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# ROLE OF PERIDOMESTIC BIRDS IN THE TRANSMISSION OF ST. LOUIS ENCEPHALITIS VIRUS IN SOUTHERN CALIFORNIA

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ABSTRACT: In response to the 1984 St. Louis encephalitis (SLE) epidemic in the Los Angeles Basin of southern California (USA), an investigative program was initiated to evaluate the interactive components of the SLE virus transmission cycle. From 1987 through 1996 (10 yr), 52,589 birds were bled and their sera tested for SLE and western equine encephalomyelitis (WEE) virus antibodies by the hemagglutination inhibition (HAI) test. Eighty-three percent of the birds tested were house finches (*Carpodacus mexicanus*) (48.7%) and house sparrows (*Passer domesticus*) (34.6%); 1.1% of these birds were positive for SLE antibodies. Prevalence of WEE antibodies was negligible. The analysis of 5,481 sera from rock doves (*Columbia livia*) yielded 3.6% SLE positives and 0.4% WEE positives. Collection sites were maintained as study sites when identified as positive bird, mosquito, and SLE virus activity localities; others were abandoned. Serial serum samples from 7,749 banded house sparrows and 9,428 banded house finches from these selected sites demonstrated year-round SLE virus transmission. One location exhibited significant numbers of house finches undergoing annual SLE seroconversion and a number of seroconversion-reversion-reconversion sequences suggesting either viral reinfection from mosquitoes or recrudescence by latent virus. A proportion of both bird species also lived for longer than 1 yr, thus, increasing the possibility of virus carry-over from autumn to spring. Assessment of concurrently collected mosquitoes indicated no correlative association between mosquito populations and SLE seroconversion and reconversion. European house sparrows introduced in the 1800's may have provided a supplemental link to the existing SLE virus enzootic cycle involving endemic house finches. Meteorological factors are reviewed as possible important correlates of SLE epidemics. The house finch/house sparrow serosurveillance system is also evaluated for use as an "Early Warning" indicator of SLE virus activity.

*Key words:* Arbovirus surveillance, epidemiology, house finch, house sparrow, mosquitoes, overwintering, recrudescence, St. Louis encephalitis virus, virus foci.

## INTRODUCTION

During the autumn of 1984, an unexpected St. Louis encephalitis (SLE) epidemic occurred in the greater Los Angeles Basin of southern California (USA). Twenty-four human SLE cases (one fatality) were diagnosed; five of these occurred in Orange County. Analysis of earlier epidemics (Monath, 1980) suggest that there were probably thousands of SLE virus-infected individuals, but inadequate surveillance under evaluated the extent of the Los Angeles epidemic.

The SLE virus is maintained and transmitted in a cycle involving mosquitoes (*Culex* spp.) and birds (Passeriformes and Columbiformes). When conditions are optimal, some SLE virus-infected mosquitoes (e.g., *C. quinquefasciatus* and *C. tarsalis*) bite susceptible individuals resulting in human infection.

An initial survey conducted in the Los Angeles Basin in 1986 identified a number of bird species exposed to SLE virus and several locations where transmission occurred (McLean et al., 1988). SLE virus transmission dynamics and those of western equine encephalomyelitis (WEE) remained unanswered. In order to address these issues, a serum screening program was started in 1987 to ascertain which species may be involved in local bird-mosquito-virus transmission cycles. In addition, mosquito collection systems were deployed at bird trapping sites making it possible to obtain concurrent mosquito, bird, and arbovirus data.

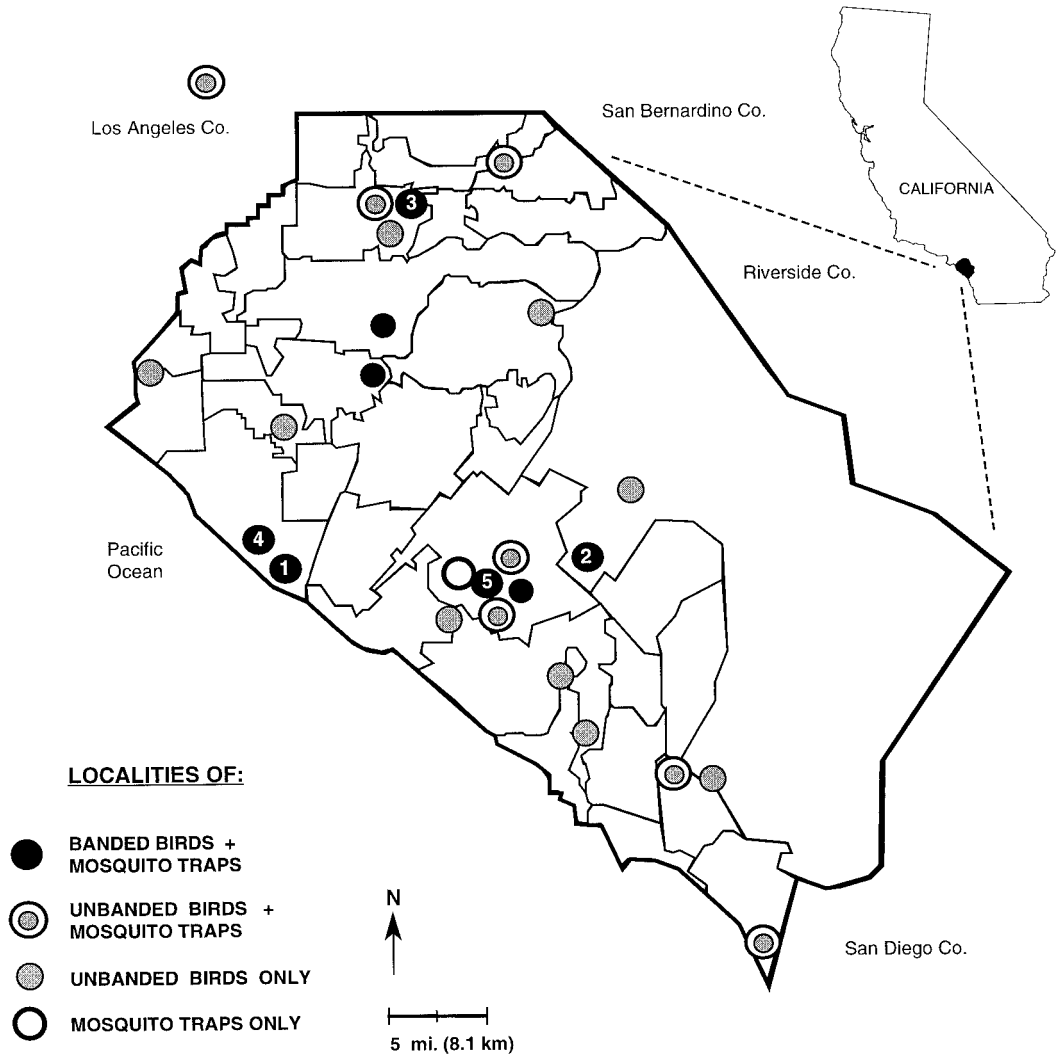


FIGURE 1. Wild bird and mosquito collection sites, Orange County, California, 1987–96; 1 = Central Park, 2 = El Toro Fire Station, 3 = Fullerton College, 4 = Huntington Beach Residence, 5 = 20 Ranch Duck Club.

