THE 1958 ENCEPHALITIS OUTBREAK IN NORTHERN UTAH. 2. INFECTION RATES IN BIRDS, MAMMALS, AND MOSOUITOES

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During an outbreak of encephalitis in northern Utah in the late summer of 1958, efforts were made to determine infection rates in birds, mammals, and mosquitoes from August 15 to September 9. The results of this study are reported here.

Figure 1 shows the area involved from Preston, Idaho, to Payson, Utah, where collections of mosquitoes or blood specimens were obtained during the study. Intensive work was limited to the area between Preston, Idaho, and Brigham City, Utah, but a few blood specimens were collected much further south at Richfield, Utah

METHODS

Prevalence of infection in mosquitoes was determined by making collections of mosquitoes at various localities, sorting them into species, and testing for virus infection by inoculation in appropriate laboratory animals. The agents isolated were identified by the standard neutralization test.

In addition to virus isolations, serological techniques were employed to determine the incidence of infection in birds, mammals, and humans. The tests used were hemagglutination-inhibition (HI), complementfixation (CF), and virus neutralization. The HI and neutralization tests detect both recent and past infections; the CF test, recent infection.

RESULTS

Only 7 of 80 pools of 3,741 Culex tarsalis

Incidence of Infection in Mosquitoes.

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collected from the entire study area yielded western equine encephalitis (WEE) virus and none yielded St. Louis encephalitis (SLE) virus. The over-all infection rate in C. tarsalis was at least 1 in 534 (0.2%).

Data on incidence of infection in C. tarsalis are presented in Table 1. Five of 26 pools of 1,304 C. tarsalis collected at Bothwell were positive for WEE virus, an infection rate of at least 1 in 261 (0.4%). The infection rate at Wellsville was at least 1 in 150 (0.7%) and at Brigham City at least 1 in 1,115 (0.09%).

In addition, 3 Culiseta inornata collected at Fielding, 100 C. inornata collected at Wellsville, and 50 Culex pipiens collected at Bothwell were tested for virus. No isolations were made from these mosquitoes.

ISOLATION OF WEE VIRUS FROM MAN. An agent was isolated from brain tissue, obtained upon autopsy of a 2-month-old infant from Fielding, and was identified as WEE virus.

Serology. The results of testing human, horse, and chicken sera for the presence of HI, CF, or neutralizing antibodies are shown in Table 2. The sera examined in one test are not necessarily the same sera examined in another test. Thirty-two human sera, mostly from close associates of clinical encephalitis patients, were collected from an area extending from Smithfield to Payson, Utah. Three sera (9%) had neutralizing antibodies but none of the sera showed CF antibodies. The positive sera were from Smithfield and Tremonton. None of the sera was tested for HI antibodies.

Horse sera were tested for HI, CF, and neutralizing antibodies. Eighteen of 20 (90%) had HI antibodies, 37 of 67 (55%) had CF antibodies, and 62 of 71 (87%) had neutralizing antibodies. The

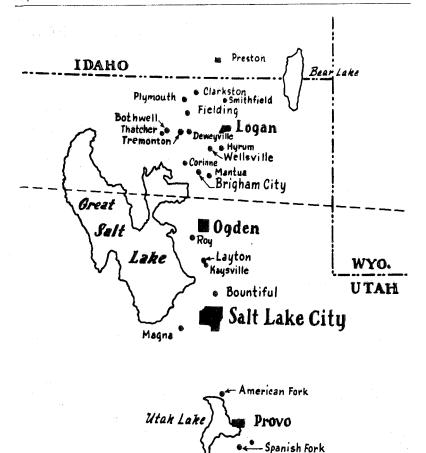


Fig. 1.—Northern Utah, showing area involved in encephalitis outbreak of 1958.

Payson

positive sera were collected in an area extending from Plymouth to Richfield.

Approx. 16-5 Mi.

