OHIO: *Erie County:* Castalia, *R. F. Griggs* 638a, July 10, 1900 (OSU, as *S. cordata* × *candida*). Cedar Point, *A. D. Selby* 1449, 1450 ("7 ft, densely woolly"), 1451 ("4 ft., densely woolly"), 1456 ("4–5 ft"), 1458 ("8–10 ft"), all May 18, 1900 (all OES); on a sand dune, *Griggs*, Aug. 14, 1900 (OSU, as *S. cordata*, sprout with leaves up to 6.5–7 cm wide and more than 15 cm long; also in CRB); *F. O. Grover*, June 9, 1902 (OSU, as *S. cordata*); *Griggs*, June 28, 1904 (2 sheets, as *S. adenophylla*, OSU).

The following sterile specimens are referred somewhat doubtfully to the new variety, pending collection of fruiting material from these or other localities remote from Lake Erie:

MICHIGAN: *Huron County:* Single clump of small sprouts 3–4 ft high on land side of second row of dunes on Sand Point, Bay Port, *C. R. Ball* 70b, Sept. 17, 1902 (CRB, as *S. glaucophylla*).

VIRGINIA: *Roanoke County:* Back Creek, near Starkey, "much whiter looking than ordinary willow," *J. Fauntleroy* 663, Aug. 8, 1914 (USN, as *S. cordata*). The leaves are rather large, lanceolate, cordate at base, closely beset with incurved teeth, branchlets white-pilose, also the bud scales, petioles, midribs, and blades of younger leaves.

Montgomery County: Norris Run, near New River, *A. B. Massey*, Sept. 28, 1938 (VPI, as *S. cordata*), the leaves white pubescent and white-glaucous.

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MEDICAL ENTOMOLOGY.—*Mosquito transmission of encephalomyelitis, or brain fever, of horses.*[1]

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The well-recognized economic importance of mosquitoes has been further emphasized by the recent discovery that these insects are capable of transmitting equine encephalomyelitis, or brain fever, of horses. This malady has appeared periodically for many years in outbreak proportions in various parts of the United States, although until recently the disease has not been clearly differentiated from forage poisoning.

The disease is caused by a minute organism so small that it can not be seen with the highest-power microscopes, and it passes through the clay filters that catch and hold the germs responsible for most diseases.

[1] Read before the 13th Annual Meeting of the Florida Anti-Mosquito Association, Daytona Beach, Fla., April 17, 1939. Received May 27, 1939.

There are recognized at present two rather distinct types of the disease, usually spoken of as the eastern and western. The former is confined to the Eastern Seaboard and is generally acute, producing a mortality of 80 to above 90 percent. The western type is less severe, the mortality usually being about 20 percent.

The economic losses due to the disease are very heavy, and practically all parts of the country have felt the blow. For instance, the Bureau of Animal Industry, United States Department of Agriculture, has collected records of 184,662 cases in 1938 in 39 States, with a mortality for the entire country of 21.4 percent, or a death loss of about 39,518 horses and mules. Florida was among these States. The cases reported in this State were few but the mortality was high. The loss due to the occurrence of the disease among horses during 1938 may be conservatively placed at $10,000,000. In 1937, 173,889 cases were reported in 37 States, with a mortality in different areas ranging from about 20 percent in most western States to 100 percent in some eastern and southern States.

One of the most severe and widespread epizootics of the disease occurred in 1912. This outbreak reached its most devastating proportions in central and western Kansas and Nebraska, where the death loss was estimated at 35,000 head. Epizootics appear to recur at intervals of 10 to 15 years, with numerous sporadic or enzootic appearances between. The losses are intensified by the fact that work animals are often stricken in the midst of harvest, and in not a few cases a farmer has lost every animal at that critical period.