## MATERIALS AND METHODS

Sites were selected in Orange County, California during this study period (Fig. 1) where both mosquitoes and SLE virus antibody positive birds were collected. These included Central Park (33°42′30″N, 118°00′00″W), El Toro Fire Station (33°37′30″N, 117°41′00″W), Fullerton College (33°53′59″N, 117°53′16″W), Huntington Beach Residence (33°40′27″N, 117°59′35″W), and 20 Ranch Duck Club (=San Joaquin Wildlife Sanctuary) (33°39′37″N, 117°50′28″W).

Birds were trapped using Japanese mist nets (3 m × 6 m; 24, 30, and 36 mm mesh) and modified Australian Crow Traps (McClure, 1984). Modified CDC/$CO_2$ light traps (Service, 1993) and Reiter/Cummings gravid female traps (R. F. Cummings, pers. comm.) also were placed at designated bird trapping sites. Beginning in 1989, house finches (*Carpodacus mexicanus*) and house sparrows (*Passer domesticus*) from selected localities (Fig. 1) were banded using sequentially numbered leg bands (Gruwell et al., 1990). Recapture records were maintained for each of the trapping sites. Individual birds were bled from the jugular vein using a tuberculin syringe (1 ml) fitted with a 25 gauge, 5/8 inch hypodermic needle. A volume of 0.1 ml of blood was removed and placed in a tube containing 0.9 ml 0.75% bo-

vine albumin/PBS (phosphate-buffered saline) diluent.

Serum hemagglutination inhibition (HAI) tests (Lennette, 1964) were performed (Gruwell et al., 1988). Sera samples were first treated to remove agglutinins and non-specific inhibitors of hemagglutination by adding equal volumes of 25% Kaolin suspension to 1:10 serum samples. Sera were incubated for 20 min at room temperature (RT), and then spun at 2500 RPM for 10 min. Next, 0.25 ml of 50% goose red blood cells (RBC) was added, then incubated for 20 min at 4 C, and spun at 1500 RPM for 10 min; supernatant is 1:20.

The HAI plating protocol used at the OCVCD Laboratory was as follows. 1. Add 0.025 ml of diluent to appropriate wells of microtiter "U" plate; 2. Add 0.050 ml of sample serum (1:20) to first wells. Dilute two-fold; 3. Add 0.025 ml of diluted antigen to each well and incubate at 37 C for 1 hr.; 4. Add 0.050 ml of 1:80 RBC suspension in corrected pH buffers to each well and incubate at RT for 1 hr.; and 5. Record results (Hemagglutination = viral antigen and serum = HAI negative; hemagglutination inhibition = viral antigen and anti-serum = HAI positive).

During the first 3 yr of the program the HAI tests were done by the Arbovirus Research Unit (ARU; University of California, Berkeley, California, USA); the Department of Epidemiology (University of California, Los Angeles, California, USA); and/or the Orange County Department of Health Services (OCDHS; Santa Ana, California, USA). From 1990 to 1996, the tests were conducted by the Orange County Vector Control District laboratory (OCVCD; Garden Grove, California, USA). Positive HAI sera were sent to the UCB and the Centers for Disease Control (CDC; Fort Collins, Colorado, USA), for plaque-reduction neutralization tests (PRNT) for confirmation (McLean et al., 1988).

The birds were released immediately after bleeding. Mortality due to trauma from the bleeding procedure was low (<1.0%).

References to selected statistical analyses were from Snedecor and Cochran (1980). Values for means, medians, and standard errors were determined using Minitab Descriptive Statistics (Minitab for Windows, 1995 edition, State College, Pennsylvania, USA). A chi square ($\chi^2$) test was used to determine if there were more reconversions in house finches and house sparrows on a seasonal basis ($X_1$ = January–March, $X_2$ = April–June, $X_3$ = July–September, $X_4$ = October–December).

In the following presentation the term seropositive is used when a tested serum yields a positive titer to a given antigen and may be seen at any time in a blood sampling sequence. A seroconversion is a specified seropositive result that follows at least one previous seronegative outcome in a serial bleeding of the same individual. A reversion is a negative serological result from an individual whose serum has previously tested positive. A seropositive titer resulting after a reversion is referred to as a seroreconversion.

## RESULTS

### Positive SLE seroprevalence/1987–1989

More than 24,000 birds were collected, bled, and tested for SLE and WEE virus antibodies during 1987–89. Forty avian species were trapped and sampled (Table 1). The house finch was the most frequently collected species (46.6%) followed by the house sparrow (23.6%). These two species and the pigeon (=Rock Dove; *Columba livia*) consistently tested positive for SLE virus antibodies. The WEE virus seropositive house finches and house sparrows were negligible, and positive sera from pigeons represented less than 1% of the sample (Table 1). In earlier studies, pigeons experimentally infected with SLE virus rarely developed detectable viremias (Reisen et al., 1992a). Therefore, a countywide system was set up (Fig. 1) designed to primarily collect house finches and house sparrows.

### House sparrows and house finches/1990–1996

In this period, 28,410 birds were sampled and tested for SLE and WEE antibodies (Table 1). Of the 12,520 house sparrows, 2.2% were positive (SE = 0.0013) for SLE antibodies; 1.1% of the 14,335 house finches were positive (SE = 0.0076). Although more house finches were bled and tested, twice as many house sparrows yielded positive sera for SLE antibodies. One house finch serum (1996) and four house sparrow sera (one in 1994, three in 1996) tested positive for WEE antibodies.

