Animals:			
Calf	+315; −315	−315	
Cat	−154	−154	
Chipmunk	+154; −154	−154	
Colt	−315	−315	
Cow	+154, 158, 160, 315; −154, 158, 315	+154, 158; −154, 158	+160(?), 158; −158
Dog	+154; −154	+154; −154	
Goat	+154; −154	+154; −154	
Gopher, pocket	−154	+154; −154	
Horse	+154, 158	+144, 154, 158, 160, 293, 315; −154, 315	+154
Monkey		+374	
Mouse, field	+154; −154	−154	
Mouse, whitefoot	+154; −154	−154	
Mule	+154	+154	
Pig	+154; −154	+154; −154	
Rabbit, cottontail	−154, 158, 315	+154; −154, 158, 315	−158
Rabbit, jack	+154; −154, 315	+154, 315; −154, 315	
Rat, Alexander	−154	+154; 154	
Rat, Norway	−154	+154; −154	
Rat, wild	−158	−158	−158
Sheep	+154; −154	+154; −154	
Skunk			+4, 22
Squirrel, ground	+154; −154	+154; −154	
Squirrel, red fox	−315	+315	
Weasel	+154; −154	−154	

[1] Names as cited in literature.

NOTE: + or − preceding bibliographic number indicates positive or negative results during a particular trial. (?) indicates indefinite statement in paper reviewed.

The longevity of SLE has been investigated in mites (*368*, *369*), in ticks (*48*), and in mosquitoes (*423*). Mitamura et al. (*261*) have found no evidence that the virus of Japanese B encephalitis overwinters in the mosquito; this has also been implied by Wooley and Armstrong (*428*), working on SLE virus. Winter collections of mosquitoes from Yakima Valley, Wash., were found to be virus negative (*137*, *149*). In one case (*9*), the test mites on doves did not outlast the winter.

A few ecologic studies on suspected or known natural arthropod vectors of these diseases appear in the literature. A recent paper by Abdel-Malek made substantial contributions to the biology of *Aedes trivittatus* (*27*). Preliminary notes on the ecology and distribution of certain aedines in California are to be found in reference 29 as well as in notes on the culicines of Colorado (*46*). A useful paper on the distribution of *Aedes* sp. in New England was written by Feemster and Getting (*99*). The feeding habits of Saskatchewan mosquitoes have been described by Rempel et al. (*323*); comparisons may be made with similar data on Washington mosquitoes (*313*) and those from California (*149*, *159*, *163*). Rempel has an unpublished manuscript on ecologic data on mosquito infestations with respect to outbreaks of encephalomyelitis in Saskatchewan. Gjullin et al. (*118*) have contributed to the ecology of *Aedes* sp. in the northwestern United States. Merrill et al. (*247*) studied *Aedes* sp. in connection with encephalomyelitis in New Jersey, Delaware, and Virginia in 1934. A seasonal study has been made of St. Louis County mosquitoes, especially *C. pipiens* (*88*). Ecology of mosquitoes in relation to encephalitis and irrigation has commanded the attention of California workers (*310*, *402*, *403*). Interesting observations were made on the ecology of Trinidad mosquitoes in respect to their possible Venezuelan origin (*116*). Basic work on *C. tarsalis* has been reported by several authors (*35*, *59*, *109*, *190*, *290*, *317*), and of these studies,

Table 5. Results of tests to find encephalitis virus in warm-blooded animals in nature.

Hosts studied [1]	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis
Birds:			
Blackbird, red-winged	+13, 379	−379	−379
Chicken	−127		
Chicken, prairie	+83		
Crow	−127		
Duck, wild	−127		
Grackle			+26,207
Hawk	−127		
Magpie	+13, 379	−379	−379
Owl	−127		
Pheasant	−127		+40, 41, 344, 408, 411
Pigeon			+102
Animals:			
Cat	−127		
Deer	+83, 348		
Horse [2]	+186, 252, 256	+84(?)	+52, 62, 112, 136, 196, 199, 226, 231, 256, 298, 341, 383
Monkey			+231
Mouse, field		−122	
Mouse, meadow		−122	
Pig	+241; −127		
Rabbit, jack	−127		
Rat, wild	−127		
Squirrel, ground	+126(?), 127		

[1] Names as cited in the literature.

[2] Virus isolations have been made from horse materials in these States: Alabama (WEE and EEE); Arizona (WEE); Arkansas (EEE); California (WEE); Colorado (WEE); Delaware (EEE); Florida (EEE); Georgia (EEE); Idaho (WEE); Illinois (WEE); Iowa (WEE); Kansas (WEE); Kentucky (WEE); Louisiana (EEE); Maryland (EEE); Massachusetts (EEE); Michigan (WEE and EEE); Minnesota (WEE); Missouri (EEE); Montana (WEE); Nevada (WEE); New Jersey (EEE); North Carolina (EEE); North Dakota (WEE); South Carolina (EEE); South Dakota (WEE); Texas (WEE and EEE); Utah (WEE); and Virginia (EEE).

NOTE: + or − preceding bibliographic number indicates positive or negative results during a particular trial. (?) indicates indefinite statement in paper reviewed.

that of Jenkins (*190*) is a particularly informative summary.[12] Of the miscellaneous arthropods indicted in the carrying of these viruses, *Triatoma sanguisuga* has been investigated bionomically (*123*, *209*). Also, there has been much research on the ecology of *D. andersoni* in connection with the transmission of WEE (*389–390*, *393*), and other diseases.

[12] Valuable data on this subject are to be found in this work: Brookman, B.: The bionomics of *Culex tarsalis* Coquillett in irrigated areas of a Lower Sonoran environment. Library, University of California, Berkeley (unpublished thesis), January 1950.

Endoparasites of warm-blooded animals have been suspected of transmitting these viruses (*163*). No virus was found in strongylid ova or larvae from feces of a horse infected with EEE, or from strongylid adults or *Gasterophilus* larvae collected post mortem (*24*). *Strongylus* sp., taken from encephalitic and

Table 6. Reference index of various hosts used in laboratory studies

Hosts studied [1]	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis
Birds, wild	320	—	
Chicken	93, 94, 145	35, 139, 369	24, 114, 344, 395, 396
Cowbird	—	—	87
Dove, white	23	23, 131, 139	
Duck	114, 145, 320	139	114
Egret	—	—	395, 396
Goose	262, 320	—	
Guinea fowl	349	—	349
Hawk	320, 330	—	
Owl	392	—	392
Pigeon	111, 114	131	87, 204
Sea Gull	330	—	
Sparrow, English	—	—	87
Stork, white	320, 321	—	
Turkey	—	—	395, 396
Vulture	320, 322	—	
Alligator	330	—	
Calf	111, 114	—	
Cat	111, 114, 262	139	114
Chipmunk	262	—	
Cow	262	35	114
Dog	111, 114, 335	35	114
Ferret	262	18	
Fox	262	—	
Goat	114, 231, 262	—	114, 253
Gopher	388, 389,[2] 390	—	
Guinea pig	174, 175, 193, 262, 390	18, 139	87, 100
Hamster	262, 415	—	415
Horse	84, 174, 193, 234, 253, 300, 302, 338, 345, 351, 413	18, 35, 84, 139, 153, 351	24, 25, 100, 196, 247, 257
Monkey	—	183, 273	
Mouse	106, 114, 262, 386, 405	47, 58, 131, 164, 165, 166, 286, 332, 421	24, 87, 114, 204
Mouse, field	—	122	
Mouse, meadow	—	122	
Mule	—	18	
Opossum	391	—	391
Pig	114, 241, 262	35	114
Rabbit	114, 262	18	114
Rabbit, cottontail	391	—	391
Rabbit, jack	391	—	
Rat, cotton	124	—	124
Rat, white	114, 262	364	114
Rat, wood	124	—	124
Sheep	111, 114, 262	35	114
Snake	330	—	
Vole, field	391	—	391
Woodchuck	391	—	391

[1] Names as cited in the literature.
[2] Listed as *Citellus richardsonii*.

Table 7. Historical highlights with special reference to WEE, SLE, and EEE

Year	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis	Miscellaneous encephalitides
1847	First report of horse epidemic in United States (*249*).	------	Horse disease (EEE?) in Long Island, New York (*112*).	
1867	Early report of horse epidemics in United States (*220*).	------	------	
1902	------	------	Horse disease (EEE?) in North Caroline (*249*).	
1912	Horse disease in Kansas and Nebraska (*343*).	------	Horse disease (EEE?) in Maryland, New Jersey, and Virginia (*112* and *249*).	
	Horse disease in Colorado (*97*).			
	Term "equine encephalomyelitis" first used (*12*).			
1915	------	------	------	Earliest recognition of encephalitis as clinical entity (*85*).
1918	Horse disease in South Dakota (*343*).	------	------	Encephalitis lethargica in Great Britain (*232*).
1919	Horse epidemic in Montana (*343*).	------	------	First recognition of von Economo's disease in United States (*276*).
	Horse disease in Colorado (*97*).	------	------	Acute encephalomyelitis (X disease) in Australia (*76*).
1924	------	------	------	Original discovery that a filtrable virus was cause of European equine encephalomyelitis (*433*).
1926	Human disease in California (*43*).	------	------	
1930	First isolation of virus from horse (*252*). Horse disease in California (*167*).	------	------	
1931	Virus isolation from horse—California (*252*).	------	------	First diagnosis of Rift Valley fever in man (*86*).
1932	Recovery of virus from blood of test animals (*174*). Horse disease in Colorado (*97*); in Nevada (*299*). Prediction of human infection by virus (*248*).	Outbreak in Paris, Ill. probably SLE (*79*).	------	
1933	Laboratory transmission of disease to horses and guinea pigs by bite of mosquito, *Aedes aegypti* (*193*). Calves and pigeons found susceptible (*111*).	Outbreak in St. Louis (*79*). Human cases in New York (*419*). First isolation of virus from human brain (*273*). Recognition of new disease entity (*418*).	Epizootic along Atlantic coast (*112*). Isolation of virus from animal brains—Virginia (*112*). First identification of eastern strain—Massachusetts (*397*). Epizootic along Atlantic coast (*112*).	
1934	Argentine equine encephalomyelitis and WEE "immunologically identical" (*329*).	No cross immunity with Japanese B virus (*420*). First indictment of mosquitoes as SLE vector (*229*). Recognition of SLE as new disease entity (*272*).	Virus transmission by *Aedes sollicitans*, *A. cantator* and *A. aegypti* (*247*).	Viruses of vesicular stomatitis and encephalitis are distinct (*81*).

Table 7. Historical highlights with special reference to WEE, SLE, and EEE—Continued

Year	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis	Miscellaneous encephalitides
1935	First recorded cases in horses in Canada (*67*). Disease transmission by bite of mosquito, *Aedes vexans* (*194*).	Mosquitoes not indicted as SLE vector (*18*).	Existence of eastern and western viruses confirmed (*346*). Virus transmission by *Aedes taeniorhynchus* and *A. vexans* (*398*).	Similarities of WEE and Argentina strain noted (*176*).
1936	Horse disease in Montana (*82*). Tick, *Dermacentor andersoni*, indicated as vector (*389*). Transmission by *Aedes albopictus* (*355*).	----------	----------	SLE and Japanese B viruses antigenically distinct (*192*). Japanese B encephalitis virus established nosologically (*192*).
1937	Horse disease in Canada (*106*). Disease transmission by bite of mosquito, *Aedes taeniorhynchus* (*195*); by *Dermacentor andersoni* (*390*).	First transmission of virus by bite of culicine mosquitoes (*260*). SLE established endemically in St. Louis, Mo. (*121*).	Horse disease in Panama (*196*).	
1938	Human disease in Minnesota (*93*). Human disease in California (*66*). Growth of virus in vitro (*405*). First recovery of virus from blood of man (*180*). First virus isolation from brain of man (*178*). Human encephalitis in North Dakota (*56*). Epizootic in Canada (*106*). Use of vaccines in man (*37*).	Human encephalitis in North Dakota (*56*).	Epizootic in New England (*251*). Birds are possible reservoirs (*395*). Virus isolation from human brain—Massachusetts (*103*). Recovery of virus from pigeon brain in nature—Massachusetts (*102*). Isolation of virus from pheasants—New Jersey (*408*).	
1939	Isolation of virus from horse brains in Texas (*52*). Human disease in California (*66*).	----------	Epizootic along south Atlantic coast (*112*). Virus transmission by *Aedes atropalpus* and *A. triseriatus* (*87*).	Mosquito transmission of lymphocytic choriomeningitis (*80*). Horse disease in Argentina (*330*). Isolation of VEE virus (*216*). Use of vaccination against VEE (*216*).
1940	Antibody survey in Colorado with positive results (*79*). Virus isolated from *Triatoma sanguisuga*—Kansas (*209*); from ground squirrel—Canada (*126*).	Virus transmission in *Culex pipiens* (*105*).	----------	
1941	Epidemic in North and South Dakota, Minnesota, and Nebraska (*79*). Isolation of virus from prairie chicken and deer—North Dakota (*83*); from pig—Iowa (*241*). Virus isolation from *Aedes dorsalis*—California (*147*); from *Culex tarsalis* (*151*).	Antibodies detected in blood of man—Minnesota (*92*); Colorado (*79*). Virus isolation from *Culex tarsalis* (*151*). Virus transmission in *Dermacentor variabilis* (*47*).	Epizootic in Texas (*348*).	

