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AVIAN HOSTS OF ST. LOUIS ENCEPHALITIS VIRUS

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DISTRIBUTION AND HUMAN DISEASE

St. Louis encephalitis (SLE) virus normally infects birds and is transmitted between birds by mosquito vectors. Thus it is an arthropod-borne virus (arbovirus). SLE virus is found throughout the Americas, and human disease has been reported from the northern part of the hemisphere. Human epidemics have been reported only from northern Mexico (Gonzalez Cortes, et al. 1975), the United States, and southern Canada (CDC 1977). Other viruses closely related to SLE (dengue, yellow fever, and Rocio viruses) cause epidemics in Central and South America. In the United States, SLE is the most important mosquito-borne disease of humans in terms of the number of cases and fatalities, and birds are the usual source of SLE virus for mosquitoes that infect humans.

Human cases have been reported from all regions of the U.S. and from most of the states, but epidemics occur predominantly in the Mississippi and Ohio River watersheds, Texas, and Florida. The intensity of human cases oscillates dramatically over a 6-10 year period, with epidemic levels of disease spanning several years. The last peak of human SLE occurred in 1975; and only a few sporadic cases have been reported in the past three years (Vector-Borne Diseases Division 1977a, 1978a, 1979a). The infection in humans has a spectrum of responses: no apparent illness; mild illness with slight fever; febrile headache, often with nausea and vomiting; aseptic meningitis with sudden onset of fever and neck stiffness; and encephalitis with fever and symptoms of disorientation, confusion, stupor or coma, tremors of the face or hands, neck rigidity with or without paralysis, and occasionally death, particularly in individuals over 55 years of age (Brinker and Monath 1980). Human infection and disease are a result of an extension of SLE virus transmission out of the natural bird-mosquito cycle. Humans are dead-end hosts for the virus and therefore do not contribute to additional transmission or maintenance.

MAINTENANCE SYSTEMS AND TRANSMISSION CYCLES

SLE virus is maintained primarily in bird populations and *Culex* mosquito populations within natural foci in the United States. Mammals are also important hosts for SLE virus in South and Central America, and possibly Florida, but birds appear to be the most important host of SLE in most of North America. The SLE maintenance system involves complex interrelationships among vertebrate hosts, vectors, virus, and environmental factors. The components of this system, except the virus, could fluctuate independently, and some may influence the fluctuation of others. Viral maintenance depends upon the ecological overlap in time and space of hosts and vectors. The size, availability and susceptibility of the host and vector populations greatly influence the size of the virus population and the rate of transmission. Permanent SLE foci in temperate climates depend upon regular and dependable transmission among vertebrate hosts by mosquitoes during the summer months and upon some mechanism to allow the virus to survive during the winter when continuous transmission does not occur because of an interruption of mosquito breeding and activity.

No definitive mechanism is known for the winter survival of the virus when it is not actively being transmitted between vertebrates by mosquito vectors. Numerous theories for the “overwintering” mechanism have been proposed. Annual reintroduction of SLE virus in the spring by migratory birds has been investigated with negative results (Calisher et al. 1971, 1974). This theory of viral introduction in the spring to locally breeding mosquitoes in temperate foci probably does not occur for two reasons. First, it would require permanent SLE foci in Central America or northern South America for the birds to become infected during the northern migration. These foci have not been found. Second, the viremic period in infected birds (presence of virus in the peripheral blood) is shorter than the period of time it takes most bird species to complete their migration north. By the time the birds arrive at their northern destination, their viremia would have

disappeared, and thus no virus would be available to infect local mosquitoes. Another mechanism suggested for winter virus survival is chronic, latent infections In vertebrate hosts. The virus remains dormant within the host during the winter months; and after a relapse in the spring, the virus is recirculated in the peripheral blood. Emerging mosquitoes then become Infected by feeding upon this chronically infected host and subsequently transmit the virus to susceptible hosts initiating summer transmission cycles. Though this mechanism has not been demonstrated for SLE virus, except possibly in bats (Allen et. al. 1970), it deserves further evaluation.