During this time period, trap sites were evaluated for house sparrow and/or house finch activity and also assessed for SLE and WEE virus activity. Figure 1 illustrates

TABLE 1. Prevalence of St. Louis encephalitis and western equine encephalomyelitis virus-specific antibody (tested by HAI) among wild birds sampled in Orange County California, (USA).

| Common name | Scientific name | Total | % Total samples | HI Ab positive | | % Positive | |
|---|---|---|---|---|---|---|---|
| | | | | SLE | WEE | SLE | WEE |
| 1987–89 | | | | | | | |
| House Finch | *Carpodacus mexicanus* | 11,264 | 46.6 | 47 | 2 | 0.4 | <0.1 |
| House Sparrow | *Passer domesticus* | 5,694 | 23.6 | 32 | 2 | 0.6 | <0.1 |
| Rock Dove | *Columba livia* | 4,454 | 18.4 | 151 | 22 | 3.4 | 0.5 |
| White-crowned Sparrow | *Zonotrichia leucophrys* | 1,153 | 4.8 | 6 | 0 | 0.5 | 0 |
| Red-winged Blackbird | *Agelaius phoeniceus* | 372 | 1.5 | 1 | 0 | 0.3 | 0 |
| Song Sparrow | *Melospiza melodia* | 268 | 1.1 | 0 | 1 | 0 | 0.4 |
| Mourning Dove | *Zenaida macroura* | 214 | 0.9 | 2 | 0 | 0.9 | 0 |
| Brown-headed Cowbird | *Molothrus ater* | 173 | 0.7 | 0 | 1 | 0 | 0.6 |
| Say's Phoebe | *Sayornis saya* | 154 | 0.6 | 2 | 1 | 1.3 | 0.7 |
| Common Ground Dove | *Columbina passerina* | 101 | 0.4 | 0 | 0 | 0 | 0 |
| Common Crow (American) | *Corvus brachyrhynchos* | 58 | 0.2 | 0 | 0 | 0 | 0 |
| Brewer's Blackbird | *Euphagus cyanocephalus* | 40 | 0.2 | 0 | 0 | 0 | 0 |
| European Starling | *Sturnus vulgaris* | 36 | 0.2 | 0 | 0 | 0 | 0 |
| Northern Mockingbird | *Mimus polyglottos* | 29 | 0.1 | 0 | 0 | 0 | 0 |
| California Gull | *Larus carlifornicus* | 24 | 0.1 | 0 | 0 | 0 | 0 |
| Scrub Jay | *Aphelocoma coerulescens* | 24 | 0.1 | 0 | 0 | 0 | 0 |
| Oregon Junco | *Junco oreganus* | 14 | <0.1 | 0 | 0 | 0 | 0 |
| Common Raven | *Corvus corax* | 12 | <0.1 | 0 | 0 | 0 | 0 |
| Black Phoebe | *Sayornis nigricans* | 11 | <0.1 | 0 | 0 | 0 | 0 |
| Black-headed Grosbeak | *Pheucticus melanocephalus* | 11 | <0.1 | 0 | 0 | 0 | 0 |
| Loggerhead Shrike | *Lanius ludovicianus* | 10 | <0.1 | 0 | 0 | 0 | 0 |
| Golden-crowned Sparrow | *Zonotrichia atricapilla* | 7 | <0.1 | 0 | 0 | 0 | 0 |
| Cliff Swallow | *Hirundo pyrrhonota* | 7 | <0.1 | 0 | 0 | 0 | 0 |
| Bullock's Oriole | *Icterus bullockii* | 7 | <0.1 | 0 | 0 | 0 | 0 |
| Audubon's Warbler | *Dendroica coronata* | 7 | <0.1 | 0 | 0 | 0 | 0 |
| Yellowthroat | *Geothylpis trichas* | 6 | <0.1 | 0 | 0 | 0 | 0 |
| Western Meadowlark | *Sturnella neglecta* | 5 | <0.1 | 0 | 0 | 0 | 0 |
| Spotted Towhee | *Pipilo erythrophthalmus* | 4 | <0.1 | 0 | 0 | 0 | 0 |
| Yellow Warbler | *Dendroica petechia* | 3 | <0.1 | 0 | 0 | 0 | 0 |
| Common Bushtit | *Psaltriparus minimus* | 4 | <0.1 | 0 | 0 | 0 | 0 |
| Lark Sparrow | *Chondestes grammacus* | 2 | <0.1 | 0 | 0 | 0 | 0 |
| California Quail | *Callioelpa californica* | 2 | <0.1 | 0 | 1 | 0 | 50.0 |

TABLE 1. Continued.

| Common name | Scientific name | Total | % Total samples | HI Ab positive | | % Positive | |
|---|---|---|---|---|---|---|---|
| | | | | SLE | WEE | SLE | WEE |
| Western Gull | *Larus occidentalis* | 2 | <0.1 | 0 | 0 | 0 | 0 |
| Ringed Turtle Dove | *Streptopelia risoria* | 1 | <0.1 | 0 | 0 | 0 | 0 |
| Swamp Sparrow | *Melospiza georgiana* | 1 | <0.1 | 0 | 0 | 0 | 0 |
| Western Kingbird | *Tyrannus verticalis* | 1 | <0.1 | 0 | 0 | 0 | 0 |
| Spotted Dove | *Streptopelia chinensis* | 1 | <0.1 | 0 | 0 | 0 | 0 |
| California Towhee | *Pipilo crissalis* | 1 | <0.1 | 0 | 0 | 0 | 0 |
| Anna's Hummingbird | *Calypte anna* | 1 | <0.1 | 0 | 0 | 0 | 0 |
| Short-billed Marsh Wren | *Cistothorus platensis* | 1 | <0.1 | 0 | 0 | 0 | 0 |
| | Subtotal | 24,179 | 100.0 | | | | |
| 1990–96 | | | | | | | |
| House Finch | *Carpodacus mexicanus* | 14,335 | 50.5 | 150 | 0 | 1.1 | 0 |
| House Sparrow | *Passer domesticus* | 12,520 | 44.1 | 244 | 0 | 2.2 | 0 |
| Rock Dove | *Columba livia* | 1,027 | 3.6 | 49 | 0 | 4.8 | 0 |
| White-crowned Sparrow | *Zonotrichia leucophrys* | 528 | 1.9 | 7 | 0 | 1.3 | 0 |
| | Subtotal | 28,410 | 100.0 | | | | |
| | | Total | 52,589 | | | | |

the sites tested for bird/virus activity and depicts the banded bird locations where relatively high levels of both bird and virus activity was discovered (Table 2) among these sites (1990–96). The SLE virus activity was present in the house sparrow and/or house finch populations in each of these for at least two of the 7 yr.

#### Seroreconversions in house finches and house sparrows

Seven banded house finches demonstrated seroreconversions at the Huntington Beach residence location; two in 1990, four in 1993, and one in 1994 (Table 3). The only other reconversion in a house finch was seen in 1991 from the 20 Ranch Duck Club. The four reconversions in 1993 included two that exhibited third reconversions, each with at least one negative serological test in between positive seroconversions. Most of the reconversions (6/7) occurred 2 to 3 mo after the initial positive seroconversions and at least two seronegative results were observed before a subsequent positive serological test. One house finch exhibited a second reconversion at a titer of 1:40 following three negative serological tests; a positive seroconversion seen 2 mo earlier converted at a titer of 1:20.