Table 7. **Historical highlights with special reference to WEE, SLE, and EEE—Continued**

Year	Western equine encephalomyelitis	St. Louis encephalitis	Eastern equine encephalomyelitis	Miscellaneous encephalitides
1942	Virus isolation from *Culex pipiens, C. tarsalis, Culiseta inornata, Anopheles freeborni*—Washington (*137* and *156*).	Virus isolation from *Culex pipiens, C. tarsalis*—Washington (*137* and *156*). SLE in Airzona (*243*); in Africa (*374*). Transmission by *Culex pipiens* (*314*).		
1943	Virus isolation from hog—Iowa (*241*); from human spinal fluid (*128*). Virus isolation from *Culex stigmatasoma*—California (*147*).	------------------------	Epizootic in Michigan (*187*).	California strain isolated from *Aedes dorsalis* (*147*). Human infection with VEE (*227*).
1944	Virus isolation from *Culex restuans*—Canada (*281*); from *Dermanyssus gallinae*—Texas (*381*).	Virus isolation from *Aedes dorsalis*—California (*149*); from *Dermanyssus gallinae*—Missouri (*367*).	------------------------	VEE transmitted by *Mansonia titillans* (*116*). VEE in Trinidad (*215*). Neurotropic virus in Colombia mosquitoes (*328*).
1946	------------------------	Virus isolation from human blood—California. (*51*)	------------------------	Japanese B transmitted by United States mosquitoes (*307*).
1947	Virus isolation from *Liponyssus bursa*—Texas (*382*).	------------------------	Epizootic in Texas and Louisiana (*360*); in Panama (*380*). Virus isolation from chicken mites and lice—Tennessee (*187*). EEE in Philippines (*231*).	
1948	Outbreak in Garden City, Kans. (*79*). Virus isolation from *Liponyssus sylvarium*.)	Virus isolation from *Liponyssus sylvarium*—California (*162*).	Virus isolation from *Mansonia perturbans*—Georgia (*188*). Epizootic in Dominician Republic (*94*).	
1949	Outbreak in North Dakota and Colorado (*79*).	------------------------	Epizootic in Louisiana and Arkansas (*206*).	
1950	Virus isolation from *Dermanyssus americanus* — Colorado (*254*); from redwinged blackbird and magpie—Colorado (*379*).	------------------------	Virus isolation from *Culiseta melanura*—Louisiana (*73*). Virus isolation from purple grackle — Louisiana (*207*).	
1951	Outbreak in North Dakota (*424*).	------------------------	Outbreak in pheasants—Connecticut — Beadle, 1952. See footnote 6, page 4.	
1951	First virus isolation from wild birds (*207*).	------------------------	First virus isolation from wild birds (*207*).	

dead horses and injected into guinea pigs, failed to produce viremia (*100*). EEE virus was not isolated from *Capillaria* sp. of sick chickens (*39*).

Transmission of neurotropic encephalitides by means other than arthropod bite has attracted little attention. Wood rats and cotton rats are susceptible to subcutaneous injections of WEE and EEE and to the contamination of abraded skin (*124*). The feces of *T. sanguisuga*, when injected into guinea pigs, produce infections of WEE (*124*), and it is possible that local contamination of the bite by reduviid feces is the important factor here, as in Chagas' disease. Horses have been infected with EEE by lingual abrasions and injections (*345*). The ability of mice to become infected with SLE by eating an infected member of the colony has been presented as an unusual mode of transmission among rodents (*165*), but the virus apparently is not passed from one mouse to another by pure contact. Horses do not transmit WEE by contacting presumably contaminated feeding racks, food buckets, or other such objects (*413*). However, in a single test, Remlinger and Bailly (*319*) were able to transmit WEE (Argentine strain) to a donkey, through virus-contaminated drinking water. The donkey became paralyzed. There seems to be no published laboratory study on the use of atomized virus suspensions as a mode of infection via the respiratory tract nor on any method which simulates the carrying of these viruses by dusts.

The role of winds in the spread of these diseases has been pondered (*95, 110, 116, 168, 297*), especially with respect to the St. Louis epidemic. Windblown dust as a vector was viewed with suspicion by Udall (*409*). Dust as a vector, in addition to a known arthropod vector, has become important in the laboratory transmission of Venezuelan equine encephalomyelitis. It has been proposed that careful study be made of human laboratory infections started via the respiratory intake of fine materials drifting up out of the litter beds of infected animals (*227*). Lymphocytic choriomeningitis, which can be experimentally transmitted via arthropods (*80, 258, 352*), is also thought to be dustborne (*30*).

Concerning the transmission cycle of equine encephalomyelitis, about which all epidemiological factors and especially potential disease control revolve, Hammon has described a striking series of cycles based on the gradual historical development in these diseases (*141*), starting with the simplest known cycle (horse-to-mosquito) and ending with the horse and man delimited from the main stream of events and acting as fortuitous recipients of virus from *Aedes* sp., with the horse also possibly being affected by assassin bugs. Since 1948, concepts on the infective chain of WEE have changed, with aedines and reduviids assuming a less important status. In the main stream of events, domestic and wild birds (*156*; see also *161* on Japanese B virus in bird blood), especially young birds, are thought to be important, with these animals being parasitized by both virus-laden *C. tarsalis* and mites (of which the latter probably transmit virus transovarially). In his paper (*156*), Hammon presents a clear-cut statement of the present status of the implications of arthropods as vectors. He has also presented an intriguing stem-virus theory which postulates the mutation of already existing viruses (*142*).

There is interesting deduction in the unpublished report of Lumsden (*229*) on the epidemiological factors of the St. Louis epidemic of encephalitis. In this report, it was indicated by a series of arguments that *Culex* sp. were the most probable vectors of the disease since large numbers of these mosquitoes were breeding in the stagnant, polluted streams in particular areas of the city with the greatest case incidence. He commented: "The abundance of sewage-bred mosquitoes (*Culex pipiens* and *C. quinquefasciatus*) appeared to be the only consistent factor of difference between communities which were heavily affected by the disease in this epidemic and those that were not affected or only slightly affected." Lumsden noted that not a case of SLE occurred among 4,000 inmates of a well-screened institution who were not allowed the privilege of a walk at dusk, but that cases did occur in poorly screened infirmaries. The official published report (*18*) on the epidemiology of this same epidemic generally ruled out mosquitoes in preference to contact transmission.

Immunity and Prevention

Immunity to viremia develops naturally, apparently from infections of whatever degree. In Japan most adults have endured mild infections of Japanese B encephalitis with resulting immunity. Evidence of immunity to WEE, EEE, and SLE, through the use of the complement-fixation technique (*131*) or the neutralization test, has been commonly shown in great numbers of animals (table 1) and in man in many parts of North America. Development of artificial immunization of man is not practiced except in special circumstances (*331, 333*). The papers of Howitt and Van Herick (*176, 185, 186*) on immunological aspects of these viruses in horses are cited. Mice were partially protected from the intracerebral inoculation of WEE by the use of deep ether anesthesia following injection of virus, and central nervous system symptoms were said to be delayed (*386*).

Vaccines have been developed and used successfully in equines in endemic agricultural areas of the United States and Canada with few deaths and very little hypersensitivity. Immunity is said to last up to 13 months, but probably no longer (*340*), and vaccine should be given 1 month before the time for seasonal outbreaks. The United States Army has consistently used WEE and EEE vaccines on all equines (*200*). Early in 1935, Traub and TenBroeck (*407*) built up immunity in test horses, apparently by massive doses of virus, to the point where they resisted intracerebral inoculations of virus. Specialists of the Bureau of Animal Industry have assisted veterinarians in the development and proper usage of these vaccines (*340*).

Vaccines for general human use are practical and are used extensively. They have been prepared as bivalent material for EEE and WEE (*37–38*) and are available for VEE.

Admonitions with respect to prevention and control of this group of diseases may be summarized as follows: Except for vaccines, farmers have been given no directions, in the literature reviewed, in the management of the equine disease except simple directions concerning the isolation of infected animals and the utility of insect control (*5*, *20*, *113*, *133*, *203*, *264*, *301*, *336*). Treatment has been supportive, based on symptomatic principles. Control of warm-blooded hosts has, in general, been viewed as impractical. Control of arthropod vectors or suspected vectors forms the basis of the only feasible preventive method (*133*). Recent thinking on this subject is outlined by Reeves.[13] Control of the production of arthropod vectors (especially mosquitoes) of encephalitis is gradually assuming a more important role in public health activities (*5*, *318*), especially with respect to the continued widespread use of irrigation water[14] and other phases of water resources development in the United States (*78*, *97*, *239*, *289*, *304*). A recent unusual form of mosquito control in the endemic focus—Yakima Valley of Washington—has been cited (*311*) and was based on the idea that very widespread use of organic agricultural insecticides has greatly reduced the general insect populations in that area.[15] Since certain species of mosquitoes in the United States (*A. dorsalis*, *Aedes nigromaculis*, *Culex pipiens molestus*, *Culex pipiens pipiens*, *C. quinquefasciatus*, *C. tarsalis*, *Culiseta incidens*, and *Culiseta inornata*) appear to be possible vectors of Japanese B encephalitis, some concern has been expressed as to the possible introduction of that dreaded virus disease into the country (*161*, *307*).

[13] Reeves, W. C.: The encephalitis problem in the United States. Am. J. Pub. Health 41: 678–686 (1951).

[14] Anonymous: Mosquito problems in irrigated areas and their prevention. Atlanta, Ga., U. S. Communicable Disease Center, 1951.

[15] Reeves, W. C.: Yakima, Washington, controls mosquitoes and flies at no cost—why can't we? Proc. and Papers 18th annual meeting, California Mosquito Control Association, pp. 13–15, 1950.

Related Diseases

There are a number of related or similar diseases which may be compared to the arthropod-borne virus encephalitides under survey. The viruses of vesicular stomatitis (*81*) and equine encephalomyelitis may be related but are not identical as ascertained by cross-immunity tests (*394*). Forms of canine (*120*, *214*), avian (*210*), moose (*205*), and mouse (*243*, *401*) encephalitis occur. Mammals, birds, and arthropods have been studied as possible hosts for the related disease, poliomyelitis (*135*). The syndrome of epidemic tremor in chickens definitely is of an encephalitic nature (*191*, *410*); moreover, the pheasants from which EEE was isolated (*411*) were thought to be ill with such a disease, which is fairly widespread in the United States. During the period from 1938 through 1946, there occurred 13 outbreaks in New Jersey pheasants described as EEE (*40*). There is a decidedly neurotropic form of Rift

Valley fever (*86, 208*). In horses, veterinarians for some time have been concerned with the proper etiology of moldy corn disease (*343*) and forage poisoning, so often associated with equine sleeping sickness (*409*). The "second epizootic" of Cox and Philip (*82*) is cited as a form of equine encephalomyelitis which occurred in the Bitterroot Valley, Mont., epidemic. Marsh (*236*) also studied these horse losses of unknown etiology following a program of immunization. Shahan et al. (*350*) reviewed these and other similar unexplained outbreaks.

Summary

This paper presents a review and selected bibliography of the literature of the epidemic encephalitides in the Americas, with reference particularly to entomological aspects of these diseases for the attention and general information of investigators and gives certain tabular summations on the arthropod-vector problem, essentially encompassing historical developments in the first 20 years of the recognition of these neurotropic diseases.

Bibliographic Symbols

The bibliography which follows is organized so that by the use of symbols W, S, E, V, or G following the citation, references can be selected pertaining to certain of these diseases. The symbol "W" means western, "E," eastern, and "V," Venezuelan equine encephalomyelitis; "S," St. Louis encephalitis. Symbol G means that more than one of the three viruses (W, S, or E) are mentioned or that generalized statements are made. The abbreviations in the references follow the form used by the *Quarterly Cumulative Index Medicus.* Every reference has been utilized one or more times in the text or in the tables. A few references (selected after the closing date of the manuscript) are appended as footnotes to the text. The literature essentially covers the period from the early work of Meyer, about 1930, through 1951.

Bibliography

(*1*) Anonymous: A new virus disease of horses. North Am. Vet. 22: 651–652 (1941). (W)

(*2*) Anonymous: Bibliography on eastern, western, and Venezuelan equine encephalomyelitis. Frederick, Md., Camp Detrick Technical Library, 1951, 54 pp. Mimeographed. (G)

(*3*) Anonymous: Como fue comatido y exterminada el brote de encefalomielitis equina en la Republica Dominicana. Agricultura 70: 1–41 (July–September 1949). (E)

(*4*) Anonymous: Encephalitis investigations. CDC Bull., April–June 1949, p. 47. (G)

(*5*) Anonymous: Encephalitis in the California and Pacific area. California Med. 65: 294–295 (1946). (G)

(*6*) Anonymous: Encephalitis investigations. Virus and rickettsia studies. CDC Bull., October–December, 1949, p. 53. (G)

(*7*) Anonymous: Encephalitis, infectious. *In* The control of communicable diseases in man. Ed. 7. New York, American Public Health Association, 1950, pp. 45–47. (G)

(*8*) Anonymous: Encephalitis investigations. CDC Bull. 9: 15–17 (December 1950). (W)

(*9*) Anonymous: Encephalitis investigations. CDC Bull. 9: 11–12 (March 1950). (G)

(*10*) Anonymous: Epidemic encephalitis in retrospect. New England J. Med. 240: 983–984 (1949). (G)

(*11*) Anonymous: Equine encephalomyelitis and human encephalitis: Three neurotropic viruses now known to affect both humans and animals. North Am. Vet. 22: 665–669 (1941). (S)

(*12*) Anonymous: Equine encephalomyelitis. J. Am. Vet. M. A. 113: 464 (1948). (G)

(*13*) Anonymous: Find encephalitis virus in birds, insects. J. A. M. A. 147: 272 (1951). (W)

(*14*) Anonymous: Infectious encephalitis. Pub. Health Rep. 50: 542–545 (1935). (G)

(*15*) Anonymous: Infectious equine encephalomyelitis in the United States in 1939. Pub. Health Rep. 55: 601–602 (1940). (G)

(*16*) Anonymous: Murray Valley encephalitis. M. J. Australia 1: 526–527 (1951).

(*17*) Anonymous: New developments in knowledge of encephalitis. J. A. M. A. 117: 1361–1363 (1941). (G)

(*18*) Anonymous: Report on St. Louis outbreak of encephalitis. Pub. Health Bull. No. 214, 1935, 117 pp. (S)

(*19*) Anonymous: Something about "sleeping sickness." Illinois Health Messenger 13: 111–112 (1941). (G)

(*20*) Anonymous: Suggestions for the prevention and control of equine encephalomyelitis. Extension Service in Agriculture. Home Economics Bull. No. AH 784. University of Illinois, Urbana, 1938, 4 pp. (G)

(*21*) Anonymous: The Trinidad outbreak of equine encephalomyelitis. J. A. M. A. 124: 1204 (1944). (G)

(*22*) Anonymous: Virus and rickettsia studies. CDC Bull., October–December, 1949, pp. 53–54. (G)

(*23*) Anonymous: Virus and rickettsia studies. CDC Bull. 9: 16 (1950). (W)

(*24*) Anonymous: Virus and rickettsia studies. CDC Bull. 9: 20–21 (1950). (G)

(*25*) Anonymous: Virus and rickettsia studies. CDC Bull. 9: 24–25 (1950). (G)

(*26*) Anonymous: Virus and rickettsia studies. CDC Bull. 9: 28 (1950). (G)

(*27*) Abdel-Malek, A.: The biology of *Aedes trivittatus.* J. Econ. Entom. 41: 951–954 (1948). (G)

(*28*) Adams, R. D., and Weinstein, L.: Clinical and pathological aspects of encephalitis. New England J. Med. 239: 865–876 (1948). (G)

(*29*) Aitken, T. H. G.: The relationship of the distribution of cases of equine encephalomyelitis (human and equine) and mosquitoes in California. Proc. Conference California Mosquito Control Association, 11th annual, 1940, pp. 8–15. (W)

(*30*) Armstrong, C.: Studies on choriomeningitis and poliomyelitis. Harvey Lect. 36: 39–65 (1940–1941).