The only evidence of overwintering mechanisms for SLE has been the recovery in the spring of adult female mosquitoes infected with SLE virus that had survived the winter months (Bailey et al. 1978). In addition, recent experimental Information suggests that transovarial transmission of SLE virus from infected adult mosquitoes to their progeny may occur (D. B. Francy, *personal communication).* Both of these mechanisms would allow adult female mosquitoes infected with SLE virus to be available in the spring to initiate transmission cycles in vertebrate host populations.

Summer transmission cycles of SLE in the U.S. have been described as a bird-*Culex tarsalis* mosquito cycle in the western states and a bird-*Culex piplens* complex mosquito cycle in the Ohio and Mississippi River watersheds. *C. tarsalis* breeds predominantly in agricultural irrigation water and consequently, many human cases occur in rural areas. The house finch *(Carpodacus mexicanus)*, mourning dove *(Zenaidura macroura),* tricolored blackbird *(Agelaius tricolor),* Brewer's blackbird *(Euphagus cyanocephalus),* and house sparrow *(Passer domesticus)* are the most important avian hosts (McLean and Bowen, 1980) in the western SLE cycle. The *C. pipiens* complex occurs predominantly in urban environments where there are ample amounts of polluted water conducive to the breeding of these two peridomestic mosquito sub-species (C. p. *pipiens* in the northern states and *C. p. quinquefasciatus* in the southern states). Bird species which are involved with urban transmission cycles in the central and eastern regions are peridomestic species such as house sparrows, pigeons *(Columbia livia),* and other species closely associated with urban-suburban neighborhoods such as blue jays *(Cyanocitta cristata),* robins *(Turdus migratorius),* cardinals *(Richmondena cardinalis),* mourning doves, and mockingbirds *(Mimus polyglottos)* (McLean and Bowen 1980). Rural transmission cycles most likely occur in the same regions and involve other vector mosquito species (such as *C. salinarius* and *C. restuans),* which thrive in woodland habitats, and possibly additional wild bird species (e.g. catbird *(Dumetella carolinensis),* woodthrush *(Hylocichla mustelina),* bobwhite *(Colinus virginianus),* and others. In addition to involving birds, the rural transmission cycle in Florida probably involves mammals and *C. nigripalpus* mosquito vectors which breed primarily in fresh water swamps. Important bird species in Florida are the pigeon, mourning dove, blue jay, cardinal, and house sparrow (Jennings 1969). The raccoon *(Procyon lotor)* and cotton rat *(Sigmodon hispidus)* may be important mammal hosts for SLE in some areas of Florida (F. M. Wellings, *personal communication).*

FACTORS DETERMINING BIRD SPECIES ROLE AS HOST

Many bird species are potential hosts for SLE virus; however, bird species are not equal as hosts. The effectiveness of a bird species as host for SLE depends upon a number of factors. The bird species must be susceptible to infection with SLE virus, as determined by virus isolation and specific antibody from field studies of natural infection. Their susceptibility can be further evaluated experimentally by investigating the magnitude and duration of viremias. Experimental studies are necessary to show that a bird species determined susceptible from field studies can in fact be an effective host in infecting mosquitoes due to a sufficiently high and long lasting viremia.

The SLE infection should not adversely affect survival or reproductive potential of host bird species. Species must be attractive to and tolerant of mosquito feeding. Bird seasonal activities and abundance must coincide properly in time and space with those of the mosquito vector to insure ecological associaton between hosts and vectors. Other factors that affect the distribution and amplitude of SLE virus in bird populations are variation in vector species susceptibility and transmission efficiency, and variation in the pathogenecity of SLE viral strains.

NATURAL SLE INFECTION

Accumulated information on the natural infection of birds with SLE virus is presented in Tables 1 and 2. Some orders and families have not been adequately sampled or comprise such a small proportion of the avifauna that no meaningful discussion about their involvement as hosts for SLE can be made. Passeriformes, Columbiformes, and Galliformes have been sampled most often and have high virus or antibody prevalences (% positive). No virus isolations have been reported from the Galliformes. Most samples are from domestic chickens *(Gallus gallus).* Aquatic birds (Pelicaniformes, Anseriformes, Ciconiiformes, and Charadriiformes) appear to be frequently exposed to SLE virus, but only domesticated Anseriformes occur in close contact with man. The high prevalence of virus in Pelicaniformes was due to isolations from sick cormorants in Panama (McLean and Bowen, 1980). Columbiformes and Passeriformes are most frequently collected by investigators, perhaps because they comprise the majority of North American birds and live in close contact with man. Of the 27 families of Passeriformes that occur in North America, 22 have been tested for SLE infection (Table 2). Corvidae (jays and crows), Mimidae (thrashers and mockingbird), Turdidae (thrushes), Ploceidae (house sparrow), and Thrauplidae (tanagers) have high virus and antibody prevaiences of the families that have been adequately sampled. House sparrows have been sampled most often and show a low antibody prevalence compared to the relatively high prevalence of SLE virus, but only for house sparrows have there been substantial numbers of nestling birds tested for virus. This is significant because in general arboviruses are more often isolated from nestlings than adults.