Seroreconversions in house finches exhibited a significant ($P < 0.05$) seasonal distribution in the cooler months of autumn and winter (October–December). House sparrow reconversions were fewer in number (4) and were recorded in March, July, August, and September ($P > 0.05$) (Table 3).

#### House finch and house sparrow longevity

Minimum age was calculated from banding records of 2,206 house finches and 2,158 house sparrows (Table 4). Among the house finches, 18 (0.8%) individuals were older than 1 yr with an average age of 713.6 days. This average was underestimated because the dates of birth and death are unknown. Seven banded house sparrows (0.4%) lived longer than 1 yr, averaging 559 days.

Eleven of the older 18 (61%) house finches were from the Huntington Beach residence location and six of the birds were bled during the 1993 season. Five of the 18 (28%) finches were from the 20 Ranch Duck Club site and all also were bled during the 1993 season. Two of the 11 (18%) house finches from Huntington Beach reconverted, and one of the five house finches from 20 Ranch Duck Club seroconverted (Table 4). Three older house sparrows seroconverted with one seroreconversion (Table 4).

#### Cooler months transmission

An assessment of the SLE positive seroconversion data from the banded and serially bled house sparrows and house finches yielded evidence that SLE virus transmission may occur during the cooler months of autumn/winter (Table 5) as well as during warmer seasons. Of the 2,206 house finches, 60 (2.7%) tested positive for SLE antibodies with one positive during late autumn; two of 66 SLE positive house sparrows (2,158 total blood samples) demonstrated December SLE virus transmission. These sera were collected from the house finches during 1990 to 1996 and during 1992 to 1996 from the house sparrows.

#### Bird population dynamics

Based upon house sparrow : house finch population ratios (Vanderpool, 1993, Fig. 2), Orange County may be partitioned into three major bird association habitat types; house finch, house finch/house sparrow, and house sparrow. These bird-habitat types were correlated with the age of man-made improvements, i.e., house finch—underdeveloped ≤5 yr human occupied, house finch/house sparrow—5 to 35 yrs occupied, house sparrow—20>50 yr occupied. Vanderpool's (1993) results yielded statistically significant (chi-square) differences within and between trap sites. Eight yr of collection data from a number of sites during this study, particularly three locations (Fig. 2), illustrate the consistency of

TABLE 2. St. Louis encephalitis antibody seropositives and reconversions[a] in banded house finches and house sparrows in Orange County (California, USA).

| | Location | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Hungtington Beach Residence | | Central Park | | Fullerton College | | 20 Ranch Duck Club | | El Toro Fire Station | |
| Year | SLE positive/ tested | percent positive | SLE positive/ tested | percent positive | SLE positive/ tested | percent positive | SLE positive/ tested | percent positive | SLE positive/ tested | percent positive |
| House Finches | | | | | | | | | | |
| 1990 | 9(2)[a]/200 | 4.5 | — | — | — | — | 9(0)/408 | 2.2 | — | — |
| 1991 | 1(0)/131 | 0.8 | — | — | — | — | 3(1)/111 | 2.7 | — | — |
| 1992 | 6(0)/176 | 3.4 | 3(0)/77 | 3.9 | 1(0)/1 | 100.0 | 2(0)/118 | 1.7 | 6(0)/122 | 4.9 |
| 1993 | 11(5)/182 | 6.0 | 1(0)/78 | 1.3 | 0/5 | 0.0 | 4(0)/147 | 2.7 | 0/41 | 0.0 |
| 1994 | 3(2)/103 | 2.9 | 0/54 | 0.0 | 0/5 | 0.0 | 1(0)/31 | 3.2 | 0/53 | 0.0 |
| 1995 | 0/7 | 0.0 | 0/40 | 0.0 | — | — | — | — | 0/13 | 0.0 |
| 1996 | 0/0 | 0.0 | 2(0)/77 | 2.6 | — | — | — | — | 0/26 | 0.0 |
| House Sparrows | | | | | | | | | | |
| 1992 | 15(0)/273 | 5.5 | 14(0)/222 | 6.3 | 7(1)/139 | 5.0 | 0 | 0 | 4(0)/127 | 3.1 |
| 1993 | 4(0)/133 | 3.0 | 1(0)/197 | 0.5 | 1(1)/82 | 1.2 | 0 | 0 | 1(1)/44 | 2.3 |
| 1994 | 2(0)/215 | 0.9 | 2(0)/213 | 0.1 | 0/29 | 0.0 | 0 | 0 | 0/53 | 0.0 |
| 1995 | 0/79 | 0.0 | 0/63 | 0.0 | — | — | — | — | 0/54 | 0.0 |
| 1996 | 13(1)/167 | 7.8 | 2(0)/45 | 4.4 | — | — | — | — | 0/23 | 0.0 |

[a] Number of reconversions in parentheses.

TABLE 3. Seroreconversions St. Louis encephalitis in Orange County (California, USA).

| Band number | Location | Seroconversions | | | Reconversions | | |
|---|---|---|---|---|---|---|---|
| | | Total number bleeds | 1st date-titer | Negative bleeds | 2nd date-titer | Negative bleeds | 3rd date-titer |
| House Finches 1989–96 | | | | | | | |
| 101 | Huntington Bch | 33 | 9/21/90-1:40 | 2 | 11/16/90-1:20 | — | — |
| 329 | Huntington Bch | 13 | 6/29/90-1:20 | 7 | 10/19/90-1:20 | 2 | — |
| 6471 | Huntington Bch | 29 | 7/7/93-1:20 | 9 | 2/9/94-1:20 | 13 | — |
| 6589 | Huntington Bch | 23 | 9/15/93-1:40 | 5 | 12/7/93-1:20 | 3 | 2/24/94-1:40 |
| 6601 | Huntington Bch | 6 | 8/18/93-1:20 | 4 | 10/26/93-1:20 | — | — |
| 6622 | Huntington Bch | 8 | 8/31/93-1:40 | 3 | 10/26/93-1:20 | 2 | — |
| 6623 | Huntington Bch | 11 | 8/18/93-1:20 | 1 | 9/15/93-1:20 | 2 | 11/11/93-1:20 |
| House Sparrows 1992–96 | | | | | | | |
| 6008 | Fullerton | 27 | 6/9/92-1:20 | 4 | 9/8/92-1:40 | 12 | 7/23/93-1:40 |
| 6037 | El Toro | 10 | 11/27/92-1:40 | 1 | 3/8/93-1:20 | — | — |
| 7159 | Huntington Bch | 9 | 7/17/96-1:20 | 2 | 8/29/96-1:20 | 3 | — |

these habitat types. Unexplained changes in the relative proportions of the house finch and house sparrow populations occurred from 1991–96 at the Huntington Beach site (Fig. 3).