The importance of the disease and of mosquitoes that might serve as carriers of it was further emphasized by the determination last year that encephalomyelitis of horses is transmissible to man. L. D. Fothergill and associates[2] demonstrated the presence, in the brain of a child who had died of acute encephalitis, of a virus indistinguishable from the eastern type of encephalomyelitis which appeared among horses in Massachusetts during the summer of 1938. This identification was confirmed by L. T. Webster and F. H. Wright,[3] who described similar findings in four other fatal human cases. The diagnosis of the first case referred to above was further confirmed by H. W. Schoening, L. T. Giltner, and M. S. Shahan.[4]

R. F. Feemster,[5] of the Massachusetts Department of Public Health, published a general account of the outbreak in Massachusetts

[2] New England Journ. Med. 219: 411. 1938.
[3] Science 88: 305–306. 1938.
[4] Science 88: 409–410. 1938.
[5] Amer. Journ. Public Health 28: 1403–1410. 1938.

with comment on its relation to the epizootic among horses in that State. He points out that there is "a universal history of mosquito bites" among the cases investigated. He states also that no connection between cases indicating contact infection has been traced and that no contact at all with horses could be discovered in some of the cases. The incidence was high among children, 37 percent being under 2 years of age and 69 percent under 10 years. The mortality was high. Death occurred in 25 of the 38 cases definitely diagnosed as being caused by the eastern virus of encephalomyelitis or strongly suspected of being that disease.

In November 1938, C. M. Eklund and Alex Blumstein[6] reported the occurrence in 1937 of six cases of encephalitis in farmers in Minnesota, the serum from one neutralizing equine virus of the western type. One of the farmers had had no contact with sick horses. Two of the men died.

The occurrence of the disease in horses has attracted most attention, but a number of other animals and birds have been shown to be susceptible to artificial exposure. Among these are sheep, cattle, rabbits, guinea pigs, pigeons, monkeys, rats, mice, and ducks. Recently E. E. Tyzzer, A. W. Sellards, and B. L. Bennett[7] demonstrated the occurrence of the disease in pheasants in nature, and many other species of birds have been found to be susceptible to virus diseases of this group. L. D. Fothergill and J. H. Dingle[8] found the disease in common wild pigeons not in confinement.

The onset of the disease in horses and man is sudden, and nervous symptoms are apparent almost immediately and become progressively more pronounced, clearly showing involvement of the central nervous system.

Although it was suspected that some insect might be responsible for the sudden appearance and rapid spread of the malady in a given locality, it was not until 1933 that R. A. Kelser[9] announced experiments in which the western strain of the disease was transmitted from guinea pigs to numerous other guinea pigs, and to a horse by the bite of the yellow-fever mosquito [*Aedes aegypti* (L.)]. He showed that the virus was picked up by the mosquitoes only when they were fed on the artificially inoculated animals 48 to 120 hours after these animals had been injected, or during the fever period.

[6] Journ. Amer. Med. Assoc. **111**: 1734–1735. 1938.
[7] Science **88**: 505–506. 1938.
[8] Science **88**: 549–550. 1938.
[9] Journ. Amer. Vet. Med. Assoc. **82** (n.s. **35**): 767–771. 1933.

In 1934 M. H. Merrill, C. W. Lacaillade, Jr., and Carl Ten Broeck[10] reported experiments in which they had repeatedly transmitted the disease to healthy guinea pigs with the salt-marsh mosquito [*Aedes sollicitans* (Walk.)]. When the mosquitoes were fed on a guinea-pig brain, western virus suspension, and normal horse blood, they did not become infective until the seventh day. This mosquito transmitted the eastern type of the disease to healthy guinea pigs 11 days after the infective feeding and subsequently at least up to the thirty-third day. The authors also succeeded in transmitting the western virus with *Aedes aegypti* but failed to transmit the eastern type. They reported that a few tests with the brown salt-marsh mosquito [*Aedes cantator* (Coq.)] indicate that it will transmit the eastern virus. The northern house mosquito (*Culex pipiens* L.) and the common malaria mosquito (*Anopheles quadrimaculatus* Say) failed to transmit either type of the disease. These authors showed that in order to become infective the mosquitoes must feed on animals when the virus content of the blood is high and that the virus multiplies greatly in the mosquito. It has been shown that the virus is present in body fluids and in all parts of the body and even in the legs of mosquitoes.