During investigations of avian populations associated with human SLE epidemics (Table 3), more virus isolations were again made from house sparrows than from any other bird species. It should be noted that many of these isolations were from nestling sparrows. Domestic birds (chickens, ducks, and geese) had the highest antibody prevalences, but only one virus isolation was obtained. Therefore these birds probably do not contribute much to SLE transmission but are good Indicators of SLE viral activity in the urban environments. Pigeons and blue jays appear to be major amplifying hosts for SLE virus during urban epidemics. Robins had the highest antibody prevalence listed, though the number tested is small. Most of these positives were found during the largest urban epidemic in Chicago in 1975 (Vector-Borne Diseases Division 1976a) where robins appeared to be one of the primary amplifying hosts. Catbirds and cardinals had lower antibody and virus prevalences than species mentioned above but adequate enough to consider them important hosts in urban environments. House sparrows had a similar SLE antibody prevalence to cardinals but have been sampled more than any other species during epidemics. In some cities, house sparrows comprised the major portion of the avian population and may have contributed most to SLE transmission (Lord et al. 1974). An additional factor that increases the potential of house sparrows as SLE hosts in urban settings is that they commonly live in close association with humans and peridomestic mosquito vectors.

EXPERIMENTAL INFECTION

Of the 13 bird species experimentally inoculated with SLE virus, the blue jay, white-crowned sparrow *(Zonotrichia leucohprys),* tri-colored blackbird, house finch, duck, and chicken appeared to be most susceptible as judged by the low dose of virus that produced viremic responses in 100% of the birds inoculated (Table 4.). The house sparrow, starling *(Sturnus vulgaris),* and mourning dove were intermediate in their response, and the brown-headed cowbird *(Molothrus ater)* and red-winged blackbird *(Agelaius phoeniceus)* were relatively resistant to experimental viral infection. For a few species like the house sparrow, brown-headed cowbird, red-winged blackbird, and tri-colored blackbird, the percent of birds that were antibody positive was less than the percent viremic.

The magnitude of viremic responses varies among species. Some species like the chicken and domestic duck (Hammon et al. 1946) circulated only small amounts of virus ($10^{0.7}$ to $10^{2.7}$ 50% lethal dose [LD_{50}]/ml) while other species like the white-crowned sparrow (Hammon et al. 1951) circulated large quantities of virus (trace to $10^{5.6}$ LD_{50}/ml).

Infection thresholds for mosquitoes feeding upon viremic birds varies among mosquito species and among local populations of a single mosquito species. Magnitude and duration (days) of avian viremias, however, favorably affect the efficiency and probability of infecting mosquitoes that feed upon viremic hosts.

Age, for some species, also influences the magnitude of viremia. Young birds, especially newly hatched precocial birds and nestling altricial birds, circulate more virus

when they are infected with SLE virus than older birds. This point is illustrated by Table 5, which shows the magnitude of viremias decreased with increasing age of chickens inoculated with identical doses of SLE virus (Sudia and Chamberlain 1959).

The quantity of SLE virus inoculated can influence avian viremic responses (Table 6). Prevalence of infection generally increases with increasing doses of virus inoculated until a maximum dose is reached, at which time increased dosage will not increase the prevalence of infection. Bird species differ in the virus dose required to reach this maximum threshold. In addition, a minimum viral dose is required before a detectable viremic response is observed. Mourning doves and chickens appeared to be the most susceptible bird species tested, since 100% became viremic at a relatively low virus dose (100 LD_{50}); whereas four times that dose was required for house sparrows and 100 times for red-winged blackbirds to produce 100% viremias. Quantititative results should, however, be interpreted with caution because different SLE viral strains and methods were used in the experiments in Table 6.