### Mosquito species and numbers

*Culex quinquefasciatus* and *Culex tarsalis* were more abundant at the 20 Ranch Duck Club, a wetland area, than at three other suburban sites (Fig. 4). Suburban sites generally yielded greater numbers of *C. quinquefasciatus* although a number of locations (e.g., Huntington Beach) yielded high levels of *C. tarsalis.* In general, *C. tarsalis* activity peaked in July, subsided by the end of October, and began again in February or March, whereas, *C. quinquefasciatus* adults were trapped year-round although at very low levels during the cooler months of autumn and winter (Fig. 5). *Culex stigmatosoma, Culiseta incidens,* and *Culiseta inornata* also were collected, albeit in fewer numbers, at infrequent intervals, and from scattered locations.

## DISCUSSION

This Serosurveillance Program commenced in January 1987 in Orange County under the assumption that the earliest indicator of arbovirus (SLE and WEE) transmission was through the detection of virus activity in wild birds. The traditional method of demonstrating viral infection was by testing avian sera for positive antibodies. Scott (1988) in independent studies stated that the best indication of arbovirus transmission is virus isolation from wild birds. However, because of the rarity of virus recovery from avian hosts due to short-term (2 to 4 days) viremias, antibody surveys were conducted to ascertain evidence of virus infection. Wild birds have long been considered the most important vertebrate reservoir hosts for both SLE and WEE viruses (Hardy and Reeves, 1990). We selected the HAI test over the neutralization test in our study because of its duration and lower cost (J. L. Hardy, pers. comm.). The utility of the HAI test

TABLE 4. Minimum age (>1 year) of banded house finches and house sparrows in Orange County (1989–95).

| Locality | Band number | First sample | Last sample | Total samples | Number days alive |
|---|---|---|---|---|---|
| House Finches (*Carpodacus mexicanus*) | | | | | |
| Huntington Beach Residence | 100[a] | 12/29/89 | 10/4/95 | 110 | 2,075 |
| Huntington Beach Residence | 176 | 12/29/89 | 4/14/93 | 4 | 1,185 |
| Huntington Beach Residence | 148 | 12/15/89 | 2/21/92 | 5 | 786 |
| Huntington Beach Residence | 9931 | 2/22/91 | 3/17/93 | 10 | 745 |
| Huntington Beach Residence | 335 | 5/18/90 | 2/21/92 | 2 | 633 |
| Huntington Beach Residence | 6471[b] | 4/28/93 | 9/30/94 | 29 | 512 |
| Huntington Beach Residence | 101[b] | 8/11/89 | 11/16/90 | 33 | 455 |
| Huntington Beach Residence | 10061 | 8/6/91 | 11/11/92 | 5 | 455 |
| Huntington Beach Residence | 309 | 3/23/90 | 6/21/91 | 22 | 448 |
| Huntington Beach Residence | 341 | 2/21/92 | 5/12/93 | 10 | 441 |
| Huntington Beach Residence | 6226 | 7/22/92 | 9/15/93 | 6 | 413 |
| 20 Ranch Duck Club | 4763[a] | 4/4/91 | 11/22/93 | 43 | 948 |
| 20 Ranch Duck Club | 512 | 8/7/91 | 12/13/93 | 36 | 846 |
| 20 Ranch Duck Club | 543 | 2/19/92 | 12/13/93 | 21 | 654 |
| 20 Ranch Duck Club | 513 | 8/7/91 | 4/19/93 | 19 | 612 |
| 20 Ranch Duck Club | 541 | 2/19/92 | 10/25/93 | 21 | 606 |
| Mason Park | 193 | 11/2/89 | 2/20/91 | 17 | 468 |
| El Toro | 6027 | 5/21/92 | 12/13/93 | 14 | 562 |
| Range = 413-2,075, SE = 93.6, $\bar{x}$ = 713.6, Median = 609 | | | | | |
| House Sparrows (*Passer domesticus*) | | | | | |
| Central Park | 6273[a] | 10/21/92 | 8/10/94 | 2 | 649 |
| Central Park | 6023 | 4/21/92 | 11/23/93 | 10 | 609 |
| Huntington Beach Residence | 356 | 3/11/92 | 11/11/93 | 22 | 600 |
| Huntington Beach Residence | 6039 | 5/6/92 | 9/28/93 | 9 | 502 |
| OCVCD | 6975[a] | 4/5/94 | 9/11/95 | 30 | 516 |
| OCVCD | 6862 | 7/26/94 | 12/20/95 | 21 | 504 |
| Fullerton | 6008[b] | 2/25/92 | 8/17/93 | 27 | 532 |
| Range = 502-649, SE = 22.4, $\bar{x}$ = 559, Median = 532 | | | | | |

[a] Seroconversion, SLE.
[b] Seroreconversion, SLE.

was demonstrated in a field and experimental SLE virus study of house sparrows (McLean et al., 1983).

The first 3 yr (1987–89) of this study was comprised of a generalized trapping (mist nets and crow traps) program that identified house finches, house sparrows, and pigeons exhibiting significant prevalences of SLE virus antibody; WEE virus activity was negligible. From 1990–96, these three species were collected intensively (Table 1).

During the study, eight to 10 crow traps were in operation at any one time. Over the 10 yr period, sites were evaluated and abandoned because of vandalism, lack of bird activity, or lack of SLE virus activity, or were maintained because they represented both a bird and SLE virus active site.

An intensive bird banding operation was initiated in 1989 beginning at the Huntington Beach residence and at the 20 Ranch Duck Club. The banding later expanded to the Central Park, Fullerton College, and El Toro Fire Station trap sites in 1992 (Table 2).

Notable observations resulted from the

TABLE 5. Cooler months (Autumn) transmission of St. Louis encephalitis virus in house sparrows (HS) and house finches (HF); serological data.

| Location | Sample date | Titer | Bird species | Bird age | Band number |
|---|---|---|---|---|---|
| 20 Ranch Duck Club | | | | | |
| | 8/6/90 | <1:20 | HF | Immature | 7305 |
| | 9/20/90 | <1:20 | | Adult | |
| | 10/4/90 | <1:20 | | | |
| | 10/18/90 | <1:20 | | | |
| | 11/1/90 | <1:20 | | | |
| | 11/15/90 | <1:20 | | | |
| | 11/29/90 | <1:20 | | | |
| | 12/13/90 | 1:20 | | | |
| El Toro Fire Station | | | | | |
| | 5/21/92 | <1:20 | HS | Immature | 6037 |
| | 6/11/92 | <1:20 | | | |
| | 7/16/92 | <1:20 | | Adult | |
| | 8/27/92 | <1:20 | | | |
| | 9/16/92 | <1:20 | | | |
| | 11/18/92 | <1:20 | | | |
| | 11/27/92 | 1:40 | | | |
| Huntington Beach Residence | | | | | |
| | 6/24/92 | <1:20 | HS | Immature | 6182 |
| | 8/26/92 | <1:20 | | Adult | |
| | 9/9/92 | <1:20 | | | |
| | 11/11/92 | <1:20 | | | |
| | 12/16/92 | 1:40 | | | |

long-term serial bleeding of banded house sparrows and house finches. First, it became apparent that many of the birds habitually returned (often many times) to the same trap. No evidence of birds moving from one trap area to another was recorded. Many birds were bled more than twice. In one unusual case, a house finch was bled 110 times over a period of 6 yr (Table 4).