Sera of chickens less than a year old were collected in a much more limited area. The incidence of HI antibodies was 28 in 271 sera (10%) and of neutralizing antibodies 8 in 50 (16%). These sera were not tested for CF antibodies. The infection rate, as shown by HI antibodies, was 24% at Bothwell (21 of 88), but it was only 2% at Preston, Idaho (1 of 49), 4% at Clarkston (3 of 84), and 6% (3 of 50) in the Wellsville-Hyrum area.

Sera from one meadowlark and one sparrow collected at Mantua and from 25 English sparrows collected at Bothwell were tested for neutralizing antibodies with negative results.

Discussion

The isolation of WEE virus from mosquitoes and the serological evidence of recent infection with WEE virus in horses, and in chickens less than a year old, and the isolation of the virus from the brain

TABLE 1.—Summary of virus isolation from *Culex tarsalis* mosquito pools collected in northern Utah, 1958

Location	Number of mosquitoes tested	Number of pools tested	Number of pools positive for WEE virus
Thatcher	100	2	0
Bothwell	1304	26	5
Tremonton	14	2	0
Fielding	28	I	. 0
Deweyville	100	2	0
Wellsville	150	3	I
Corinne	311	5	0
Mantua	11	I	0
Brigham City	1,115	25	İ
Roy	50	I	0
Kaysville	100	2	0
Bountiful	350	7	0
Magna	50	1	0
Spanish Fork	58	2	0
=			_
Total	374 I	8o	7

TABLE 2.—Antibody titers for WEE virus, Idaho and Utah, 1958

Blood source	HI		C-F			Neutralization			
	No. tested	No. pos.	Percent pos.	No. tested	No. pos.	Percent pos.	No. tested	No. pos.	Percent pos.
Human Horse Chicken		18 28	90 10	32 67	0 37 —	0 55	32 71 50	3 62 8	9 87 16

of a child demonstrate the nature of the disease which was present in the area in 1958.

The field data indicate a very low infection rate in the C. tarsalis population in northern Utah. It may be significant that the mosquito infection rate at Bothwell, in the center of the outbreak, was 1:261 whereas at Brigham City it was only 1:1,115. All of the mosquito collections were made between August 15 and September 9, and thus, it seems likely that the incidence of infection detected in the mosquito population may not have been representative of that prior to, or during the peak of, the outbreak. Since the mosquitoes collected may have emerged late and, therefore, had little opportunity to acquire infection, the observed infection rate may be lower than that present earlier in the outbreak.

HI and neutralizing antibodies are known to persist for a considerable length of time following virus infection, while CF antibodies, for the most part, persist usually for less than a year. Of the 32 human sera (Table 2), mostly from close associates of clinical encephalitis patients, 3 were shown to have WEE neutralizing antibodies. Since none of the 32 sera had CF antibodies, the 3 which had neutralizing antibodies probably were from individuals who had been infected in previous years. As expected, this suggests that there was very little, if any, inapparent infection in persons associated with clinically ill patients in this outbreak.

Examination of horse sera was useful for determining the geographic extent and incidence of infection during the outbreak. The data on antibodies in horses (Table 2) indicate either previous or recent exposure

to WEE virus. Approximately 90% of the horses had neutralizing or HI antibodies which might be due either to past or to recent infection. The CF test indicated a recent infection in approximately 55% of horses examined.

Data from chicken sera (Table show evidence of recent infection in chickens since all of the chickens tested were less than I year old. Two to 24% of chickens in different areas had HI antibodies and about 16% in the entire area had neutralizing antibodies against WEE. Results of HI tests on sera collected in 1957 from the same areas and from approximately the same number of chickens showed no significant difference in the percentage of chickens showing antibodies, though the rates were slightly higher in 1958. Infection rates obtained from testing these chicken sera collected in 1957 and 1958 for the presence of HI antibodies against St. Louis virus were o% and approximately 6 percent, respectively.

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Summary

During the 1958 outbreak of encephalitis in northern Utah, western equine encephalitis virus was isolated from human tissue and from mosquitoes. There was no evidence of recent infection in humans associated with clinical cases. Serological tests indicated recent exposure of horse and chicken populations to the virus. The few wild bird sera examined did not show evidence of infection. The highest infection rates in both mosquitoes and chickens occurred in the center of the human outbreak.

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