SURVEILLANCE OF SLE

Because of strong correlations between SLE infections in bird populations and reported human SLE cases (Holden et al. 1973), birds have been used as sentinels by health officials to determine the annual nature of the SLE virus activity. A number of studies have demonstrated the value of chickens as sentinels (Reeves and Hammon 1962 and Holden et al. 1973), but cost and lack of mobility (detecting virus activity only in the immediate vicinity of a holding facility) has limited their use. Wild birds living in close association with human populations have more recently been used as sentinels (Lord et al. 1974), because several species are equally as important hosts and because they become exposed to infected mosquitoes over a larger area than stationary birds. Conversion from negative to positive SLE antibody is routinely used to detect virus activity in stationary sentinels like chickens. Changes in the prevalence of SLE antibody must be used in free-ranging, wild bird sentinels, or the presence of antibody in immature wild birds because free-ranging birds cannot be sampled at regular intervals like captive birds. Virus isolation from nestling house sparrows was effectively used in west Texas to monitor SLE activity (Holden et al. 1973), but this technique cannot be used by most health departments because of the expense and elaborate methods involved in testing for virus.

Most health departments use the hemagglutination-inhibition (HI) test to detect SLE antibody in sentinel birds because of ease, rapidity, and low cost (LaMotte et al. 1967). Results from this test alone must be interpreted with caution, because nonspecific inhibitors and antibodies to closely related viruses could give false positives.

Avian monitoring systems for SLE have been developed for a variety of purposes and thus may have different scopes of operation. Regional, state, county, and city surveillance systems are currently in operation in many midwestern and eastern states. A state system may require only qualitative information about the distribution and annual appearance of SLE, whereas a city health department may need quantitative information about the level of virus activity and temporal data which would provide early warning before epidemics. Both types of information would be used to direct mosquito control activities that would reduce the risk to the human population.

Serologic results from wild birds sampled during 1976-78 in an 11 -state region of eastern United States show relatively low average prevalence of SLE antibody (Figure 1). The low level of SLE virus activity in the U.S. was also apparent from the few human cases that occurred. HI antibody prevalence did, however, gradually increase as the summer progressed. This correlated positively with the usual late summer (August and September) increase of reported human SLE cases (Monath 1980). The peak in antibodies during May is unexplained but could indicate an early mosquito transmission cycle or a relapse in previously infected birds.

BIRD CONTROL AND HUMAN DISEASE

Control of bird populations to prevent human epidemics of SLE has been suggested. Emergency measures for reducing populations of urban species in order to halt a human epidemic would not be effective because they would occur too late to reduce the risk to humans (bird epizootics usually preceded human epidemics by a few weeks). In fact, such measures actually could increase the risk of human infection by reducing the availability of normal bird hosts on which the infected mosquito would feed. Mosquitoes might then feed on humans more frequently.

A permanent reduction in the population size of peridomestic species like house sparrows and pigeons is feasible, could have a negative effect on the potential for SLE epizootics to occur in urban bird populations, and thus could decrease the chances of human epidemics. However, because other desirable bird species that are good SLE hosts like robins, cardinals, and blue jays would still be abundant in urban environments, it is unlikely that all human cases could be prevented by reducing pest bird populations.

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TABLE 1. Orders of birds found naturally infected with St. Louis enchalitis virus in Central and North America.[1]

	HI Antibody		N Antibody		Virus	
	No. Test.	% Pos.	No. Test.	% Pos.	No. Test	% Pos.
Tinamiformes (Tinamous)	---	---	2	0.0	---	---
Pelicaniformes (Pelicans)	83	22.9	9	22.2	88	4.54
Anseriformes (Waterfowl)	184	45.1	1527	6.7	54	1.85
Falconiformes (Hawks)	5	40.0	16	18.8	22	0.00
Galliformes (Chickens)	15778	13.1	3817	13.2	1080	0.00
Ciconiiformes (Herons)	914	10.6	120	5.8	1167	0.17
Gruiformes (Rails)	3	33.3	---	---	2	0.00
Charadriiformes (Gulls)	21	23.8	29	13.8	38	0.00
Columbiformes (Doves)	6324	10.0	5194	4.6	3090	0.29
Cuculiformes (Cuckoos)	135	18.5	2	0.0	181	0.00
Strigiformes (Owls)	17	11.8	33	30.3	55	0.00
Caprimulgiformes (Goatsuckers)	6	16.7	6	16.7	6	0.00
Apodiformes (Swifts)	3	0.0	---	---	229	0.44
Psittaciformes (Parrots)	---	---	4	0.0	---	---
Coraciiformes (Kingfishers)	---	---	19	0.0	10	0.00
Piciformes (Woodpeckers)	167	10.8	51	25.5	396	0.25
Passeriformes (Perching birds)	18534	7.3	5044	12.3	25524	0.15
	Σ= 42074	$\bar{X}$= 10.2	Σ= 15863	$\bar{X}$= 9.5	Σ= 32220	$\bar{X}$= 0.17