A number of house finches not only seroconverted to SLE virus on one occasion but demonstrated another (and sometimes a third) antibody titer after previous negative results (Table 3). In addition, these events not only occurred at only one of the five trap locations (Fig. 1, Number 4) but also five of nine of the seroreconversions happened in 1993, with two following in

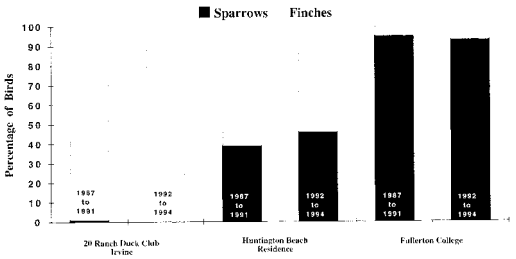


FIGURE 2. Ratio of House Sparrows to House Finches, Orange County (1987–91 from Vanderpool, 1993; 1992–94 from OCVCD data).

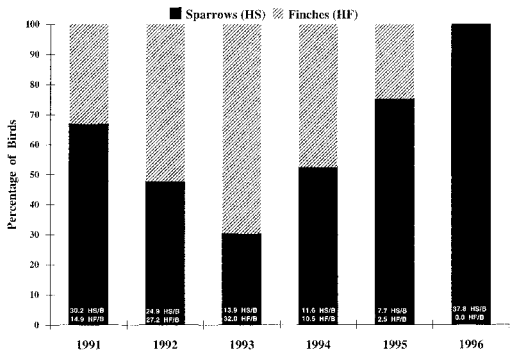


FIGURE 3. Proportion of House Finches: House Sparrows at the Huntington Beach Residence Trap Site, Orange County, 1991–96; B = bleeding event.

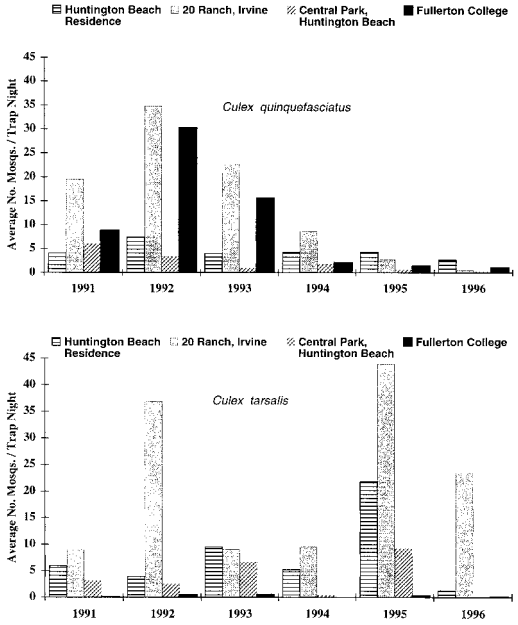


FIGURE 4. Average number of *Culex tarsalis* and *Culex quinquefasciatus* per trap night (CDC/CO2 traps) collected at four crow trap locations, Orange County, 1991–96.

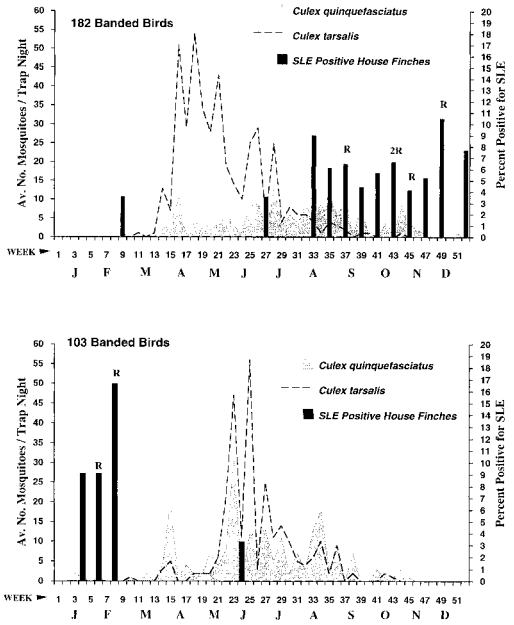


FIGURE 5. Host-seeking mosquitoes and SLE activity in banded house finches from a residence in Huntington Beach during 1993 (above), 1994 (below); R = Bird with re-emerged SLE virus activity.

early 1994. The two serological events occurring in 1994 were probably extensions of the 1993 season, which began in the middle of September and continued through February 1994 (Fig. 5).

The Huntington Beach trap site was an exclusive focus of a significant ($P < 0.05$) number of seroreconversions in house finches concentrated in the fall and winter of 1993–94 (Table 3). In contrast, seroreconverions in house sparrows ($P > 0.05$) were few, occurred in the early spring and summer, and were geographically scattered (Table 3).

Possible explanations for the observations of the seroreconversions in these house finches include (1) non-specific inhibitors of hemagglutination may be simulating the presence of viral antibodies in test sera; (2) negative results between positive reactions may include false negative results because of the standard cut-off values chosen for positive responses; (3) SLE virus-infected mosquitoes may be reinfecting the birds; (4) other serologically cross-reactive flavivirus(-es) may be infecting the birds; and (5) SLE virus may be sequestered into tissue during viremia and, following a latent period, reenters the blood thereby triggering an anamnestic humeral immune response after a short viremia.

Non-specific inhibitors consist mostly of free cholesterol, phospholipids, and/or free fatty acids (Salminen, 1962), substances which may vary among species (Gresikova and Sekeyova, 1969) and within species according to individual diet, stress, and seasonal behavior. Successful removal by Kaolin adsorption and/or acetone extraction is not always achieved, and sometimes it is at the expense of the specific viral immunoglobulins (Clarke and Casals, 1958). In the case of possible non-specific HAI reactions, sera were systematically retreated and retested.

In this study the HAI test results were consistently reported as positive when clear titers ≥1:20 were observed. Using this standard interpretation, all results positive at 1:10 or indistinctly positive at 1:20

were systematically dismissed. It is possible however that these latter titers may include false negative sera. Nonrandomness of our results and the large number of negative test results between positive seroconversions (Table 3) for most of the reconverting hosts strongly suggest that false negative results in the second explanation is not the answer.