(*31*) Armstrong, C., and Lillie, R. D.: Experimental lymphocytic choriomeningitis of monkeys and mice produced by a virus encountered in studies of the 1933 St. Louis encephalitis epidemic. Pub. Health Rep. 49: 1019–1027 (1934). (S)

(*32*) Ayres, J. C., and Feemster, R. F.: Public-health aspects of the virus encephalitides. New England J. Med. 240: 966–975 (1949). (G)

(*33*) Ayres, J. C., and Feemster, R. F.: The sequelae of eastern equine encephalomyelitis. New England J. Med. 240: 960–962 (1949). (E)

(*34*) Baker, A. B.: The non-suppurative forms of encephalitis. Arch. Path. 19: 213–232 (1935). (G)

(*35*) Bang, F. B., and Reeves, W. C.: Mosquitoes and encephalitis in the Yakima Valley, Washington. III. Feeding habits of *Culex tarsalis* Coquillet, a mosquito host of the viruses of western equine and St. Louis encephalitis. J. Infect. Dis. 70: 273–274 (1942). (S)

(*36*) Bates, M.: Natural history of mosquitoes. New York, Macmillan & Co., 1949, 379 pp. (G)

(*37*) Beard, J. W., Beard, D., and Finkelstein, H.: Human vaccination against equine encephalomyelitis virus with formalized chick embryo vaccine. Science 90: 215–216 (1939). (G)

(*38*) Beard, J. W., Beard, D., and Finkelstein, H.: Vaccination of man against the virus of equine encephalomyelitis (eastern and western strains). J. Immunol. 38: 117–136 (1940). (G)

(39) Beaudette, F. R.: Equine encephalomyelitis in avian hosts. Proc. U. S. Livestock Sanitary Association, 43d annual meeting, 1939, pp. 185–203. (E)

(40) Beaudette, F. R., and Black, J. J.: Equine encephalomyelitis in New Jersey pheasants in 1945 and 1946. J. Am. Vet. M. A. 112: 140–147 (1948). (E)

(41) Beaudette, F. R., and Hudson, C. B.: Additional outbreaks of equine encephalomyelitis in New Jersey pheasants. J. Am. Vet. M. A. 107: 384–386 (1945). (E)

(42) Beck, C. E., and Wyckoff, R. W. G.: Venezuelan equine encephalomyelitis. Science 88: 530 (1938). (V)

(43) Bender, W. L.: Epidemic encephalitis: Present status of etiology and treatment. California & West. Med. 27: 625–629 (1927). (W)

(44) Bishopp, F. C.: Mosquito transmission of encephalomyelitis, or brain fever, of horses. J. Washington Acad. Sc. 29: 495–501 (1939). (G)

(45) Bishopp, F. C., and Smith, C.: Résumé of mosquito work throughout the world in 1933: Equine encephalomyelitis and herpes-encephalitis. Proc. the New Jersey Mosquito Extermination Association, 21st annual meeting, 1934, pp. 46–47. (G)

(46) Black, W. C., Absher, W. K., and McDonald, J. G.: Epidemic encephalitis in Colorado. Rocky Mountain M. J. 42: 354–359 (1945). (S)

(47) Blattner, R. J., and Heys, F. M.: Experimental transmission of St. Louis encephalitis to white Swiss mice by *Dermacentor variabilis*. Proc. Soc. Exper. Biol. Med. 48: 707–710 (1941). (S)

(48) Blattner, R. J., and Heys, F. M.: Blood-sucking vectors of encephalitis: Experimental transmission of St. Louis encephalitis to white Swiss mice by the American dog tick, *Dermacentor variabilis* Say. J. Pediat. 23: 371–375 (1943). (S)

(49) Blattner, R. J., and Heys, F. M.: Blood-sucking vectors of encephalitis: Experimental transmission of St. Louis encephalitis (Hubbard strain) to white Swiss mice by American dog tick, *Dermacentor variabilis* Say. J. Exper. Med. 79: 439–454 (1944). (S)

(50) Blattner, R. J., and Heys, F. M.: St. Louis encephalitis: Occurrence in children in the St. Louis area during non-epidemic years 1939–44 J. A. M. A. 129: 854–857 (1945). (S)

(51) Blattner, R. J., and Heys, F. M.: Isolation of St. Louis encephalitis virus from the peripheral blood of a human subject. J. Pediat. 28: 401–406 (1946). (S)

(52) Bohls, S. W., and Irons, J. V.: Laboratory observations on virus encephalitis. Texas State J. Med. 38: 260–264 (1942). (E)

(53) Bozalis, G. S., and Jones, A. B.: Epidemic encephalitis, St. Louis type: Survey of outbreak, summer and fall of 1937. J. Oklahoma M. A. 31: 164–171 (1938). (S)

(54) Brandly, C. A.: Birds as possible reservoirs of equine encephalomyelitis infection. North Am. Veterinarian 21: 95–96 (1940). (G)

(55) Bredeck, J. F.: The story of the epidemic of encephalitis in St. Louis. Am. J. Pub. Health 23: 1135–1140 (1933). (S)

(56) Breslich, P. J., Rowe, P. H., and Lehman, W. L.: Epidemic encephalitis in North Dakota. J. A. M. A. 113: 1722–1724 (1939). (W, S)

(57) Brodie, M.: Absence of antiviral substance in normal adults for the virus of the St. Louis encephalitis epidemic. Proc. Soc. Exper. Biol. & Med. 31: 1227–1229 (1934). (S)

(58) Brodie, M.: Route of transmission of St. Louis encephalitis virus in mice. Proc. Soc. Exper. Biol. & Med. 32: 1647–1649 (1934). (S)

(59) Brookman, B.: Attempts at insectary rearing of *Culex tarsalis*. Proc. Conference California Mosquito Control Association, 16th annual, 1948, p. 79. (G)

(60) Brookman, B.: Relation of arthropods to the epidemiology of virus encephalitis in Kern County, California. CDC Bull. 9: 7–9 (1950). (W)

(61) Broun, G. O., Muether, J., and Casey, A. E.: The virus of the 1937 outbreak of encephalitis in St. Louis. Week. Bull. St. Louis M. Soc. 32: 40 (1937). (S)

(62) Brown, G. C.: Studies on equine encephalomyelitis in Michigan. J. Infect. Dis. 81: 48–54 (1947) (W, E)

(63) Brueckner, A. L., Poelma, L. J., Everson, C. L., and Reed, R. C.: Equine encephalomyelitis. Maryland Agr. Exper. Sta. Bull. No. 369, 1934, pp. 137–146. (G)

(64) Bucher, J. C., Boshell-Manrique, J., Roca-Garcia, M., and Osorno-Mesa, E.: Epidemiology of jungle yellow fever in Eastern Colombia. Am. J. Hyg. 39: 16–51 (1944).

(65) Burns, K. F., and Matumoto, M.: Japanese equine encephalomyelitis: Review of literature. J. Am. Vet. M. A. 115: 112–115 (1949).

(66) Buss, W. C., and Howitt, B. F.: Human equine encephalomyelitis in Kern County, California, 1938, 1939, and 1940. Am. J. Pub. Health 31: 935–944 (1941). (W)

(67) Cameron, G. D. W.: Western equine encephalitis. Canad. J. Pub. Health 33: 383–387 (1942). (W)

(68) Casals, J., Curnen, E. C., and Thomas, L.: Venezuelan equine encephalomyelitis in man (laboratory workers). J. Exper. Med. 77: 521–530 (1943).

(69) Casey, A. E.: The geographic pathology of St. Louis encephalitis. South. M. J. 35: 869–880 (1942). (S)

(70) Casey, A. E., and Broun, G. O.: Epidemiology of St. Louis encephalitis. Science 88: 450–451 (1938). (S)

(71) Chamberlain, R. W., and Sikes, R. K.: A safe method of handling mosquitoes for virus transmission experiments. J. Parasitol. 36: 499–500 (1950). (G)

(72) Chamberlain, R. W., and Sikes, R. K.: Laboratory rearing methods for three common species of bird mites. J. Parasitol. 36: 461–465 (1950). (G)

(73) Chamberlain, R. W., Rubin, H., Kissling, R. E., and Eidson, M. E.: Recovery of virus of eastern equine encephalomyelitis from a mosquito, *Culiseta melanura* (Coquillett). Proc. Soc. Exper. Biol. & Med. 77: 396–397 (1951). (E)

(74) Cheever, F. S.: A possible relationship between viruses of St. Louis encephalitis and epidemic kerato conjunctivitis. Proc. Soc. Exper. Biol. & Med. 77: 125–129 (1951). (S)

(75) Chumachov, M. P., and Seitlemok, N. A.: Tick-borne human encephalitis in the European part of U.S.S.R. and Siberia. Science 92: 263–264 (1940).

(76) Cleland, J. B., and Campbell, A. W.: Experimental investigation of the Australian epidemic of acute encephalomyelitis. J. Hyg. 18: 272 (1919).

(77) Cockburn, T. A., Price, E. R., and Rowe, J. A.: Virus encephalitis in the Missouri River Basin. CDC Bull. 9: 13–24 (February 1950). (W)

(78) Cockburn, T. A., Rowe, J. A., and Price, E. R.: Mosquito control indicated in Missouri Basin. Pub. Health Rep. 65: 1690 (1950). (G)

(79) Cockburn, T. A., Price, E. R., and Rowe, J. A.: Encephalitis in the Midwest. I. A review of the problem. J. Kansas M. Soc. ~~50~~ 52: 316–318 (1951). (W, S)

(80) Coggeshall, L. T.: The transmission of lymphocytic choriomeningitis by mosquitoes. Science 89: 515–516 (1939).

(81) Cox, H. R., and Fite, G. L.: Serological distinctions between the viruses of encephalitis in St. Louis, 1933, equine encephalomyelitis and vesicular stomatitis. Proc. Soc. Exper. Biol. & Med. 31: 499–500 (1934). (G)

(82) Cox, H. R., and Philip, C. B.: Observations incident to an outbreak of equine encephalomyelitis in the Bitterroot Valley of western Montana. J. Am. Vet. M. A. 93: 225–232 (1938). (W)

(83) Cox, H. R., Jellison, W. L., and Hughes, L. E.: Isolation of western equine encephalomyelitis virus from a naturally infected prairie chicken. Pub. Health Rep. 56: 1905–1906 (1941). (W)

(84) Cox, H. R., Philip, C. B., and Kilpatrick, J. W.: Susceptibility of horses to St. Louis encephalitis virus. Pub. Health Rep. 56: 1391–1392 (1941). (S)

(85) Cruchet, R., Moutier, F., and Calmette, A.: Quarante cas d'encephalomyelite subaigue. Bull. et mém. Soc. med. d. hop. de Paris 44: 1279 (1920). (G)

(86) Daubney, R., Hudson, J. R., and Garnham, P. C.: Enzootic hepatitis or Rift Valley fever. J. Path. & Bact. 34: 545–579 (1931).

(87) Davis, W. A.: A study of birds and mosquitoes as hosts for the virus of eastern equine encephalomyelitis. Am. J. Hyg. 32: 45–59 (1940). (E)

(88) Day, M. F.: Report on mosquitoes collected in St. Louis County. Tr. St. Louis Acad. Sc. 31: 29–45 (1943). (S)

(89) Dingle, J. H.: The encephalitides of virus etiology. New England J. Med. 225: 1014–1022 (1941). (G)

(90) Donovan, C. R., and Bowman, M.: Some epidemiological features of poliomyelitis and encephalitis, Manitoba, 1941. Canad. J. Pub. Health 33: 246–257 (1942). (W)

(91) Durand, P., Girond, P., Larrive, E., and Mestrallet, A.: Transmission experimentale a l'homme de la maladie des porchers. Compt. rend. Acad. d. sc., 203: 830–832 (1936). (G)

(92) Eklund, C. M.: Human encephalitis of the western equine type in Minnesota in 1941: Clinical and epidemiological study of serologically positive cases. Am. J. Hyg. 43: 171–193 (1946). (S, W)

(93) Eklund, C. M., and Blumstein, A.: The relation of human encephalitis to encephalomyelitis in horses. J. A. M. A. 111: 1734–1735 (1938). (W)

(94) Eklund, C. M., Brennan, J. M., and Bell, J. F.: Final report to the Pan American Sanitary Bureau regarding the 1948–49 outbreak of eastern equine encephalomyelitis in the Dominican Republic. Bol. Ofic. san. panam. 29: 493–508 (1950). (E)

(95) Eklund, C. M., Bell, J. F., and Brennan, J. M.: Antibody survey following an outbreak of human and equine disease in the Dominican Republic, caused by the eastern strain of equine encephalomyelitis virus. Am. J. Trop. Med. 31: 312–328 (1951). (E)

(96) Farber, S., Hill, A., Connerley, M. L., and Dingle, J. H.: Encephalitis in infants and children. J. A. M. A. 114: 1725–1731 (1940). (E)

(97) Farquharson, J.: Infectious equine encephalomyelitis. J. Am. Vet. M. A. 94: 459–465 (1939). (W)

(98) Feemster, R. F.: Outbreak of encephalitis in man due to the eastern virus of equine encephalomyelitis. Am. J. Pub. Health 28: 1403–1410 (1938). (E)

(99) Feemster, R. F., and Getting, V. A.: Distribution of the vectors of equine encephalomyelitis in Massachusetts. Am. J. Pub. Health 31: 791–802 (1941). (E)

(100) Foster, A. O., and Shahan, M. S.: Unsuccessful attempts to transmit encephalomyelitis from horses to guinea pigs by endoparasites. Proc. Helminth. Soc., Wash. 9: 20–21 (1942). (G)

(101) Fothergill, L. D.: A tentative classification of virus diseases of the central nervous system, with a consideration of certain epidemic types of encephalitis. *In* Harvard University sym-

posium on virus and rickettsial diseases. Cambridge, Mass., Harvard University Press, 1940, pp. 617–641. (G)

(*102*) Fothergill, L. D., and Dingle, J. A.: A fatal disease of pigeons caused by the virus of the eastern variety of equine encephalomyelitis. Science 88: 549–550 (1938). (E)

(*103*) Fothergill, L. D., Dingle, J., Farber, S., and Connerley, M. L.: Human encephalitis caused by the virus of the eastern variety of equine encephalomyelitis. New England J. Med. 219: 411 (1938). (E)

(*104*) Fothergill, L. D., Holden, M., and Wyckoff, R. W. G.: Western equine encephalomyelitis in a laboratory worker. J. A. M. A. 113: 206–207 (1939). (W)

(*105*) Fulton, J. D., Gruetter, J. E., Muether, R. O., Hanss, E. B., and Broun, G. O.: Observations concerning *Culex pipiens* as a possible carrier of St. Louis encephalitis. Proc. Soc. Exper. Biol. & Med. 44: 255–256 (1940). (S)

