

ENZOOTIC EASTERN ENCEPHALITIS ACTIVITY IN MASSACHUSETTS¹

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Outbreaks of eastern encephalitis (EE) in Massachusetts that have affected humans and horses occurred during 1938, 1955, and 1956. In the 1938 outbreak, 38 humans and 248 horses were stricken; 4 humans and 44 horses were infected in 1955; in 1956, there were 12 human and 41 horse cases (Feemster, 1938; Feemster *et al.*, 1958). This sporadic occurrence of human and equine outbreaks might suggest the absence of EE virus from Massachusetts during interepidemic periods, but both virus and antibodies for EE were detected in wild birds in 1953, when no human or equine cases were reported (Feemster *et al.*, 1958). Subsequent stud-

ies during 1957, 1958, and 1959 also revealed the presence of EE antibodies in juvenile wild birds and resident domestic chicken flocks, even though there was no evidence of clinical disease in humans or horses (Hayes *et al.*, in manuscript). Thus, it appears that EE virus remains active in enzootic foci in Massachusetts during interepidemic periods. The objective of the present study was to measure the level of EE virus activity in an enzootic swamp focus as compared with nearby upland habitats.

METHODS AND MATERIALS. EE virus transmission indices were obtained by maintaining a chicken flock of approximately 50 birds at each of the study sites near Taunton, Massachusetts, during the period of June 22 to October 16, 1959. The birds were kept in the type of standard sentinel shed described by Rainey *et al.* (1960). The chickens were about 11 weeks old when placed in the field. Pre-bleeding of a random sample from

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the same series indicated that none previously had been infected with EE virus. The birds were bled at intervals during the season, and the percent showing hemagglutination-inhibition antibodies (Casals and Brown, 1954) at the end of the season provided an index of EE transmission for each of the study sites. The blood was obtained from the wing veins of the chickens, and the specimens of serum were stored at -65°C . until tested.

One sentinel flock was placed at each of four study sites. Site 1 was near the center of a large fresh-water swamp known as "Pine Swamp," in which there was heavy breeding of *Culiseta melanura*, the primary enzootic vector of EE. The dominant trees in the swamp are red maple (*Acer rubrum*) and white cedar (*Chamaecyparis thyoides*). Sites 2, 3, and 4 were east of Pine Swamp, and no other swamp was closer to each of the three sites than was Pine Swamp. Site 2 was located 0.1 mile from the edge of Pine Swamp, and the dominant trees were black spruce (*Picea mariana*) and white pine (*Pinus strobus*). Site 3 was located among black cherry (*Prunus serotina*) and red maple (*Acer rubrum*) trees 0.4 mile from the swamp. Site 4 was 2.0 miles from Pine Swamp in a hillside habitat characterized by black oak (*Quercus velutina*), red maple (*Acer rubrum*), and white pine (*Pinus strobus*).

RESULTS. The results of hemagglutination-inhibition antibody (HAI) tests with sera collected from each of the sentinel flocks at the end of the season are summarized in Table 1. The transmission rates (HAI antibody rates) at sites 2, 3, and 4 were all about the same (35, 36,

and 35 percent, respectively), but the rate at site 1 was 65 percent—almost twice as high as at the other sites. This difference is highly significant when analyzed by fourfold contingency table tests (Mainland *et al.*, 1956). Since eastern and western viruses both belong to Group A and may cross react in HAI tests, the results can only be taken as evidence that HAI antibodies against Group A antigens were present. However, mouse neutralization tests with a selected sample of the positives indicated that most of the immunity was undoubtedly due to EE rather than WE (western encephalitis) infection.

DISCUSSION. No human or equine cases of encephalitis were reported from Massachusetts during 1959. The results of these studies show conclusively, therefore, that high levels of virus activity may occur in enzootic swamp foci during non-epidemic years. They also indicate that this activity may extend considerable distances beyond the enzootic foci into inhabited areas without evidence of clinical disease in the human or equine populations. Long term observations are being continued in the Pine Swamp area to determine the consistency of these patterns of EE transmission and the various ecological factors that may influence them.

SUMMARY. From June 22 to October 16, 1959, four flocks of sentinel chickens were maintained near Taunton, Massachusetts, in the vicinity of a swamp which produced large numbers of *Culiseta melanura*. Serum specimens collected from the sentinel chickens at the end of the season and tested for hemagglutination-inhibition antibodies against EE antigen revealed a high transmission rate (65 percent positive) near the center of the swamp, and lower rates (about 35 percent positive) at the other sites located up to two miles from the edge of the swamp. Since no human or equine cases of arthropod-borne encephalitis were reported during 1959, it appears that high levels of enzootic transmission may occur in Massachusetts without involving horses or humans.

TABLE 1.—HAI antibody rates in chicken sentinel flocks maintained in Pine Swamp area, Taunton, Massachusetts, during the 1959 transmission season

Flock location	Miles from swamp	No. sera tested	Percent with EE antibodies
Site 1	0	37	65
Site 2	0.1	37	35
Site 3	0.4	42	36
Site 4	2.0	43	35

References Cited

CASALS, J., and BROWN, L. V. 1954. Hemagglutination with arthropod-borne viruses. *Jour. Expt. Med.*, 99:429-49.

FEEFSTER, R. F. 1938. Outbreak of encephalitis in man due to the eastern virus of equine encephalomyelitis. *Amer. Jour. Publ. Hlth.*, 28:1403-10.

FEEFSTER, R. F., WHEELER, R. E., DANIELS, J. B., ROSE, H. D., SCHAEFFER, M., KISSLING, R. E., HAYES, R. O., ALEXANDER, E. R., and MURRAY, W. A. 1958. Field and laboratory studies on equine encephalitis. *New England Jour. Med.*, 259:107-13.

HAYES, R. O., DANIELS, J. B., ANDERSON, K. S., PARSONS, M. A., LAMOTTE, L. C., and MAXFIELD, H. K. 1960. Detection of eastern encephalitis virus antibodies in wild and domestic birds in Massachusetts, 1957-1959. (In manuscript.)

MAINLAND, D., HERRERA, L., and SUTCLIFFE, M. I. 1956. Statistical tables for use with binomial samples-contingency tests, confidence limits, and sample size estimates. 83 pp. New York Univ. College Med., New York, N. Y.

RAINEY, M. B., WARREN, G. V., HESS, A. D., and BLACKMORE, J. S. 1960. A sentinel chicken shed and mosquito trap for use in encephalitis field studies. Unpubl. ms.