The third explanation also does not seem likely based upon the negative results received from the California State Viral and Rickettsial Disease Laboratory (Berkley, California) for tests of mosquito pools, which averaged 318 pools per year from 1989 through 1996 (OCVCD, unpubl.). These mosquito pools consisted of specimens trapped concurrently with the bird bleeding program at the same locations. Also, although the data support autumn/winter SLE virus transmission, it is at a very low level, primarily because of the small numbers of vector mosquitoes (*C. quinquefasciatus*) active during the cooler months.

The fourth explanation does not seem likely because there is no evidence of flavivirus infections other than SLE virus in birds from southern California (McLean et al., 1983; Milby and Reeves, 1990).

The fifth explanation, that of persistent latent infection and induced relapse of SLE viremia, seems to best fit the results obtained from this study.

Other researchers interpreting similar results also speculated that the reappearance of antibody is due to the relapse of cryptic infections of viruses, including SLE (McLean and Scott, 1979) and eastern equine encephalomyelitis (EEE; Emord and Morris, 1984; Crans et al., 1994). WEE virus was isolated from a house sparrow and a house finch approximately 8 mo after they were inoculated in the laboratory (Reeves et al., 1958b).

McLean and Scott (1979) suggested that chronic, latent infection in the birds explains overwintering by SLE virus, which would then recirculate in the peripheral blood during the following spring. Mosquitoes may become infected by feeding on the viremic overwintered birds and subsequently initiate the spring/summer enzootic cycle. Crans et al. (1994), investigating interactions among cryptic EEE virus, resident swamp birds, and *Culiseta melanura* in New Jersey (USA), attributed isolation of EEE virus in the early spring to virus recrudescence stimulated by avian physiological changes elicited by stress factors, such as those involved in migration, breeding, and territory establishment. Emord and Morris (1984) ascribed the stresses of feeding vectors and interaction with other vertebrates as stimulants for the recrudescence of latent EEE virus in locally breeding wetland birds in New York (USA). Under laboratory conditions, virus shedding was observed in stressed immune ducks infected with latent duck enteritis virus (Hudson, 1994). Upon demonstrating the isolation of WEE virus from the blood of a house sparrow and the brain tissue of a house finch months after inoculations, Reeves et al. (1958a) suggested the potential of avian hosts as long-term reservoirs and sources of virus for vectors. Reeves (1961), in advancing a number of feasible hypotheses to explain the apparent survival of viruses through adverse periods and their reappearance under favorable conditions, conjectured that vertebrate hosts may exhibit chronic relapsing infections which may serve as sources of vector infection following inclement circumstances. Wild birds, including house sparrows and house finches, were implicated as long-term reservoirs of WEE virus (Reeves and Hammon, 1962) because of apparent chronic latent infections seen during studies in Kern County (California).

McLean and Scott (1979) suggested that SLE virus attains a cryptic or latent state in certain bird species during the fall/winter season, followed in the spring by viremia. The present data (Table 3) generally support this observation with some modifications. Two of the house finches in this study seroreconverted for SLE virus HAI

antibody (Table 3) in February 1994, whereas others seroreconverted in September, October, and November 1993. These results may reflect a temporal vacillation at its most extended points, i.e., from the ending of late summer mosquito activity to early spring-renewed mosquito activity. In Orange County, *C. tarsalis* activity usually ends by mid-October and begins again in February or March (Fig. 5). The shifting of virus activity in the birds from late summer to early spring and back again may be coherent with changing relevant environmental conditions (e.g., house sparrow and house finch population dynamics, vector mosquito population oscillations, etc.). Reeves et al. (1958b), on the basis of their studies during the 1950's in Kern County, California, concluded that vertebrates were likely the overwintering hosts of an encephalitis virus, in this case WEE virus. This paradigm of seasonal fluctuation of correlated bird/virus/mosquito interactions may explain both the overwintering process and the occasional spillover of SLE virus into human populations during the late summer when viremic SLE virus-recrudescing birds and ornitho-anthropophilic mosquito species experience optimal interactions.

Sites were selected during this 10 yr study and maintained if mosquito, bird, and SLE virus activity were present. If over a period of 6 to 12 mo the site yielded neither birds nor SLE virus-active birds, the site was abandoned. As a result, these data highlighted some bird trapping locations as consistent foci of SLE virus activity and others as places of no viral activity, which seems to correlate with Pavlovsky's (1964) "nidality" concepts. Pavlovsky (1964) stated that a nidus (a focus of infection) is a specific location characterized by climate, vegetation, soil, and microclimate favorable for the maintenance of the vectors, reservoir hosts, and susceptible recipients of the disease agent; in this case SLE virus. In the event that more SLE virus recrudescence activity manifests during the warmer summer and environmental, bird, mosquito, and human activity factors are optimal at widely spaced foci (nidi), then SLE virus transmission (from birds to mosquitoes, and under certain conditions from mosquitoes to humans) may result at a number of geographically separated locations during the same time frame. Whether at one, a few, or many SLE foci, a set of host/vector/virus conditions must merge in concert with optimal environmental factors to facilitate virus transmission. These interactive factors have been evaluated at least in part by a number of investigators (e.g., Chamberlain et al., 1959; Day and Carlson, 1985; Hardy and Reeves, 1990; McLean, 1991; Meyer et al., 1988; Monath and Tsai, 1987; Reeves, 1967). Smith et al. (1983) identified persistent endemic foci of SLE virus in Mississippi (USA) during studies of SLE cases and infections of house sparrows.

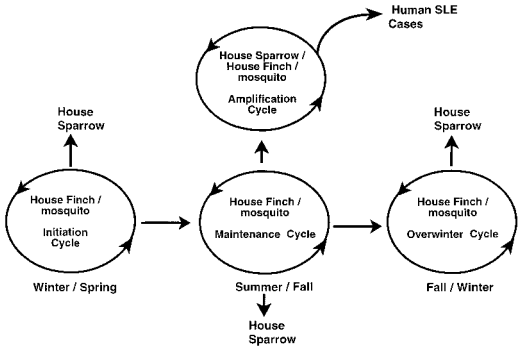


FIGURE 6. Transmission cycle of SLE virus in house sparrows and house finches at SLE foci.