[1]Data derived from McLean and Bowen (1980).

TABLE 2. Families of Passeriformes birds found naturally infected with SLE Virus.[1]

Family	HI Antibody		N Antibody		SLE Virus	
	No. Test.	% Pos.	No. Test.	% Pos.	No. Test.	% Pos.
Pipridae (Manakins)	---	---	---	---	3	100[2]
Tyrannidae (Flycatchers)	285	13.3	95	2.1	708	0.00
Alaudidae (Larks)	90	5.6	---	---	---	---
Hirundinidae (Swallows)	95	1.1	99	16.2	561	0.00
Corvidae (Jays and Crows)	582	10.5	299	40.8	1159	0.17
Paridae (Chickadees)	106	14.2	25	16.0	426	0.00
Sittidae (Nuthatches)	15	0.0	3	0.0	9	0.00
Certhiidae (Creepers)	---	---	---	---	14	0.00
Troglodytidae (Wrens)	79	6.3	19	31.6	352	0.00
Mimidae (Mockingbird and Thrasher)	1803	15.0	764	20.1	2037	0.15
Turdidae (Thrushes)	1551	11.0	319	21.6	1675	0.18
Sylviidae (Kinglets)	---	---	---	---	160	0.00
Motacillidae (Pipits)	---	---	---	---	5	0.00
Laniidae (Shrikes)	28	5.5	14	21.4	26	0.00
Sturnidae (Starlings)	113	1.8	76	12.5	121	0.00
Coerebidae (Honeycreepers)	21	14.3	---	---	15	0.00
Vireonidae (Vireos)	795	7.3	20	10.0	1084	0.00
Parulidae (Warblers)	810	3.3	132	3.8	1507	0.00
Ploceidae (House Sparrow)	6975	5.9	2614	8.8	10123	0.28
Icteridae (Blackbirds)	2667	4.0	510	7.1	2455	0.00
Thraupidae (Tanagers)	598	12.7	5	0.00	432	0.23
Fringillidae (Sparrows and Finches)	1713	4.8	751	14.6	2751	0.04

[1]Data derived from McLean and Bowen (1983).
[2]Only the number of SLE virus isolations were reported and not the number tested.

Table 3. Virologic and serologic results for SLE virus in avian populations investigated during human epidemics in urban environments.[1]

Species	No. of Investigations	No. Tested	No. Virus Positive	No. Antibody Positive[2]	% Antibody Positive
House Sparrow	10	2260	7[3]	321	14.2
Pigeon	8	914	3	241	26.4
Chicken	7	513	0	147	28.7
Blue Jay	5	285	2	66	23.2
Mockingbird	2	182	1	18	9.9
Cardinal	5	177	1	28	15.8
Common Grackle	3	139	0	9	6.5
Robin	3	81	1	44	54.3
Catbird	2	44	1	9	20.5
Other wild birds	5	523	2[4]	39	7.5
Other domestic birds	3	111	1[4]	50	45.0

[1]Data derived from McLean and Bowen (1980).
[2]Data from both the neutralizing and hemagglutination-inhibition tests for antibody were combined.
[3]Includes 3 virus isolations from nestlings.
[4]Virus isolations were from the yellow-shafted flicker, chimney swift and domestic goose.

Table 4. Data on experimental infection of birds with SLE virus.