(*106*) Fulton, J. S.: A report of two outbreaks of equine encephalomyelitis in Saskatchewan. Canad. J. Comp. Med. 2: 39–46 (1938). (W)

(*107*) Fulton, J. S.: Relation of equine encephalomyelitis to the epidemic of human encephalitis in Saskatchewan in 1938. Canad. J. Pub. Health 32: 6–12 (1941). (W)

(*108*) Geiger, J. C., Dickson, E. C., and Meyer, K. F.: The epidemiology of botulism. Pub. Health Bull. No. 127, 1922, 119 pp. (G)

(*109*) Geo. Wm. Hooper Foundation: A final summary of flight range studies on *Culex tarsalis* and notes on wild bird malaria in Kern County. Proc. California Mosquito Control Association, 16th annual conference, 1948, pp. 58–59. (G)

(*110*) Getting, V. A.: Equine encephalomyelitis in Massachusetts: An analysis of the 1938 outbreak, a follow-up of cases and a report of a mosquito survey. New England J. Med. 224: 999–1006 (1941). (E)

(*111*) Giltner, L. T., and Shahan, M. S.: Transmission of infectious equine encephalomyelitis in mammals and birds. Science 78: 63–64 (1933). (W)

(*112*) Giltner, L. T., and Shahan, M. S.: The 1933 outbreak of infectious equine encephalomyelitis in the eastern States. North Am. Vet. 14: 25–27 (1933). (E)

(*113*) Giltner, L. T., and Shahan, M. A.: Horse disease, known as encephalomyelitis, yielding to research. *In* Yearbook of Agriculture. Washington, D. C., U. S. Government Printing Office, 1935, pp. 233–236. (G)

(*114*) Giltner, L. T., and Shahan, M. S.: The present status of infectious equine encephalomyelitis in the U. S. J. Am. Vet. M. A. 88: 363–374 (1936). (G)

(*115*) Giltner, L. T., and Shahan, M. S.: Equine encephalomyelitis. Yearbook of Agriculture. Washington, D. C., U. S. Government Printing Office, 1942, pp. 375–391. (G)

(*116*) Gilyard, R. D.: Mosquito transmission of Venezuelan virus of equine encephalomyelitis in Trinidad. Bull. U. S. Army M. Dept. No. 75, 1944, pp. 96–107. (G)

(*117*) Godelfer, L.: Equine encephalomyelitis. Am. J. M. Tech. 15: 117–126 (1949). (E)

(*118*) Gjullin, C. M., Yates, W. W., and Stage, H. H.: Studies on *Aedes vexans* (Meig.) and *Aedes sticticus* (Meig.), flood-water mosquitoes, in the Lower Columbia River Valley. Ann. Ent. Soc. Am. 43: 262–275 (1950). (G)

(*119*) Gold, H., and Hampil, B.: Equine encephalomyelitis in a laboratory technician with recovery. Ann. Int. Med. 16: 556–569 (1942). (W)

(*120*) Green R. G.: The problem of encephalitis in dogs. J. Am. Vet. M. A. 99: 45–48 (1941).

(*121*) Greutter, J., and Brown, G. O.: Further studies on the virus of the 1937 outbreak of encephalitis in St. Louis. Proc. Soc. Exper. Biol. & Med. 37: 284–285 (1937). (S)

(*122*) Greutter, J. E., Fulton, J. D., Muether, R. O., Hanss, E. V., and Broun, G. O.: Suspectibility of field mice and meadow mice to St. Louis encephalitis. Proc. Soc. Exper. Biol. & Med. 44: 253–254 (1940). (S)

(*123*) Grundmann, A. W.: Studies on the biology of *Triatoma sanguisuga* (Le Conte) in Kansas (Reduviidae, Hemiptera) J. Kansas Entom. Soc. 20: 77–85 (1947).

(*124*) Grundmann, A. W. Kitselman, C. M., Roderick, L. M., and Smith, R. C.: Studies on the transmission of the western virus of equine encephalomyelitis by the American dog tick, *Dermacentor variabilis* Say and by *Triatoma sanguisuga* (Le Conte). J. Infect. Dis. 72: 163–171 (1943). (W, E)

(*125*) Gwatkin, R.: Further examinations of small mammals and birds in a search for carriers of equine encephalomyelitis virus. Canad. J. Comp. Med. 5: 113–116 (1941). (G)

(*126*) Gwatkin, R., and Moore, T.: Indication of the presence of the virus of equine encephalomyelitis in the brain of ground squirrel. Canad. J. Comp. Med. 4: 78–82 (1940). (W)

(*127*) Gwatkin, R., and Moynihan, I. W.: Search for sources and carriers of equine encephalomyelitis virus. Canad. J. Res. Sec. D 20: 321–337 (1942). (W)

(*128*) Gwatkin, R., and Moynihan, I. W.: Recovery of equine encephalomyelitis virus (western type) from human spinal fluid in Alberta. Canad. J. Pub. Health 34: 42–43 (1943). (W)

(*129*) Hammon, W. M.: Encephalitis in the Yakima Valley, Washington: Mixed St. Louis and western equine types. J. A. M. A. 117: 161–167 (1941). (S)

(*130*) Hammon, W. M.: Animal reservoirs, general epidemiological summary, and possible control measures for the American summer encephalitides. Proc. California Mosquito Control Association, 12th annual conference, 1941, pp. 37–51. (G)

(*131*) Hammon, W. M.: Encephalitis (arthropod-borne virus encephalitides and lymphocytic choriomeningitis). *In* American Public Health Association: Diagnostic procedures for virus and rickettsial diseases. New York, American Public Health Association, 1948, pp. 187–217.

(*132*) Hammon, W. M., and Reeves, W. C.: *Culex tarsalis* Coq. a proven vector of St. Louis encephalitis. Proc. Soc. Exper. Biol. & Med. 51: 142–143 (1942). (S)

(*133*) Hammon, W. M.: Suggestions for the possible control of the American summer encephalitides. J. A. M. A. 118: 66–68 (1942). (G)

(*134*) Hammon, W. M.: The epidemic encephalitides of North America. M. Clinics North America 27: 632–650 (1943). (S)

(*135*) Hammon, W. M.: Studies on possible mammalian, avian and arthropod hosts of poliomyelitis. J. Bact. 45: 89–90 (1943). (G)

(*136*) Hammon, W. M.: Encephalitis: Eastern and western equine and St. Louis types, as observed in 1941 in Washington, Arizona, New Mexico, and Texas. J. A. M. A. 121: 560–566 (1943). (S, E, W.)

(*137*) Hammon, W. M., Reeves, W. C., Benner, S. F., and Brookman, B.: Human encephalitis in the Yakima Valley, Washington, 1942, with 49 virus isolations (western equine and St. Louis types) from mosquitoes. J. A. M. A. 128: 1133–1139 (1945). (W, S)

(*138*) Hammon, W. M.: The encephalitides of virus origin with special reference to those of North America. Clinics 4: 485–503 (1945). (G)

(*139*) Hammon, W. M., Reeves, W. C., and Izumi, E. M.: St. Louis encephalitis virus in the blood of experimentally inoculated fowls and mammals. J. Exper. Med. 83: 175–183 (1946). (S)

(*140*) Hammon, W. M.: Encephalitis. Radio broadcast for Lederle, Inc., March 19, 1946. (G)

(*141*) Hammon, W. M.: Advances in knowledge of the vectors and arthropod reservoirs of certain of the encephalitides. Proc. California Mosquito Control Association, 16th annual conference, 1948, pp. 4–6. (G)

(*142*) Hammon, W. M.: The arthropod-borne virus encephalitides. Am. J. Trop. Med. 28: 515–525 (1948). (G)

(*143*) Hammon, W. M.: Encefalitis por virus transmitidas por arthropodos. Medicina, Mex. 30: 240–242 (1950). (G)

(*144*) Hammon, W. M., and Howitt, B. F.: Epidemiological aspects of encephalitis in the Yakima Valley: Mixed St. Louis and western equine types. Am. J. Hyg. 35: 163–185 (1942). (S)

(*145*) Hammon, W. M., and Reeves, W. C.: Laboratory transmission of western equine encephalomyelitis virus by mosquitoes of the genera *Culex* and *Culiseta*. J. Exper. Med. 78: 425–434 (1943). (W)

(*146*) Hammon, W. M., and Reeves, W. C.: Laboratory transmission of St. Louis encephalitis virus by three genera of mosquitoes. J. Exper. Med. 78: 241–253 (1943). (S)

(*147*) Hammon, W. M., and Reeves, W. C.: Recent advances in the epidemiology of the arthropod-borne virus encephalitides including certain exotic types. Am. J. Pub. Health 35: 994–1004 (1945). (S)

(*148*) Hammon, W. M., and Reeves, W. C.: Western equine encephalomyelitis virus in the blood of experimentally inoculated chickens. J. Exper. Med. 83: 163–173 (1946). (W)

(*149*) Hammon, W. M., and Reeves, W. C.: Interepidemic studies on arthropod-borne virus encephalitides and poliomyelitis in Kern County, California, and the Yakima Valley, Washington, 1944. Am. J. Hyg. 46: 326–335 (1947). (S, W)

(*150*) Hammon, W. M., Gray, J. A., Jr., Evans, F. C., and Izumi, E. M.: Western equine and St. Louis encephalitis antibodies in the sera of mammals and birds from an endemic area. Science 94: 305–307 (1941). (S, W)

(*151*) Hammon, W. M., Reeves, W. C., Brookman, B., Izumi, E. M., and Gjullin, C.: Isolation of the viruses of western equine and St. Louis encephalitis from *Culex tarsalis* mosquitoes. Science 94: 328–330 (1941). (W, S)

(*152*) Hammon, W. M., Reeves, W. C., Brookman, B., and Gjullin, C. M.: Mosquitoes and encephalitis in the Yakima Valley, Washington: V. Summary of the case against *Culex tarsalis* Coquillett as a vector of the St. Louis and western equine viruses. J. Infect. Dis. 70: 278–283 (1942). (S)

(*153*) Hammon, W. M., Carle, B. N., and Izumi, E. M.: Infection of horses with St. Louis encephalitis virus, experimental and natural. Proc. Soc. Exper. Biol. & Med. 49: 335–340 (1942). (S)

(*154*) Hammon, W. M., Lundy, H. W., Gray, J. A., Evans, F. C., Bang, F., and Izumi, E. M.: A large-scale serum-neutralization survey of certain vertebrates as part of an epidemiological study of encephalitis of the western equine and St. Louis types. J. Immunol. 44: 75–86 (1942). (S, W)

(*155*) Hammon, W. M., Reeves, W. C., and Izumi, E. M.: Mosquitoes and encephalitis in the Yakima Valley, Washington. II. Methods for collecting arthropods and for isolating western equine and St. Louis viruses. J. Infect. Dis. 70: 267–272 (1942). (W, S)

(*156*) Hammon, W. M., Reeves, W. C., Brookman, B., and Izumi, E. M.: Mosquitoes and encephalitis in the Yakima Valley, Washington: I.

Arthropods tested and recovery of western equine and St. Louis viruses from *Culex tarsalis* Coquillett. J. Infect. Dis. 70: 263–266 (1942). (S)

(*157*) Hammon, W. M., Reeves, W. C., and Gray, M.: Mosquito vectors and inapparent animal reservoirs of St. Louis and western equine encephalitis viruses. Am. J. Pub. Health 33: 201–207 (1943). (S)

(*158*) Hammon, W. M., Reeves, W. C., and Irons, J. V.: Survey of the arthropod-borne virus encephalitides in Texas with particular reference to the lower Rio Grande Valley in 1942. Texas Rep. Biol. & Med. 2: 366–375 (1944). (W)

(*159*) Hammon, W. M., Reeves, W. C., and Galindo, P.: Epidemiologic studies of encephalitis in the San Joaquin Valley of California, 1943, with the isolation of viruses from mosquitoes. Am. J. Hyg. 42: 299–306 (1945). (G)

(*160*) Hammon, W. M., Reeves, W. C., and Gray, M.: Epizoology of western equine type encephalomyelitis: Eastern Nebraska field survey of 1943 with isolation of the virus from mosquitoes. Am. J. Vet. Res. 6: 145–148 (1945). (W)

(*161*) Hammon, W. M., Reeves, W. C., and Burroughs, R.: Japanese B encephalitis virus in the blood of experimentally inoculated chickens. Proc. Soc. Exper. Biol. & Med. 61: 304–308 (1946). (J)

(*162*) Hammon, W. M., Reeves, W. C., Cunha, R., Espana, C., and Sather, G.: Isolation from wild bird mites (*Liponyssus sylviarum*) of a virus or mixture of viruses from which St. Louis and western equine encephalitis viruses have been obtained. Science 107: 92–93 (1948). (S)

(*163*) Hammon, W. M., Reeves, W. C., Burroughs, R. N., and Huelscamp, M.: Western equine encephalitis control studies in Kern County, Calif., 1945. II. An evaluation of the effectiveness of certain types of control including residual DDT on virus infection rates in *Culex* mosquitoes and in chickens. Am. J. Hyg. 47: 93–102 (1948). (W)

(*164*) Harford, C. G., and Bronfenbrenner, J.: Infection of mice by feeding with tissues containing the virus of St. Louis encephalitis. J. Infect. Dis. 70: 62–68 (1942). (S)

(*165*) Harford, C. G., Sulkin, S. E., and Bronfenbrenner, J.: Infection of mice by feeding of tissues containing the virus of St. Louis encephalitis. Proc. Soc. Exper. Biol. & Med. 41: 332–333 (1939). (S)

(*166*) Harford, C. G., Sulkin, S. E., and Bronfenbrenner, J.: Susceptibility of wild mice to the virus of St. Louis encephalitis. Proc. Soc. Exper. Biol. & Med. 41: 331–332 (1939). (S)

(*167*) Haring, C. M., Howarth, J. A., and Meyer, K. F.: An infectious brain disease of horses and mules (encephalomyelitis). California Agr. Exper. Sta. Circ. No. 322, 1931, pp. 1–14. (W)

(*168*) Haseman, L.: Possible insect carriers in the 1933 outbreak of encephalitis in Columbia, Mo. J. Econ. Entom. 29: 618–621 (1936). (S)

(*169*) Hauser, G. H.: Human equine encephalomyelitis, eastern type, in Louisiana. New Orleans M. & S. J. 100: 551–558 (1948). (E)

(*170*) Helwig, F. C.: Western equine encephalomyelitis following accidental inoculation with chick embryo virus. J. A. M. A. 115: 291–292 (1940). (W)

(*171*) Hempelmann, T. C.: The symptoms and diagnosis of encephalitis; 1933 St. Louis epidemic. J. A. M. A. 103: 733–735 (1934). (S)

(*172*) Herms, W. B., Wheeler, C. M., and Herms, H. P.: Attempts to transmit equine encephalomyelitis by means of blood-sucking insects, especially mosquitoes. J. Econ. Entom. 27: 987–998 (1934). (W)

(*173*) Holt, W. L., Jr.: Epidemic encephalitis: A follow-up study of two hundred and sixty cases. Arch. Neurol. & Psychiat. 38: 1135–1144 (1937).