In 1936 J. S. Simmons, F. H. K. Reynolds, and V. H. Cornell[11] published an account of the transmission of the western type under experimental conditions by *Aedes albopictus* Skuse, a preliminary announcement having been made in the Annual Report of the Surgeon General of the United States Army for 1934. This is an Asiatic species and hence can not have any part in the transmission of the disease in this country.

D. E. Madsen and G. F. Knowlton[12] proved that two common western species of mosquito, *Aedes dorsalis* (Meig.) and *A. nigromaculis* (Lud.), were capable of transmitting the western type of encephalomyelitis. The period during which the former species was infective was from 9 to 19 days after feeding on an infective animal, and in the latter from 4 to 10 days.

In 1937 R. A. Kelser[13] added the southern salt-marsh mosquito [*Aedes taeniorhynchus* (Wied.)] to the list of mosquitoes demonstrated to be capable of carrying the disease under experimental conditions. It is well known that this is the dominant salt-marsh mosquito in Florida.

[10] Science **80**: 251–252. 1934.
[11] Amer. Journ. Trop. Med. **16**: 289–302. 1936.
[12] Journ. Amer. Vet. Med. Assoc. **86** (n.s. **39**): 662–666. 1935; *ibid.* **89** (n.s. **42**): 187–196. 1936.
[13] Science **85**: 178. 1937.

The following year Dr. Kelser[14] described some experiments with *Aedes vexans* (Meig.) in the transmission of the western type. He experienced difficulty in keeping the mosquitoes alive for a sufficient period to make a satisfactory test, but one guinea pig out of three exposed to the mosquitoes during a period of 8 or 9 days after an infective meal was stricken.

Thus a total of 8 species of mosquitoes have been proved to be capable of transmitting the disease; and it should not be concluded that others may not be involved, as relatively few other species have been tested.

The experimental results indicate that the period in which the virus is present in the blood and can be picked up by mosquitoes is distinctly limited. This period is very early in the course of the disease, and in the case of horses it is mainly prior to the appearance of marked clinical symptoms.

Although infective mosquitoes have not been found in nature and many attempts to transmit the disease with these insects have failed, circumstantial evidence, as well as experimental results, point strongly to mosquitoes as carriers. It is noteworthy that the disease outbreaks occur mainly in summer and early in fall and are usually associated with great mosquito abundance and in the East with rainy or humid weather. The disease in the East is rather closely limited to the areas where salt-marsh mosquitoes occur. In the arid West it is closely associated with irrigated areas where mosquitoes abound. Some cases in winter have been reported, but diagnosis, at least in certain of these, is open to question, and in some of these instances it had not been possible to debar mosquitoes, for, as is well known, mosquitoes may be active in winter in warm barns and during warm periods.

In this connection attention is directed to a case in a horse at Ocala, Fla., which died on January 13, 1939. The brain of the animal was submitted by E. F. Thomas, of Ocala, to the U. S. Bureau of Animal Industry, and O. L. Osteen,[15] of that Bureau, confirmed the diagnosis as encephalomyelitis of the eastern type. During the period immediately preceding the appearance of this case the weather was very warm, several degrees above normal, and mosquitoes were undoubtedly active.

With reference to the human cases of this disease it can be said definitely that several that occurred in 1938 in Massachusetts were in individuals who had not had any direct or indirect contact with

[14] Journ. Amer. Vet. Med. Assoc. **92** (n.s. **45**): 195–203. 1938.
[15] Journ. Amer. Vet. Med. Assoc. **94** (n.s. **47**): 441–442. 1939.

horses, and some of them had had no association with any animal or bird. Some of the children, however, were reported as being "covered with mosquito bites" when admitted to the hospital.