Bird Species	Virus Dose Inoculated[1]	No. Birds	Age	% Viremic	% Antibody Positive	Reference
Domestic Duck	100	6	3-6 months	100	NT[2]	Hammon et al. 1946
Domestic Chicken	100	1*	3-6 months	100	NT	Hammon et al. 1946
	200-1,000	31	3-10 months	90	90	LaMotte et al. 1967
Pigeon	10,000	2	Adult	100	100	Chamberlain et al. 1957
Mourning Dove	10-1,000	12	Immature	75	83	Jennings 1969
Mockingbird	100-10,000	17	Adult	94	94	Jennings 1969
Blue Jay	30-300	16	Adult	100	100	Jennings 1969
House Sparrow	1.5-150	15	Adult	47	46	Jennings 1969
	40-400	8	Adult	88	0	Chamberlain et al. 1957
	100-500	6	Adult	67	NT	Hammon et al. 1951
Cowbird	80-100,000	6	Adult	20	17	Chamberlain et al. 1957
Red-winged Blackbird	80-100,000	7	Adult	43	29	Chamberlain et al. 1957
Tri-colored Blackbird	500	4	Adult	100	50	Hammon et al. 1951
House Finch	100	2	Adult	100	NT	Hammon et al. 1951
White-crowned Sparrow	50	14	Adult	100	NT	Hammon et al. 1951
Starling	4000	7	Adult	71	NT	McLean 1972

[1]Number of 50% lethal dose IC in weanling white mice.

[2]NT= not tested or reported.

Table 5. The effect of age on the viremic respose of chickens experimentally inoculated with SLE virus.[1]

Species	Virus Dose[2]	No. Birds	Age (days)	% Viremic	Mean Titer of Viremia[3]	% Antibody Positive[4]
Chicken	10,000	10	0.5	100	4.5	100
	10,000	10	7	100	3.0	100
	10,000	10	14	100	2.9	100
	10,000	10	28	100	2.7	100
	10,000	9	84	67	1.7	100

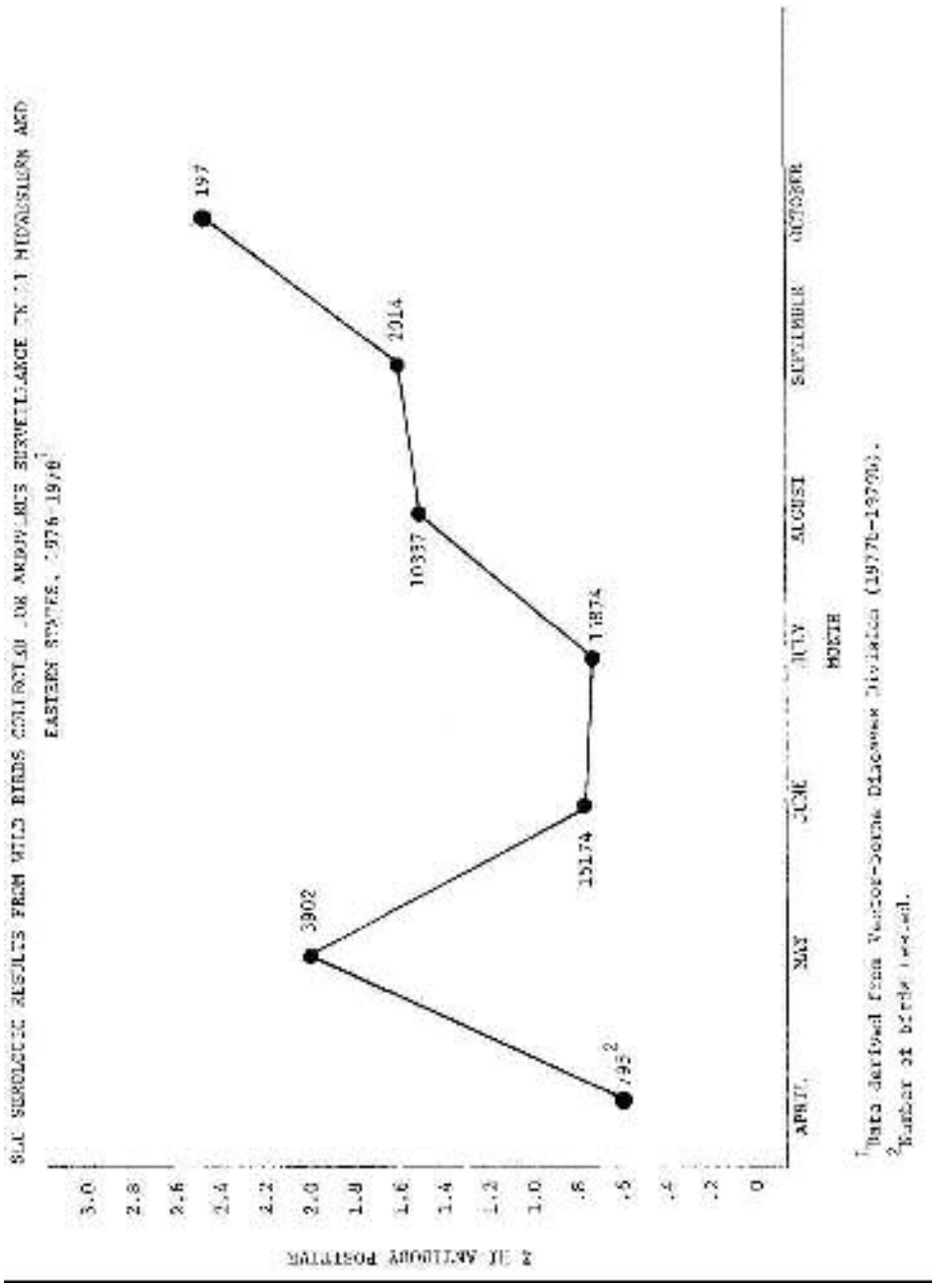
[1]Adapted from Sudia and Chamberlain (1960).
[2]Number of 50% lethal dose IC in weanling white mice.
[3]Viremia expressed as $\log_{10}LD_{50}$ per ml in weanling white mice.
[4]Antibody determined in neutralization tests in 3 week old mice.