(*174*) Howitt, B. F.: Equine encephalomyelitis. J. Infect. Dis. 51: 493–510 (1932). (W)

(*175*) Howitt, B. F.: Propagation of virus of equine encephalomyelitis after intranasal instillation in the guinea pig. Proc. Soc. Exper. Biol. & Med. 32: 58–60 (1934). (W)

(*176*) Howitt, B. F.: An immunological study in laboratory animals of 13 different strains of equine encephalomyelitis. J. Immunol. 29: 319–341 (1935). (G)

(*177*) Howitt, B. F.: The Moscow 2 strain of equine encephalomyelitic virus as compared with other strains of equine encephalitis viruses. J. Infect. Dis. 63: 269–286 (1938). (G)

(*178*) Howitt, B. F.: The recovery of the virus of equine encephalomyelitis from the brain of a child. Science 88: 455–456 (1938). (W)

(*179*) Howitt, B. F.: Viruses of equine and of St. Louis encephalitis in relationship to human infections in California, 1937–38. Am. J. Pub. Health 29: 1083–1097 (1939). (S, W)

(*180*) Howitt, B. F.: Recovery of the virus of equine encephalomyelitis (western type) from human blood serum. Science 89: 541–542 (1939). (W)

(*181*) Howitt, B. F.: Comparative susceptibility of wild and domestic birds and animals to the western virus of equine encephalomyelitis (Br. strain) in California. J. Infect. Dis. 67: 177–187 (1940). (W)

(*182*) Howitt, B. F.: Equine encephalomyelitis; Its relationship to man and animals in California. Proc. International Congress Microbiology, 3d, 1940, pp. 302–303. (G)

(*183*) Howitt, B. F.: Development of neutralizing antibodies to the viruses of equine encephalomyelitis (western strain) and St. Louis encephalitis in the blood and cerebrospinal fluid of man and animals, together with the recovery of St. Louis virus from the blood of monkeys. J. Immunol. 42: 117–131 (1941). (S)

(184) Howitt, B. F.: Human equine encephalomyelitis and St. Louis encephalitis in California, 1939–41. Am. J. Pub. Health 32: 503–515 (1942). (S)

(185) Howitt, B. F., and Van Herick, W.: Neutralizing antibodies against St. Louis and western equine encephalitis viruses in horses and fowl. Proc. Soc. Exper. Biol. & Med. 48: 247–250 (1941). (W, S)

(186) Howitt, B. F., and Van Herick, W.: Relationship of the St. Louis and the western equine encephalitis viruses to fowl and mammals in California. J. Infect. Dis. 71: 179–191 (1942). (W, S)

(187) Howitt, B. F., Dodge, H. R., Bishop, L. K., and Gorrie, R. H.: Virus of eastern equine encephalomyelitis isolated from chicken mites (*Dermanyssus gallinae*) and chicken lice (*Eomenacanthus stramineus*) Proc. Soc. Exper. Biol. & Med. 68: 622–625 (1948). (E)

(188) Howitt, B. F., Dodge, H. R., Bishop, L. K., and Gorrie, R. H.: Recovery of the virus of eastern equine encephalomyelitis from mosquitoes (*Mansonia perturbans*) collected in Georgia. Science 110: 141–142 (1949). (E)

(189) Jakmauh, P. J.: The relation of mosquitoes to equine encephalomyelitis in Massachusetts. Proc. New Jersey Mosquito Exterminators Association, 27th annual meeting 27: 12–18 (1940). (E)

(190) Jenkins, D. W.: Bionomics of *Culex tarsalis* in relation to western equine encephalomyelitis. Am. J. Trop. Med. 30: 909–916 (1950). (W)

(191) Jones, E. E.: An encephalomyelitis in the chicken. Science 76: 331–332 (1932). (G)

(192) Kawamura, R., Kodama, M., Ito, T., Yasaki, T., and Kobayakawa, Y.: Epidemic encephalitis in Japan: The causative agent compared with that in the St. Louis epidemic. Arch. Path. 22: 510–523 (1936). (S)

(193) Kelser, R. A.: Mosquitoes as vectors of the virus of equine encephalomyelitis. J. Am. Vet. M. A. 82: 767–771 (1933). (W)

(194) Kelser, R. A.: Personal communication to Dr. J. S. Simmons, 1935. Am. J. Trop. Med. 16: 289–302 (1935). (W)

(195) Kelser, R. A.: Transmission of the virus of equine encephalomyelitis by *Aedes taeniorhynchus*. Science 85: 178 (1937). (W)

(196) Kelser, R. A.: Equine encephalomyelitis in Panama. Army Vet. Bull. 31: 19–21 (1937). (E)

(197) Kelser, R. A.: Transmission of the virus of equine encephalomyelitis by *Aedes taeniorhynchus*. J. Am. Vet. M. A. 92: 195–203 (1938). (W)

(198) Kelser, R. A.: Mosquito transmission of equine encephalomyelitis. Proc. International Congress of Microbiology, 3d, 1940, p. 300. (W, E)

(199) Kelser, R. A.: Newer researches regarding tropical and subtropical diseases. Army Vet. Bull. 33: 339–354 (1939). (E)

(200) Kelser, R. A.: Prophylaxis in equine encephalomyelitis. Cornell Vet. 30: 350–358 (1940). (G)

(201) Kelser, R. A.: Equine encephalomyelitis. Army Vet. Bull. 34: 1–6 (1940). (G)

(202) Kelser, R. A.: Methods of exposing experimental animals to virus infection through mosquito vectors. Am. J. Pub. Health 30: 866–870 (1940). (G)

(203) Kelser, R. A.: Equine encephalomyelitis and its control. Proc. U. S. Livestock San. Association, 45th annual meeting, 1941, pp. 22–27. (G)

(204) King, L. S.: Studies on eastern equine encephalomyelitis. IV. Infection in the mouse with fresh and fixed virus. J. Exper. Med. 71: 95–106 (1940). (E)

(205) King, L. S.: Moose encephalitis. Am. J. Path. 15: 445–454 (1939). (G)

(206) Kissling, R. E., and Rubin, H.: Eastern equine encephalomyelitis. CDC Bull. 9: 2–5 (1950). (E)

(207) Kissling, R. E., Rubin, H., Chamberlain, R. W., and Eidson, M. E.: Recovery of virus of eastern equine encephalomyelitis from blood of a purple grackle. Proc. Soc. Exper. Biol. & Med. 77: 398–399 (1951). (E)

(208) Kitchen, S. F.: Laboratory infections with the virus of Rift Valley fever. Am. J. Trop. Med. 14: 547–564 (1934). (G)

(209) Kitselman, C. H., and Grundman, A. W.: Equine encephalomyelitis virus isolated from naturally infected *Triatoma sanguisuga* Le Conte. Kansas Agr. Exper. Sta. Tech. Bull. No. 50, 1940, pp. 1–15. (W)

(210) Kligler, I. J., and Olitsky, P. K.: Experiments on cultivation of virus of infectious avian encephalomyelitis. Proc. Soc. Exper. Biol. & Med. 43: 680–683 (1940).

(211) Knowlton, G. F.: Insect transmission of equine encephalomyelitis studies. Utah Agr. Exper. Sta. Bull. No. 250, 1934, p. 47. (W)

(212) Knowlton, G. F., and Rowe, J. A.: Preliminary studies of insect transmission of equine encephalomyelitis. Proc. Utah Acad. Sc. 11: 267–270 (1934). (W)

(213) Knowlton, G. F., and Rowe, J. A.: Handling mosquitoes on equine encephalomyelitis investigation. J. Econ. Entom. 28: 824–829 (1935). (W)

(214) Koprowski, H., Jervis, G. A., James, T. R., Burkhart, R. L., and Poppenseik, G. C.: A study of canine encephalitis. Am. J. Hyg. 51: 63–75 (1950). (G)

(215) Kubes, V.: Venezuelan-type equine encephalomyelitis virus in Trinidad. Science 99: 41–42 (1944). (V)

(216) Kubes, V., and Rios, F. A.: The causative agent of infectious equine encephalomyelitis in Venezuela. Science 90: 20–21 (1939). (V)

(217) Kudo, M., Uraguchi, K., Matsuda, S., and Hashimoto, H.: Experimental studies on encephalitis virus of the 1935 epidemic in Tokyo with special reference to its serological difference from the virus of St. Louis encephalitis. J. Immunol. 32: 129–135 (1937). (S)

(218) Laemmert, H. W., and Hughes, T. P.: The virus of Ilheus encephalitis: Isolation, serological specificity and transmission. J. Immunol. 55: 61–67 (1947). (G)

(219) Lambert, W. V., Speelman, S. R., and Osborn, E. B.: Differences in incidence of encephalomyelitis in horses. J. Heredity 30: 349–352 (1939). (G)

(220) Large, A.: Fatal epidemic among horses in the United States. The Veterinarian 40: 655–660, 727–729 (1867). (G)

(221) Leake, J. P.: Encephalitis in St. Louis. J. A. M. A. 101: 928–929 (1933). (S)

(222) Leake, J. P.: Epidemiology of encephalitis, with special reference to the 1933 epidemic. Am. J. Pub. Health 23: 1140–1143 (1933). (S)

(223) Leake, J. P.: Epidemic of infectious encephalitis. Pub. Health Rep. 56: 1902–1905 (1941). (S, W)

(224) Leake, J. P., Musson, E. K., and Chope, H. D.: Epidemiology of epidemic encephalitis, St. Louis type. J. A. M. A. 103: 728–731 (1934). (S)

(225) Lennette, E. H.: Isolation of St. Louis encephalitis virus from a fatal human case in California. Proc. Soc. Exper. Biol. & Med. 61: 206–210 (1946). (S)

(226) Lennette, E. H., and Fox, J. P.: Anticorpos neutralizantes para a amostra leste do virus de encefalomielite equina em equideos no Brasil. Mem. Inst. Oswaldo Cruz. 38: 85–92 (1943). (E)

(227) Lennette, E. H., and Koprowski, H.: Human infection with Venezuelan equine encephalomyelitis virus. J. A. M. A. 123: 1088–1096 (1943). (V)

(228) Lennette, E. H., and Koprowski, H.: Antigenic relationships of the West Nile, Japanese B encephalitis, and St. Louis encephalitis viruses. J. Immunol. 52: 235–246 (1946). (S)

(229) Lumsden, L. L.: Observations on the epidemiological features of epidemic encephalitis lethargica. Unpublished report. Washington, D. C., Office of the Surgeon General, U. S. Public Health Services, 1934, 18 pp. (S)

(230) Lytle, W. H.: Encephalomyelitis (California horse disease) spread by mosquitoes. Oregon Department of Agriculture, Bull. No. 18, 1933, pp. 19–20. (W)

(231) Mace, D. L., Ott, R. L., and Cortez, F. S.: Evidence of the presence of equine encephalomyelitis virus in Philippine animals. Bull. U. S. Army Medical Department 9: 504–507 (1949). (E)

(232) Macnalty, A.: The epidemiology of encephalitis lethargica. Proc. Roy. Soc. Med. 31: 1–8 (1937).

(233) Madsen, D. E., and Knowlton, G. F.: Mosquito transmission of equine encephalomyelitis. J. Am. Vet. M. A. 86: 662–666 (1935). (W)

(234) Madsen, D. E., Knowlton, G. F., and Rowe, J. A.: Further studies on transmission of equine encephalomyelitis by mosquitoes. J. Am. Vet. M. A. 89: 187–196 (1936). (W)

(235) Manzelli, M. A.: A survey of the arthropod vectors of equine encephalomyelitis and encephalitis. J. New York Entom. Soc. 56: 79–107 (1948). (G)

(236) Marsh, H.: Losses of undetermined cause following an outbreak of equine encephalomyelitis. J. Am. Vet. M. A. 91: 88–93 (1937).

(237) Matheson, R.: Important mosquito-borne diseases that are likely to affect the Middle Atlantic and Northeastern States: Equine encephalomyelitis. Proc. New Jersey Mosquito Exterminator Association, 29th annual meeting, 1942, pp. 8–9. (G)

(238) McCordock, H. A., Smith, M. G., and Moore, E.: Isolation of St. Louis encephalitis virus during inter- and epidemic periods. Proc. Soc. Exper. Biol. & Med. 37: 288–290 (1937). (S)

(239) McGugan, A. C.: Equine encephalomyelitis: (western type) in humans in Alberta, 1941. Canad. J. Pub. Health 33: 148–151 (1942). (W)

(240) McLintock, J.: Collecting and handling mosquitoes on western equine encephalitis investigations in Manitoba. Canad. J. Res., Sec. E., 24: 55–62 (1946). (W)

(241) McNutt, S. H., and Packer, A: Isolation of western equine encephalomyelitis and hog-cholera viruses from supposedly hog-cholera-immune swine. Vet. Med. 38: 22–25 (1943). (W)

(242) Meiklejohn, G., and Hammon, W. M.: Epidemic of encephalitis, predominantly St. Louis type, in Pinal County, Ariz. J. A. M. A. 118: 961–964 (1942). (S)

(243) Melnick, J. L.: Detection of the virus of mouse encephalomyelitis in the intestines of normal kangaroo rats. J. Immunol. 47: 231–236 (1943).