The fact that a few cases of the disease appear among horses in May or June and a period of absence or practical absence of the malady intervenes, followed by a severe outbreak in August and September, leads one to wonder why the cases do not continue to increase right through the summer, and to raise the question as to where the disease holds over during such periods and also through the winter. Some have suggested that this carry-over may take place in some bird. As is well known, birds and poultry are freely attacked by mosquitoes. There is also the possibility that the virus may be kept alive in the mosquito or that it may be carried through the egg and larva from one generation to another. The tests with hereditary transmission conducted by L. T. Giltner and M. S. Shahan[16] have, however, been negative.

The fact that the virus is short-lived when exposed and that it will remain viable and multiply in mosquitoes also favors the mosquito theory of transmission. In cooperative tests carried on by the Bureau of Animal Industry and the Bureau of Entomology and Plant Quarantine[16] yellow-fever mosquitoes when fed on healthy guinea pigs produced the disease 7 to 74 days after being engorged on an infected animal.

The comparatively low incidence of the disease among horses kept in stables at night and among those which are sprayed with insect-killing or repelling materials has been noted by several authors, and fits in with the mosquito theory.

No species of mosquito has a distribution and a seasonal prevalence that coincide with the incidence of the disease. However, there is no reason to attach responsibility to a single species when several have been shown to be effective vectors. *Aedes vexans* is the most widely distributed of the proved vectors, and it is often very abundant and attacks horses and people viciously late in summer, when the peak of the disease usually occurs. Its presence in large numbers, however, is by no means restricted to late summer and fall. *A. dorsalis* is an extremely abundant species over much of the area in the central and western States where the disease has been prevalent. *A. nigromaculis* is much more restricted in distribution, being confined to the plains and irrigated areas from Texas to Canada and New Mexico to Idaho. It is a severe biter and at times becomes abundant. *A.*

[16] Journ. Amer. Vet. Med. Assoc. **88** (n.s. **41**): 363–374. 1936.

sollicitans, as has been indicated, is often extremely abundant at the peak of an epizootic of the eastern strain and largely covers the area where that type is prevalent. The disease has been less prevalent in the area where the southern salt-marsh mosquito (*A. taeniorhynchus*) is abundant, and *A. sollicitans* is usually present along with that species. *A. aegypti* might play an important part in the transmission of the malady in the South, but this mosquito does not occur where the disease has been most prevalent. Since only one positive case of transmission of the eastern virus by this species occurred out of many attempts, it may be assumed that *Aedes aegypti* is not likely to be of importance as a vector, except in the South where the western virus is present, notably in Texas and Oklahoma. The reported lower incidence of the disease among stabled horses does not fit in with the building-inhabiting tendencies of this mosquito.

There is obviously much to be learned about this dangerous malady and the relation of insects to its transmission. However, while this information is being sought it seems logical to take action against mosquitoes and as far as practical to protect man and horses from mosquitoes and other biting insects, especially when the disease is present in the region.

ENTOMOLOGY.—*Nomenclatorial notes on Corrodentia, with descriptions of two new species of* Archipsocus.[1] ASHLEY BUELL GURNEY, U. S. Bureau of Entomology and Plant Quarantine. (Communicated by C. F. W. MUESEBECK.)

For the past several years Dr. T. E. Snyder, of the Division of Forest Insect Investigations, Bureau of Entomology and Plant Quarantine, United States Department of Agriculture, has noticed unsightly psocid webs on the trunks of trees at New Orleans, La. The psocid responsible for the webs belongs to a genus hitherto known, among living species, only in tropical regions, and probably it is an established adventive in this country. As the species does not seem to agree with the description of any previously known species, it is here described as new. A second new species of *Archipsocus*, collected in Panama by James Zetek, is also described.

The opportunity is taken to present various notes on the nomenclature of Corrodentia. While certain of these matters may remain open to debate until definitely settled by the International Commission on Zoological Nomenclature, it seems worth while to place the facts on record.

[1] Received May 28, 1939.