Table 6. The effect of various virus doses on the experimental infection of birds with SLE virus

Bird Species	Virus Dose[1]	No. Birds	Age	% Viremic	Mean Viremia[2]	% Antibody Positive	Reference
Chicken	1	5	10 weeks	0	NT	50	Jennings 1976
	10	5	10 weeks	20	NT	50	
	100	5	10 weeks	20	NT	50	
	1	3	5-7 months	100	[illegible]	NT[3]	Hammon et al. 1945
	100	6	5-7 months	100	1.6	NT	
House Sparrow	1.5	9	Adult	40	NT	67	Jennings 1969
	15	5	Adult	40	NT	50	
	150	5	Adult	60	NT	0	
	40	4	Adult	75	2.4	0	Chamberlain et al. 1957
	400	4	Adult	100	2.2	0	
	100	3	Adult	10	NT	NT	Hammon et al. 1951
	500	3	Adult	100	NT	NT	
Mourning Dove	10	4	Immature	25	NT	75	Jennings 1969
	100	4	Immature	100	NT	75	
	1000	4	Immature	100	NT	100	
Cowbird	50	2	Adult	0	—	0	Chamberlain et al. 1957
	500	2	Adult	0	—	0	
	10,000	2	Adult	50	2.4	NT	
	100,000	2	Adult	50	1.5	50	
Red-winged Blackbird	50	2	Adult	0	—	0	Chamberlain et al. 1957
	500	2	Adult	0	—	50	
	10,000	2	Adult	50	1.7	50	
	100,000	1	Adult	100	1.7	0	

[1]Number of 50% lethal doses IC in suckling white mice.
[2]Viremia expressed as log10 LD50 per ml in suckling white mice.
[3]NT = not tested or reported.

Fiaure 1


SLC SEROLOGIC RESULTS FROM WILD BIRDS COLLECTED FOR ARBOVIRUS SURVEILLANCE IN THE MISSISSIPPI AND EASTERN STATES, 1976-1978[1]
% OF ANTIBODY POSITIVES
3.0
2.8
2.6
2.4
2.2
2.0
1.8
1.6
1.4
1.2
1.0
.8
.6
.4
.2
0
APRIL
MAY
JUNE
JULY
AUGUST
SEPTEMBER
OCTOBER
MONTH
197
2014
10337
11874
25174
3902
195[2]
[1]Data derived from Vector-borne Diseases Division (1977b-1978b).
[2]Number of birds tested.