(244) Merrill, M. H.: The mosquito as a possible vector of equine encephalomyelitis. Proc. California Mosquito Control Association, 11th annual conference, 1940, pp. 23–31. (W, E)

(245) Merrill, M. H., and TenBroeck, C.: Multiplication of equine encephalomyelitis virus in mosquitoes. Proc. Soc. Exp. Biol. & Med. 32: 421–423 (1934). (G)

(246) Merrill, M. H., and TenBroeck, C.: The transmission of equine encephalomyelitis virus by *Aedes aegypti*. J. Exper. Med. 62: 687–695 (1935). (W)

(247) Merrill, M. H., Lacaillade, C. W., Jr., and TenBroeck, C.: Mosquito transmission of equine encephalomyelitis. Science 80: 251–252 (1934). (W, E)

(248) Meyer, K. F.: A summary of recent studies on equine encephalomyelitis. Ann. Int. Med. 6: 645–654 (1932). (G)

(249) Meyer, K. F.: Equine encephalomyelitis. North Am. Vet. 14: 30–48 (1933). (G)

(250) Meyer, K. F.: Neurotropic virus infections of the horse. Proc. Pacific Science Congress, 5th, 1933, pp. 2915–2925. (G)

(251) Meyer, K. F.: Equine encephalomyelitis in 1933. *In* Animal Path. Exchange. University of Illinois Agricultural Extension Service, Urbana, March, April, May, 1934, 4 pp. (G)

(252) Meyer, K. F., Haring, C. M., and Howitt, B.: The etiology of epizootic encephalomyelitis of horses in the San Joaquin Valley, 1930. Science 74: 227–228 (1931). (W)

(253) Meyer, K. F., Wood, F., Haring, C. M., and Howitt, B.: Susceptibility of non-immune, hyperimmunized horses and goats to eastern, western and Argentine virus of equine encephalomyelitis. Proc. Soc. Exper. Biol. & Med. 32: 56–58 (1935). (W)

(254) Miles, V. I., Howitt, B. F., Gorrie, R., and Cockburn, T. A.: Encephalitis in the Midwest. V. Western equine encephalomyelitis viruses recovered from mites, *Dermanyssus americanus* Ewing. Proc. Soc. Exper. Biol. & Med. 77: 395–396 (1951). (W)

(255) Miller, A. W.: Report on infectious equine encephalomyelitis in the United States in 1943. Washington, D. C., U. S. Bureau of Animal Industry, 1944, 4 pp. (G)

(256) Miller, A. W.: Report on infectious equine encephalomyelitis in the United States in 1944. Washington, D. C. U. S. Bureau of Animal Industry, 1945, 4 pp. (E, W)

(257) Milner, K. C., Kelley, G. P., and Schaeffer, M. F.: Human infection with virus of equine encephalomyelitis, eastern type, in Louisiana. New Orleans M. & S. J. 100: 270–273 (1947). (E)

(258) Milzer, A.: Studies on the transmission of lymphocytic choriomeningitis virus by arthropods. J. Infect. Dis. 70: 152–172 (1942). (G)

(259) Milzer, A.: Routine laboratory diagnosis of virus and rickettsial diseases. J. A. M. A. 143: 219–224 (1950). (G)

(260) Mitamura, T., Yamada, S., Hazato, H., Mori, K., Hosoi, T., Kitaoka, M., Watanabe, S., Okubo, K., and Tenjin, S.: Über den Infektionsmodus der epidemischen Enzephalitis. Experimentelle Untersuchungen über ihre Ansteckung durch Mücken. Tr. Jap. Path. Soc. 27: 573–580 (1937). (S)

(261) Mitamura, T., Kitaoka, M., Watanabe, S., Iwasaki, T., Ishikawa, I., Tenjin, S., Jo, K., Yoshida, H., Yoshimatsu, D., Warabioka, K., Shimizu, M., and Watanabe, T.: Über die Bedeutung der Mücken fur die Ubertragung verscheidener Enzephalitis-Virusarten. Tr. Jap. Path. Soc. 30: 561–570 (1940). (S)

(262) Mitchell, C. A., and Walker, R. V. I.: Studies in equine encephalomyelitis: Susceptibility of some mammals and birds. Canad. J. Comp. Med. 5: 314–319 (1941). (W)

(263) Mohler, J. R.: Report on outbreaks of infectious equine encephalomyelitis in the United States, 1935–47. Washington, D. C., U. S. Bureau of Animal Industry, 1938, 4 pp. (E, W)

(264) Mohler, J. R.: Control methods in infectious equine encephalomyelitis. Vet. Med. 33: 36–39 (1938). (W, E)

(265) Mohler, J. R.: Report on the 1938 outbreak of infectious equine encephalomyelitis in the United States. Washington, D. C., U. S. Bureau of Animal Industry, 1939, 5 pp. (W, E)

(266) Mohler, J. R.: Report on infectious equine encephalomyelitis in the United States in 1939. Washington, D. C., U. S. Bureau of Animal Industry, 1940, 5 pp. (W, E)

(267) Mohler, J. R.: Brief history of infectious equine encephalomyelitis and related affections in the United States. J. Am. Vet. M. A. 98: 451–453 (1941). (W, E)

(268) Mohler, J. R.: Infectious equine encephalomyelitis in the United States in 1940. J. Am. Vet. M. A. 98: 381–383 (1941). (W, E)

(269) Mohler, J. R.: Report on infectious equine encephalomyelitis in the United States in 1940. Washington, D. C., U. S. Bureau of Animal Industry, 1941, 5 pp. (W, E)

(270) Mohler, J. R.: Report on infectious equine encephalomyelitis in the United States in 1941. Washington, D. C., U. S. Bureau of Animal Industry, 1942, 8 pp. (W, E)

(271) Mohler, J. R.: Report on infectious equine encephalomyelitis in the United States in 1942. Washington, D. C., U. S. Bureau of Animal Industry, 1943, 8 pp. (W, E)

(272) Muckenfuss, R. S.: Clinical observations and laboratory investigations on the 1933 epidemic of encephalitis in St. Louis. Bull. New York Acad. Med. 10: 444–453 (1934). (S)

(273) Muckenfuss, R. S., Armstrong, C., and McGordock, H. A.: Encephalitis: Studies on experimental transmission. Pub. Health Rep. 48: 1341–1343 (1933) (S)

(274) Muckenfuss, R. S., Armstrong, C., and Webster, L. T.: Etiology of the 1933 epidemic of encephalitis. J. A. M. A. 103: 731–733 (1934). (S)

(275) Murphy, J. H.: Comparative epidemiology of encephalomyelitis and poliomyelitis. Nebraska M. J. 24: 453–457 (1939). (S)

(276) Neal, J. B.: Lethargic encephalitis. Arch. Neurol. & Psychiat. 2: 271–290 (1919). (G)

(277) Neal, J. B.: The present status of the etiology of epidemic encephalitis. J. A. M. A. 91: 231–234 (1928). (G)

(278) Neal, J. B.: Types of epidemic encephalitis: A comparison of cases seen in St. Louis in 1933 with those seen in New York City. Am. J. Pub. Health 23: 1144–1148 (1933). (S)

(279) Neal, J. B.: The encephalitis problem. J. A. M. A. 103: 726–728 (1934). (G)

(280) Neal, J. B.: Encephalitis: A clinical study. New York, Grune & Stratton Co., 1942, 563 pp. (G)

(281) Norris, M.: Recovery of a strain of western equine encephalitis virus from *Culex restuans* (Theo.) (Diptera: Culicidae). Canad. J. Res., Sect. E., 24: 63–70 (1946). (W)

(282) Oglesby, W. T.: 1947 outbreak of infectious equine encephalomyelitis in Louisiana. J. Am. Vet. M. A. 113: 267–270 (1948). (E)

(283) O'Leary, J. L., Smith, M. S., and Reames, H. R.: Influence of age on susceptibility of mice to St. Louis encephalitis virus and on the distribution of lesions. J. Exper. Med. 75: 233–246 (1942). (S)

(284) Olitsky, P. K., and Morgan, I. M.: Protective antibodies against equine encephalomyelitis virus in serum of laboratory workers. Proc. Soc. Exper. Biol. & Med. 41: 212–215 (1939). (E, W)

(285) Osteen, O. L.: Infectious equine encephalomyelitis: Midwinter case. J. Am. Vet. M. A. 94: 441–442 (1939). (E)

(286) Peck, J. L., and Sabin, A. B.: Multiplication and spread of the virus of St. Louis encephalitis in mice with special emphasis on its fate in the alimentary tract. J. Exper. Med. 85: 647–662 (1947). (S)

(287) Perdrau, J. R.: The Australian epidemic of encephalomyelitis (X disease). J. Path. & Bact. 42: 59–65 (1936).

(288) Perdrau, J. R.: The serological relationship between the viruses of Japanese and St. Louis encephalitis. J. Path. & Bact. 50: 545–547 (1940). (S)

(289) Peters, R. F., Thurman, D. C., Markos, B. G., and Mulhern, T. D.: The unfolding program of vector control in California with reference to studies of mosquito biology. J. Nat. Malaria Soc. 9: 173–175 (1950). (G)

(290) Philip, C. B.: Flowers as a suggested source of mosquitoes during encephalitis studies, and incidental mosquito records in the Dakotas in 1941. J. Parasitol. 29 (5): 328–329 (1943). (G)

(291) Philip, C. B., and Cox, H. R.: Equine encephalomyelitis in western Montana in 1936: A brief discussion of the implications of arthropod transmission. J. Parasitol. 22: 542 (1936). (W)

(292) Philip, C. B., and Smadel, J. E.: Transmission of West Nile virus by infected *Aedes albopictus*. Proc. Soc. Exper. Biol. & Med. 53: 49–50 (1943). (G)

(293) Philip, C. B., Cox, H. R., and Fountain, J. H.: Protective antibodies against St. Louis encephalitis virus in the serum of horses and man. Pub. Health Rep. 56: 1388–1391 (1941). (S)

(294) Platou, R. V.: Equine encephalomyelitis in infancy. Am. J. Dis. Child. 60: 1155–1169 (1940). (G)

(295) Price, E. R.: Western equine encephalomyelitis. CDC Bull. 9: 5–6 (1950). (W)

(296) Price, E. R.: Encephalitis as a public health problem. J. Am. Vet. M. A. 116: 182–186 (1950). (G)

(297) Randall, R.: Equine encephalomyelitis. Vet. Bull., U. S. Army 36: 157–171 (1942). (G)

(298) Randall, R., and Eichorn, E. A.: Westward spread of eastern type equine encephalomyelitis virus. Science 93: 595 (1941). (E)

(299) Records, E.: Equine encephalomyelitis in Nevada. J. Am. Vet. M. A. 80: 509 (1932). (W)

(300) Records, E., and Vawter, L. R.: Equine encephalomyelitis. Nevada Agr. Exper. Sta. Bull. No. 132, 1933, 22 pp. (W)

(301) Records, E., and Vawter, L. R.: Equine encephalomyelitis antiserum. J. Am. Vet. M. A. 82: 608–616 (1933). (W)

(302) Records, E., and Vawter, L. R.: Equine encephalomyelitis cross-immunity in horses between western and eastern strains of virus. Supplemental Report. J. Am. Vet. M. A. 86: 773–777 (1935). (E, W)

(303) Records, E. (Chairman): Report of committee on miscellaneous transmissible diseases. Encephalomyelitis of equines. J. Am. Vet. M. A. 80: 509–511 (1932). (W)

(304) Rector, N. H.: Need for regulations on mosquito control in irrigation systems. CDC Bull. 9: 25 (1950). (G)

(305) Reeves, W. C.: Newer developments in knowledge of insect hosts and vectors of western equine and St. Louis encephalitis. Proc. California Mosquito Control Association, 12th annual conference, 1941, pp. 23–36. (S, W)

(306) Reeves, W. C.: Observations on the natural history of western equine encephalomyelitis. Proc., U. S. Livestock San. Association, 49th annual meeting, 1945, pp. 150–158. (W)

(307) Reeves, W. C., and Hammon, W. M.: Laboratory transmission of Japanese B encephalitis virus by seven species (three genera) of North American mosquitoes. J. Exper. Med. 83: 185–194 (1946).

(308) Reeves, W. C.: Epidemiology of the arthropod-borne virus encephalitis: A pan-American problem. Bol. Ofic. san. Panama. 27: 347–355 (1948). (G)

(309) Reeves, W. C.: Encephalitis: Epidemic potential suggested. Pub. Health Rep. 65: 1690 (1950). (G)

(310) Reeves, W. C.: The present status of knowledge of mosquito ecology in California. Proc., California Mosquito Control Association, 18th annual conference, 1950, pp. 59–61. (W)

(311) Reeves, W. C., Hammon, W. M., Lazarus, A. S., Brookman, B., McClure, H. E., and Doetschman, W. H.: The changing picture of encephalitis in the Yakima Valley, Washington. J. Infect. Dis. 90: 291–301 (1952). (G)

(312) Reeves, W. C., and Hammon, W. M.: Mosquitoes and encephalitis in the Yakima Valley, Washington. IV. A trap for collecting live mosquitoes. J. Infect. Dis. 70: 275–277 (1942). (E)

(313) Reeves, W. C., and Hammon, W. M.: Feeding habits of the proven and possible mosquito vectors of western equine and St. Louis encephalitis in the Yakima Valley, Washington. Am. J. Trop. Med. 24: 131–134 (1944). (S, W)

(314) Reeves, W. C., Hammon, W. M., and Izumi, E. M.: Experimental transmission of St. Louis encephalitis virus by *Culex pipiens* Linnaeus. Proc. Soc. Exper. Biol. & Med. 50: 125–128 (1942) (S)

(315) Reeves, W. C., Mack, W. N., and Hammon, W. M.: Epidemiological studies on western equine encephalomyelitis and St. Louis encephalitis in Oklahoma, 1944. J. Infect. Dis. 81: 191–196 (1947). (W)

(316) Reeves, W. C., Hammon, W. M., Furman, D. P., McClure, H. E., and Brookman, B.: Recovery of western equine encephalomyelitis virus from wild bird mites (*Liponyssus sylviarum*) in Kern County, Calif. Science 105: 411–412 (1947) (W)

(317) Reeves, W. C., Brookman, B., and Hammon, W. M.: Studies on the flight range of certain *Culex* mosquitoes, using a fluorescnt-dye marker, with notes on *Culiseta* and *Anopheles*. Mosquito News 8: 61–69 (1948). (G)

(318) Reeves, W. C., Washburn, G. E., and Hammon, W. M.: Western equine encephalitis control studies in Kern County, California, 1945. I. The effectiveness of residual DDT deposits on adult *Culex* mosquito populations. Am. J. Hyg. 47: 82–92 (1948). (W)

(319) Remlinger, P., and Bailly, J.: Modes de transmission du virus de l'encephalomyelite des equides en Argentine. Compt. rend. Soc. de biol. 121: 983–985 (1935). (W)

(320) Remlinger, P., and Bailly, J.: Transmission de l'encephalomyelite argentine des equides a l'oie, au canard, a la buse et au merle. Compt. rend. Soc. de biol. 121: 146–149 (1936). (W)

(321) Remlinger, P., and Bailly, J.: Transmission du virus de l'encephalomyelite des equides a la cigogne. Compt. rend. Soc. de biol. 122: 518–519 (1936). (W)

(322) Remlinger, P., and Bailly, J.: Transmission de l'encephalomyelite des equides (virus californien) au vautour fauve (Vultur fulvis Briss). Compt. rend. Soc. biol. 123: 562–563 (1936). (W)

(323) Rempel, J. G., Riddell, W. A., and McNelly, E. M.: Multiple feeding habits of Saskatchewan mosquitoes. Canad. J. Res., Sect. E, 24 (2): 71–78 (1946). (W)

(324) Richter, R. B.: Case of western equine encephalomyelitis occurring sporadically in a metropolitan area. Report of a case. J. A. M. A. 119: 486–487 (1942). (W)

(325) Riley, W. A.: The role of insects and allied forms in the transmission of diseases due to filterable viruses. Minnesota Med. 21: 817–821 (1938). (G)

(326) Riley, W. A.: Encephalitis in Minnesota. Minnesota Med. 24: 666, 706 (1941). (G)

(327) Rivers, T. M.: Viral and rickettsial infections of man. Philadelphia, Pa., J. B. Lippincott Co., 1948, 587 pp. (G)

(328) Roca-Garcia, M.: The isolation of three neurotropic viruses from forest mosquitoes in eastern Colombia. J. Infect. Dis. 75: 160–169 (1944). (G)

(329) Rosenbusch, F.: La immunizacion activa con el virus de la encefalomielitis infecciosa en la Argentina. Estudio comparative con el virus Californiano. Ann. del la Soc. rural Argentina 68: 273 (1934). (W)

(330) Rosenbusch, F.: Equine encephalomyelitis in the Argentine and its experimental aspects. Proc. Pacific Science Congress, 6th, 1939, pp. 209–214. (W)

(331) Rosenow, E. C., and Caldwell, H. W.: Studies on the etiology and serum treatment of encephalitis during the epidemic in North Dakota and Minnesota. Proc. Staff Meet., Mayo Clin. 16 (37): 587–588 (1941). (W)

(332) Ruchman, I.: Relationship between epidemic keratoconjunctivitis and St. Louis encephalitis. Proc. Soc. Exper. Biol. & Med. 77: 120–125 (1951). (S)

(333) Sabin, A. B., Duffy, C. E., Warren, J., Ward, R., Peck, J. L., Jr., and Ruchman, I.: The St. Louis and Japanese B types of epidemic encephalitis development of noninfective vaccines: Report of basic data. J. A. M. A. 122: 477–486 (1943). (S)

(334) Schaeffer, M.: Encephalitis. CDC Bull. 9: 1–2 (1950). (G)

(335) Schlotthauer, C. F.: The susceptibility of dogs to virus of equine encephalomyelitis (western). J. Am. Vet. M. A. 99: 388–390 (1941). (W)

(336) Schoening, H. W.: Equine encephalomyelitis. J. Am. Vet. M. A. 95: 268–271 (1939). (G)

(337) Schoening, H. W.: The epizootiology of infectious equine encephalomyelitis in the United States. Vet. Bull. 34: 13–19 (1940). (G)

(338) Schoening, H. W., Giltner, L. T., and Shahan, M. S.: Equine encephalomyelitis produced by inoculation of human encephalitis virus. Science 88: 409–410 (1938). (E)

(339) Schoening, H. W., Giltner, L. T., and Shahan, M. S.: Infectious equine encephalomyelitis in 1939. Proc. U. S. Livestock Sanitary Association, 43d annual meeting, 1939, pp. 145–150. (G)

(340) Schoening, H. W., Shahan, M. S., Giltner, L. T., and Osteen, O. L.: The longevity of encephalomyelitis immunity induced in horses by vaccination. J. Am. Vet. M. A. 102: 18–21 (1943). (W)

(341) Schofield, F. S.: The isolation and identification of the virus of equine encephalomyelitis. Canad. J. Pub. Health 31: 31–32 (1940). (G)

(342) Schultz, E. W., Williams, G. F., and Hetherington, A.: Cultivation of the virus of St. Louis encephalitis. Proc. Soc. Exper. Biol. & Med. 38: 799–800 (1938).

(343) Schwarte, L. H., Biester, H. E., and Murray, C.: A disease of horses caused by feeding moldy corn. J. Am. Vet. M. A. 90: 76–85 (1937). (G)

(344) Sellards, A. W., Tyzzer, E. E., and Bennett, B. L.: The infection of birds with the virus of equine encephalomyelitis. Am. J. Hyg. 33 (B): 63–68 (1941). (E)

(345) Shahan, M. S., and Giltner, L. T.: Some aspects of infection and immunity in equine encephalomyelitis. J. Am. Vet. M. A. 84: 928–934 (1934). (G)

(346) Shahan, M. S., and Giltner, L. T.: Equine encephalomyelitis studies. I. Cross-immunity tests between eastern and western types of virus. J. Am. Vet. M. A. 86: 764–772 (1935). (G)

(347) Shahan, M. S., and Giltner, L. T.: Identity of viruses from cases of equine encephalomyelitis during 1942. J. Am. Vet. M. A. 102: 271–275 (1943). (G)

(348) Shahan, M. S., and Giltner, L. T.: A review of the epizootiology of equine encephalomyelitis in the United States. J. Am. Vet. M. A. 107: 279–288 (1945). (G)

(349) Shahan, M. S., Giltner, L. T., and Schoening, H. W.: A review of the 1938 outbreak of infectious equine encephalomyelitis in the United States. Proc. U. S. Livestock Sanitary Association, 42d annual meeting, 1938, pp. 145–157. (W, E)

(350) Shahan, M. S., Giltner, L. T., Davis, C. L., and Huffman, W. T.: "Secondary disease" occurring subsequent to infectious equine encephalomyelitis. Vet. Med. 34: 354–358 (1939). (G)

(351) Shahan, M. S., Mott, L. O. Knudson, R. L., Osteen, O. L., and Giltner, L. T.: The relationship of St. Louis encephalitis virus to the equine encephalitis problem. Vet. Med. 37: 339–345 (1942). (G)

(352) Shaughnessy, H. J., and Milzer, A.: Experimental infection of *Dermacentor andersoni* Stiles with the virus of lymphocytic choriomeningitis. Am. J. Pub. Health 29: 1103–1108 (1939).

(353) Simmons, J. S.: Transmission of encephalomyelitis in the horse and possible vectors in the human being. New England J. Med. 220: 956–958 (1938). (G)

(354) Simmons, J. S., Kelser, R. A., and Cornell, V. H.: Insect transmission experiments with herpes-encephalitis virus (preliminary note). Science 78: 243–246 (1933). (S)

(355) Simmons, J. S., Reynolds, F. H. K., and Cornell, V. H.: Transmission of virus of equine encephalomyelitis through *Aedes albopictus* Skuze. Am. J. Trop. Med. 16: 289–302 (1936). (W)

(356) Simms, B. T.: Report on infectious equine encephalomyelitis in the United States in 1945. Washington, D. C., U. S. Bureau of Animal Industry, 1946, 4 pp. (W, E)

(357) Simms, B. T.: Report on infectious equine encephalomyelitis in the United States in 1946. Washington, D. C., U. S. Bureau of Animal Industry, 1947, 4 pp. (W, E)

(358) Simms, B. T.: Report on infectious equine encephalomyelitis in the United States in 1947. Washington, D. C., U. S. Bureau of Animal Industry, 1948, 4 pp. (W, E)

(359) Simms, B. T.: Report on infectious equine encephalomyelitis in the United States in 1948. Washington, D. C., U. S. Bureau of Animal Industry, 1949, 4 pp. (W, E)

(360) Simms, B. T.: Report on infectious equine encephalomyelitis in the United States in 1949. Washington, D. C., U. S. Bureau of Animal Industry, 1950, 4 pp. (W, E)

(361) Simms, B. T.: Report on infectious equine encephalomyelitis in the United States in 1950. Washington, D. C., U. S. Bureau of Animal Industry, 1951, 4 pp. (W, E)

(362) Smith, H. F.: Epidemic encephalitis (encephalitis lethargica, Nona). Report of studies conducted in the United States. Pub. Health Rep. 36: 207–242 (1921).

(363) Smith, K. M.: The virus: Life's enemy. New York, Cambridge University Press, 1940, 176 pp. (G)

(364) Smith, M. G.: Survival of the virus of St. Louis encephalitis in rats and guinea pigs. J. Infect. Dis. 64: 307–309 (1939). (S)

(365) Smith, M. G., and Reames, H. R.: Serological relationship between the viruses of Japanese B and St. Louis encephalitis. Proc. Soc. Exper. Biol. & Med. 45: 838–842 (1940). (S)

(366) Smith, M. G., Lennette, E. H., and Blattner, R. H.: Etiologic studies of sporadic cases of encephalitis occurring in the St. Louis area in 1938. Am. J. Dis. Child. 59: 509–514 (1940). (S)

(367) Smith, M. G., Blattner, R. J., and Heys, F. M.: The isolation of the St. Louis encephalitis virus from chicken mites (*Dermanyssus gallinae*) in nature. Science 100: 362–363 (1944). (S)

(368) Smith, M. G., Blattner, R. J., and Heys, F. M.: Further isolation of St. Louis encephalitis virus; Congenital transfer of virus in chicken mites (*Dermanyssus gallinae*). Proc. Soc. Exper. Biol. & Med. 59: 136–138 (1945). (S)

(369) Smith, M. G., Blattner, R. J., and Heys, F. M.: St. Louis encephalitis: Infection of chicken mites, *Dermanyssus gallinae*, by feeding on chickens with viremia; Transovarian passage of virus into the second generation. J. Exper. Med. 84: 1–6 (1946). (S)

(370) Smith, M. G., Blattner, R. J., and Heys, F. M.: St. Louis encephalitis: Transmission of virus to chickens by infected mites (*Dermanyssus gallinae*) and resulting viremia as source of virus for infection of mites. J. Exper. Med. 86: 229–237 (1947). (S)

(371) Smith, M. G., Blattner, R. J., Heys, F. M., and Miller, A.: Experiments on the role of the chicken mite, *Dermanyssus gallinae*, and the mosquito in the epidemiology of St. Louis encephalitis. J. Exper. Med. 87: 119–138 (1948). (S)

(372) Smithburn, K. C.: Neurotropic viruses in Central Africa. Proc. Internat. Cong. Trop. Med. & Malaria, 4th, 1: 576–586 (1948). (S)

(373) Smithburn, K. C., and Haddow, A. J.: Semliki Forest virus. I. Isolation and pathogenic properties. J. Immunol. 49: 141–157 (1944).

(374) Smithburn, K. C., and Jacobs, H. R.: Neutralization-tests against neurotropic viruses with sera collected in Central Africa. J. Immunol. 44: 9–23 (1942). (S)

(375) Smithburn, K. C., Hughes, T. P., Burke, A. W., and Paul, J. H.: A neurotropic virus isolated from the blood of a native of Uganda. Am. J. Trop. Med. 20: 471–492 (1940). (S)

(376) Smithburn, K. C., Mahaffy, A. F., and Paul, J. H.: Bwamba fever and its causative virus. Am. J. Trop. Med. 21: 75–90 (1941).

(377) Smithburn, K. C., Haddow, A. J., and Mahaffy, A. F.: A neurotropic virus isolated from *Aedes* mosquitoes caught in the Semliki Forest. Am. J. Trop. Med. 26: 189–208 (1946).

(378) Sooter, C. A.: Insects and encephalitis. Proc. Am. A. Econ. Ent., N. Central States Br., 1950, p. 42. (G)

(379) Sooter, C. A., Howitt, B. F., Gorrie, R., and Cockburn, T. A.: Encephalitis in the Midwest. IV. Western equine encephalomyelitis virus recovered from nestling wild birds in nature. Proc. Soc. Exper. Biol. & Med. 77: 393–394 (1951). (W)

(380) Steele, J. H., and Habel, K.: Observations on an outbreak of encephalomyelitis in Panama. J. Am. Vet. M. A. 111: 263–268 (1947). (E)

(381) Sulkin, S. E.: Recovery of equine encephalomyelitis virus (western type) from chicken mites. Science 101: 381–383 (1945). (W)

(382) Sulkin, S. E.: Observations on the epidemiology of St. Louis encephalitis and equine encephalomyelitis. Proc. Am. Pub. Health A., Southern Br., 1949, pp. 24–28. (S)

(383) Sulkin, S. E., and Izumi, E. M.: Isolation of western equine encephalomyelitis virus from tropical fowl mites *Liponyssus bursa* (Berlese). Proc. Soc. Exper. Biol. & Med. 66: 249–250 (1947). (W)

(384) Sulkin, S. E., and Pike, R. M.: Viral infections contracted in the laboratory. New England J. Med. 241: 205–213 (1949). (G)

(385) Sulkin, S. E., Harford, C. G., and Bronfennbrenner, J.: Attempts to transmit the virus of St. Louis encephalitis to newly hatched chicks. J. Infect. Dis. 67: 252–257 (1940). (S)

(386) Sulkin, S. E., Goth, A., and Zarafonetis, C.: Influence of anesthesia on experimental western equine encephalomyelitis. Science 104: 53–54 (1946). (W)

(387) Syverton, J. T.: Discussion on epidemiology of equine encephalomyelitis. Proc. International Congress of Microbiology, 3d, 1940, pp. 306–307. (G)

(388) Syverton, J. T., and Berry, G. P.: Susceptibility of the gopher (*Citellus richardsonii*) (Sabine) to equine encephalomyelitis. Proc. Soc. Exper. Biol. & Med. 34: 822–824 (1936). (G)

(389) Syverton, J. T., and Berry, G. P.: An arthropod vector of equine encephalomyelitis, western strain. Science 84: 186–187 (1936). (W)

(390) Syverton, J. T., and Berry, G. P.: The tick as a vector for the virus disease, equine encephalomyelitis. J. Bact. 33: 60 (1937). (W)

(391) Syverton, J. T., and Berry, G. P.: Host range of equine encephalomyelitis: Susceptibility of the North American cottontail rabbit, jack rabbit, field vole, woodchuck and opossum to experimental infection. Am. J. Hyg. 32 (B): 19–23 (1940). (W, E)

(392) Syverton, J. T., and Berry, G. P.: The transmission of equine encephalomyelitis to the western burrowing owl, *Speotyto cunicularia hypugaea* (Bonaparte). Am. J. Hyg. 33 (B): 37–41 (1941). (W, E)

(393) Syverton, J. T., and Berry, G. P.: Hereditary transmission of western type of equine encephalomyelitis virus in wood tick, *Dermacentor andersoni* Stiles. J. Exper. Med. 73: 507–530 (1941). (W)

(394) Syverton, J. T., Cox, H. R., and Olitsky, P. K.: Relationship of the viruses of vesicular stomatitis and of equine encephalomyelitis. Science 78: 216–217 (1933). (G)

(*395*) TenBroeck, C.: Birds as possible carriers of the virus of equine encephalomyelitis. Arch. Path. 25: 759 (1938). (E)

(*396*) TenBroeck, C.: Transmission of equine encephalomyelitis. Proc. International Congress of Microbiology, 3d, 1940, pp. 300–301. (E)

(*397*) TenBroeck, C., and Merrill, M. H.: A serological difference between eastern and western equine encephalomyelitis viruses. Proc. Soc. Exper. Biol. & Med. 31: 217–220 (1933). (E, W)

(*398*) TenBroeck, C., and Merrill, M. H.: Transmission of equine encephalomyelitis by *Aedes aegypti.* Arch. Path. 20: 164 (1935). (W)

(*399*) TenBroeck, C., and Merrill, M. H.: Transmission of equine encephalomyelitis by mosquitoes. Am. J. Path. 11: 847 (1935). (E)

(*400*) TenBroeck, C., Hurst, E. W., and Traub, E.: Epidemiology of equine encephalomyelitis in the eastern United States. J. Exper. Med. 62: 677–685 (1935). (E)

(*401*) Theiler, M.: Spontaneous encephalomyelitis of mice, a new virus disease. J. Exper. Med. 65: 705–719 (1937).

(*402*) Thurman, D. C., Jr.: The ecology of *Aedes* mosquitoes in California. Proc. California Mosquito Control Association, 18th annual conference, 1950, pp. 62–65. (G)

(*403*) Thurman, D. C., Jr., and Mortenson, E. W.: Studies of the biology of *Aedes* mosquitoes in irrigated pastures in California during 1949. Proc. California Mosquito Control Association, 18th annual conference, 1950, pp. 66–69. (G)

(*404*) Tollock, G. S.: Mosquitoes and equine encephalomyelitis. Bull. Brooklyn Ent. Soc. 36: 35–38 (1941). (G)

(*405*) Trager, W.: Multiplication of the virus of equine encephalomyelitis in surviving mosquito tissues. Am. J. Trop. Med. 18: 387–393 (1938). (W)

(*406*) Traub, E.: Equine encephalomyelitis: Experimental studies on virus strain changed by passage through pigeon. Zentralbl. f. Bakt. 143: 7–22 (1938). (G)

(*407*) Traub, E., and TenBroeck, C.: Protective vaccination of horses with modified equine encephalomyelitis virus. Science 81: 572 (1935). (G)

(*408*) Tyzzer, E. E., Sellards, A. W., and Bennett, B. L.: The occurrence in nature of "equine encephalomyelitis" in the ring-necked pheasant. Science 88: 505–506 (1938). (E)

(*409*) Udall, D. H.: A report on the outbreak of "cerebrospinal meningitis" (encephalitis) in horses in Kansas and Nebraska in 1912. Cornell Vet. 3: 17–43 (1913). (G)

(*410*) Urban, W. D.: Avian encephalomyelitis (epidemic tremors of chicks). Bull. California Department of Agriculture 37: 142–144 (1948). (G)

(*411*) Van Roekel, H., and Clarke, M. K.: Equine encephalomyelitis virus (eastern type) isolated from ring-necked pheasant. J. Am. Vet. M. A. 94: 466–468 (1939). (E)

(*412*) Van Rooyen, C. E., and Rhodes, A. J.: Virus diseases of man. New York, T. Nelson and Sons, N. Y., 1948, 1202 pp. (G)

(*413*) Vawter, L. R., and Records, E.: Respiratory infection in equine encephalomyelitis. Science 78: 41–42 (1933). (G)

(*414*) Von Economo, C.: Encephalitis lethargica. Wien. klin. Wchnschr. 30: 581–585 (1917).

(*415*) Watson, W. D., and Smadel, J. E.: Susceptibility of hamsters to peripheral inoculation of western, eastern, and West Nile encephalitis viruses. Proc. Soc. Exper. Biol. & Med. 52: 101–104 (1943). (G)

(*416*) Webster, L. T.: Japanese B encephalitis virus: Its differentation from St. Louis encephalitis virus and relationship to louping ill virus. J. Exper. Med. 67: 609–617 (1938). (S)

(*417*) Webster, L. T.: Classification of primary encephalitides of man according to virus etiology: Present status. J. A. M. A. 116: 2840–2841 (1941). (G)

(*418*) Webster, L. T., and Fite, G. L.: A virus encountered in the study of material from cases of encephalitis in the St. Louis and Kansas City epidemic of 1933. Science 78: 463–465 (1933). (S)

(*419*) Webster, L. T., and Fite, G. L.: Contribution to the etiology of encephalitis: Differentiation of encephalitis by protection tests. Proc. Soc. Exper. Biol. & Med. 31: 344–346 (1933). (S)

(*420*) Webster, L. T., and Fite, G. L.: Serological relationship of St. Louis and Japanese encephalitis. Am. J. Path. 10: 667–668 (1934). (S)

(*421*) Webster, L. T., and Fite, G. L.: Experimental studies on encephalitis. I. Transmission of St. Louis and Kansas City encephalitis to mice. J. Exper. Med. 61: 103–114 (1935). (S)

(*422*) Webster, L. T., and Wright, F. H.: Recovery of eastern equine encephalomyelitis virus from brain tissue of human cases of encephalitis in Massachusetts. Science 88: 305–306 (1938). (E)

(*423*) Webster, L. T., Fite, G. L., and Clow, A. D.: Experimental studies on encephalitis III: Survival of encephalitis virus (St. Louis type) in *Anopheles quadrimaculatus.* J. Exper. Med. 61: 479–487 (1935). (S)

(*424*) Wenner, H. A. Kamitsuka, P., Kramer, M. C., Cockburn, T. A., and Price, E. R.: Encephalitis in the Missouri River Basin. II. Studies on a focal outbreak of encephalitis in North Dakota. Pub. Health Rep. 66: 1075–1085 (1951). (W)

(*425*) Wesselhoeft, C., Smith, E. C., and Branch, C. F.: Human encephalitis: Eight fatal cases with four due to the virus of equine encephalomyelitis. J. A. M. A. 111: 1735–1741 (1938). (E)

(*426*) William J. Matheson Commission for Encephalitis Research: Epidemic encephalitis. Third report. New York, Columbia University Press, 1939, 493 pp. (G)

(*427*) Woodward, B. T.: Entomologists aid in combating equine encephalomyelitis. California Vet. 3: (1950). (G)

(*428*) Wooley, J. G., and Armstrong, C.: The distribution of immunity against encephalitis virus of the St. Louis type in the United States as determined by the serum-protection test in white mice. Pub. Health Rep. 49: 1495–1505 (1934). (S)

(*429*) Wright, J.: Notes on the history of encephalitis lethargica: Before and after Galen. M. J. and Rec. 126: 569–571 (1927). (G)

(*430*) Wynns, H. L., and Hawley, C. J.: Epidemiology of epidemic encephalitis in California. Am. J. Pub. Health 29: 781–786 (1939). (W)

(*431*) Young, G. A., Young, R., and Koutsky, J.: Encephalitis in Nebraska during the summer of 1941. Dis. Nerv. System 4: 119–124 (1943). (S)

(*432*) Zinsser, H.: The present status of knowledge regarding epidemic encephalitis. Arch. Path. 6: 271–301 (1928). (G)

(*433*) Zwick, W., and Seifried, O.: Untersuchungen über die in Hessen gehauft auftretende seuchenhafte Gehirn und Ruckenmarksentzundung (Bornasche Krankheit) bei Pferden. Berl. tierärztl. Wchnschr. 40: 465–471 (1924).

Current Titles

Public Health Monographs

No. 8. Pretesting and evaluating health education (4 papers). Andie L. Knutson, Benjamin Shimberg, Jane S. Harris, and Mayhew Derryberry. (Public Health Service Publication No. 212) 1952. 26 pages. Illustration. 20 cents. [Published concurrently with *Public Health Reports*, 68: (1) see pages 54–55 summary.]

No. 9. Survey of antimalarial agents: Chemotherapy of *Plasmodium gallinaceum* infections; correlation of structure and action. G. Robert Coatney, W. Clark Cooper, Nathan B. Eddy, and Joseph Greenburg. (Public Health Service Publication No. 193) 1953. 323 pages. $1.25. [Published concurrently with *Public Health Reports*, 68: (1); see page 56 for summary.]

No. 10. Excess deaths from chronic disease during influenza epidemics. Selwyn D. Collins and Josephine Lehmann. (Public Health Service Publication No. 213) 1952. 22 pages. Illustrations. 20 cents. [Published concurrently with *Public Health Reports*, 68: (2); see page 173 for summary.]

No. 11. The resistance of construction materials to penetration by rats. C. M. Tarzwell, R. L. Stenburg, H. P. Nicholson, and W. D. Lynn. (Public Health Service Publication No. 277) 1953. 16 pages. Illustrations. 20 cents. [Published concurrently with *Public Health Reports*, 68: (8); see page 824 for summary.]

No. 12. Evaluation of cancer diagnostic tests (10 papers). Nils Eriksen, Lester D. Ellerbrook, Stuart W. Lippincott, Andrew C. Peacock, Joseph K. Cline, Jane Howle, Jane Reid, Leslie Johnson, S. C. Kasdon, F. Homburger, Jack H. Hill, J. Warren Kyle, Sara G. Richmond, Douglas H. Sprunt, William H. Fishman, and C. D. Bonner. (Public Health Service Publication No. 275) 1953. 51 pages. Illustrations. 30 cents. [Published concurrently with *Public Health Reports*, 68: (9); see pages 880–884 for summary article.]

No. 13. Staffing of State and local health departments—1951. Jack C. Haldeman, Bess A. Cheney, and Evelyn Flook. (Public Health Service Publication No. 279) 1953. 56 pages. Illustrations. 30 cents. [Published concurrently with *Public Health Reports* 68: (9); see pages 909–910 for summary.]

No. 14. Accident frequency, place of occurrence, and relation to chronic disease. Selwyn D. Collins, F. Ruth Phillips, and Dorothy S. Oliver. (Public Health Service Publication No. 249) 1953. 68 pages. Illustrations. 35 cents. [Published concurrently with *Public Health Reports* 68: (10); see pages 991–993 for summary article.]

No. 15. Records and reports of local health departments. Olive G. Johnson. (Public Health Service Publication No. 285) 1953. 96 pages. Illustrations. 45 cents. [Published concurrently with *Public Health Reports* 68: (11); see pages 1078–1082 for summary article.]

No. 16. Dental, eye, and preventive medical services. Selwyn D. Collins and F. Ruth Phillips. (Public Health Service Publication No. 290) 1953. 28 pages. Illustrations. 20 cents. [Published concurrently with *Public Health Reports* 68: (11); see pages 1115–1116 for summary.]

No. 17. Rehabilitation of mental hospital patients. Charlotte Green Schwartz. (Public Health Service Publication No. 297). 76 pages. 45 cents. [Published concurrently with *Public Health Reports* 68: (12); see pages 1237–1238 for summary.]

No. 18. The *Culicoides* of the eastern United States. Richard H. Foote and Harry D. Pratt. (Public Health Service Publication No. 298) 1953. 56 pages. 11 plates (126 figures). 40 cents. [Published concurrently with *Public Health Reports* 69: (2); see pages 123–129 for summary article.]

No. 19. Medical service areas and distances traveled for physician care in western Pennsylvania. (2 papers.) Antonio Ciocco and Isidore Altman. (Public Health Service Publication No. 248) 1954. 32 pages. Illustrations. 25 cents. [Published concurrently with *Public Health Reports* 69: (3); see pages 321–322 for summary.]

No. 20. Gamma globulin in the prophylaxis of poliomyelitis. (Public Health Service Publication No. 358) 1954. 178 pages. Illustrations. $1.25. [Published concurrently with *Public Health Reports* 69: (5); see page 519 for summary.]

No. 21. Why some sanitary engineers leave the field. Irwin M. Rosenstock and Arthur P. Miller. (Public Health Service Publication No. 359) 1954. 16 pages. Illustrations. 20 cents. [Published concurrently with *Public Health Reports* 69: (9); see pages 865–866 for summary.]

No. 22. A study of the Bethesda-Ballerup group of paracolon bacilli. Mary G. West and Philip R. Edwards. (Public Health Service Publication No. 362) 1954. 35 pages. 30 cents. [Published concurrently with *Public Health Reports* 69: (10); see pages 1013–1014 for summary.]

No. 23. Biological factors in the transmission of American arthropod-borne virus encephalitides. Frederick F. Ferguson. (Public Health Service Publication No. 372) 1954. 37 pages. 30 cents. [Published concurrently with *Public Health Reports* 69: (10); see pages 1095–1096 for summary.]

PHR

Public Health Monographs are edited and issued by *Public Health Reports*, the monthly journal the Public Health Service.

Readers of Public Health Monographs will find much of interest in each issue of *Public Health Reports*. If you would like your own personal subscription, send $4.25 (75 cents additional for foreign mailing) direct to the Superintendent of Documents, Government Printing Office, Washington 25, D. C. Enclose check, post office money order, or Government Printing Office coupons. Do not send currency or stamps.

On direct request to the Public Health Service, a sample copy of *Public Health Reports* will be sent to you.

ORDER BLANK FOR PHR

To: Superintendent of Documents
Government Printing Office
Washington 25, D. C.

Please enter my subscription for Public Health Reports. I am enclosing Money Order ☐ Check ☐ for this subscription. $4.25 a year; 75 cents additional for foreign mailing.

Please address the PHR as follows: ______________________

____

____

☆ U. S. GOVERNMENT PRINTING OFFICE: 1954 O—300402

PUBLIC HEALTH MONOGRAPHS

are edited and issued by *Public Health Reports* under the general direction of the Board of Editors. The monographs present contributions to knowledge in the fields of public health, particularly material that is extensive, detailed, or specialized. All manuscripts considered for publication as monographs receive the same technical and scientific review as papers submitted to *Public Health Reports*. The opinions expressed are those of the authors and do not necessarily reflect the views of *Public Health Reports* or the Public Health Service. Trade names are used for identification only and do not represent an endorsement by the Public Health Service.

Public Health Monographs are listed in the table of contents of the issue of *Public Health Reports* with which they are concurrently published. They are also listed in its annual index, in the *Quarterly Cumulative Index Medicus* (American Medical Association), the *Current List of Medical Literature* (Armed Forces Medical Library), and the *Industrial Arts Index*.

The monographs replace in whole or in part a number of publications series previously issued by the Public Health Service, including Public Health Bulletins, Bulletins of the National Institutes of Health, Venereal Disease Bulletins, and Supplements to the *Journal of Venereal Disease Information* and to the former weekly *Public Health Reports*.

Single copies of most Public Health Monographs are available upon request to the Public Inquiries Branch, U. S. Public Health Service, Washington 25, D. C. Orders for quantities should be sent to the Superintendent of Documents, U. S. Government Printing Office, Washington 25, D. C., where they are available at the prices shown, with a 25-percent reduction on orders for 100 or more. In ordering, give title, monograph number, and Public Health Service Publication number and enclose cash, check, or money order. Stamps are not acceptable.