Beginning with bird hosts (house finches and house sparrows) as the initiation point of the SLE enzootic transmission cycle (Fig. 6), viremic birds and vector mosquitoes interact through a blood feeding/virus transmission event chain reaction, which could result in viral amplification in the local bird populations through cascading infections. In at least some of the birds, the SLE virus apparently leaves the blood and enters different tissues. Which tissues are not known. Evidence of SLE virus isolation from the crop of an avian

host has been documented (Chamberlain et al., 1957). Data (Table 3) suggest that certain SLE virus-infected birds become immune, lose their circulating antibodies, and then subsequently exhibit a detectable antibody titer (≥1:20), which implies reactivation of virus replication and viremia. This reactivation of viral replication may be induced by a stimulus such as stress. Mosquitoes become infected, then infective, and subsequently may transmit virus to a susceptible bird. If the recrudescence sequence is limited to a single bird, then the amplification cycle is probably restricted to a single geographic location (focus) and the extent of the cycle is dependent upon continuing favorable conditions, including optimal mosquito population dynamics. Reisen et al. (1992b) assessed the role of several bird-feeding mosquito species in the SLE virus transmission cycle in the Los Angeles Basin. These conditions probably represent the maintenance system, periodically yielding low level virus activity at each focus.

When stimuli that evoke recrudescence impact birds occupying a broader geographic area, then amplification of virus takes place at a number of separate sites (multiple foci) and somewhat synchronously, which in turn could spawn additional foci and/or coalesce into larger foci. If favorable conditions for host bird/vector mosquito interaction diminish significantly, then localized enzootic virus transmission is reduced to low levels, a condition seen in surveillance data when there are only a few SLE virus antibody positive birds seen at different locations. Virus may remain at the focus in the latent state sequestered in birds that live longer than 1 yr. When the bird/mosquito conditions reach threshold levels and stimuli initiate viremia in latently-infected older birds, then the enzootic cycle is reestablished. This initiation of a renewed cycle may be at one or a few local sites, resulting in a limited transmission episode; or it may be extensive, yielding geographically widely occurring and separate events. It appears that these cause and effect interactions may be seen with both house sparrows and house finches.

The key to human SLE cases seems to be connected to house sparrow/virus amplification cycles and, in Orange County (including the Los Angeles Basin), to house finch/virus maintenance cycles (Fig. 6). House finches have existed in suitable habitats in California for eons (Miller, 1937). A mosquito-bird enzootic SLE virus cycle has probably been maintained here with this bird species for a long time as well. The recrudescence of latent SLE virus in house finches may be an overwintering strategy of the SLE virus to facilitate virus transfer to reappearing vector mosquito populations in the early spring; late fall and winter viral recrudescence in house finches increases the probability of SLE virus attaining a mosquito and/or bird host to survive through the colder months when there is diminished vector mosquito activity. The house finch assists in the maintenance part of the enzootic cycle through the sequestering/reemerging virus (viremia) sequence exhibited by certain birds; an actuating cycle involving older viriferous birds reestablishes enzootic cycles that have extinguished or supplements ones still active. Minimum estimates of the age of the longer living cohorts of house finches and house sparrows in these results were 1.9 yr for house finches and 1.5 yr for house sparrows. Milby and Wright (1976) found the median survival time after banding for house finches to be 1.9 yr and for house sparrows to be 1.2 yr in Kern County. In this study, twice as many house finches as house sparrows survived longer than 1 yr (Table 4). In cycles revolving around the active house finch populations, spillover of SLE virus from recrudescing finches into peak vector mosquito (*Culex* spp.) populations is rare because of the time of the year resulting in very few human SLE virus infections (Fig. 7).

In a review of SLE epidemics and factors affecting SLE virus transmission,

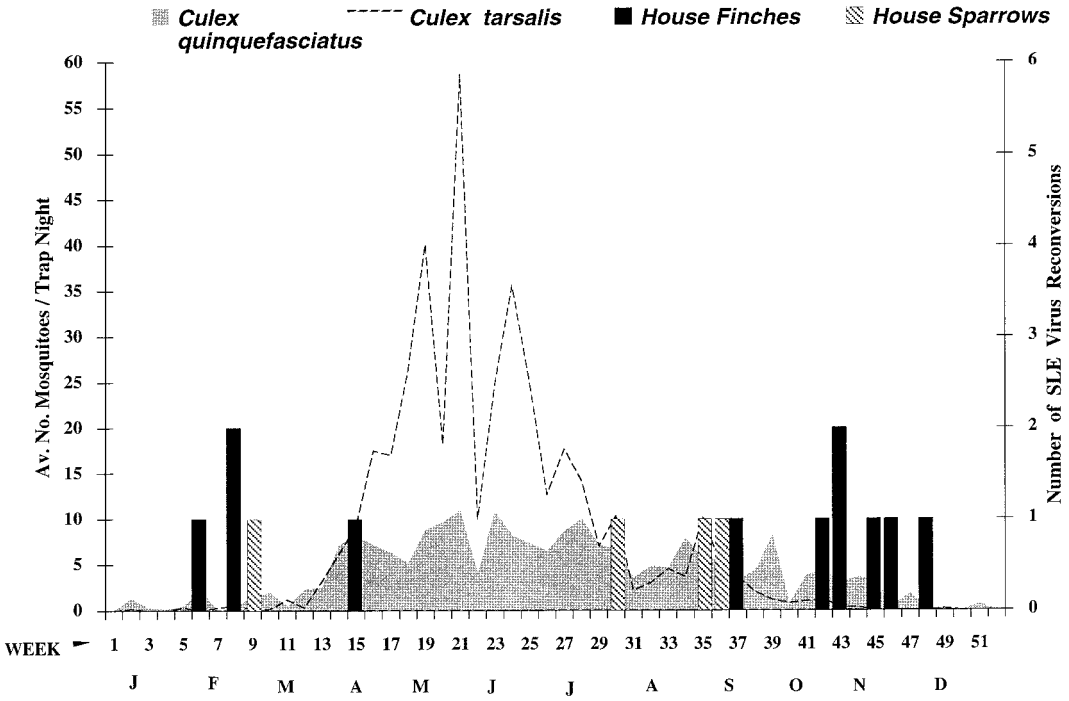


FIGURE 7. Weekly SLE virus reconversions in banded sparrows (2,158) and finches (2,206) in Orange County compared with 6 yrs (1992–97) of vector mosquito collection data from Huntington Beach. Reconversions in house finches during October, November and December were significant ($P < 0.05$).

Monath and Tsai (1987) recounted the research elucidating the immunological response by SLE virus-challenged immune nesting birds. Nestling house sparrows from eggs of immune mothers were challenged with SLE virus at various intervals after hatching. Certain birds, compared to controls, responded with viremias of greater duration and magnitude, a condition referred to as viremia enhancement. Monath and Tsai (1987) projected the importance of this phenomenon onto SLE virus amplification cycles and the increase of epidemic virus activity probabilities. Ludwig et al. (1986) specifically mentioned this phenomenon with house sparrows. House sparrows and house finches (that exhibit recrudescing SLE virus episodes) should therefore be evaluated for possible correlations between viremia enhancement and SLE virus amplification in avian populations.

House sparrows were introduced into the United States (New York) in the 1850's and are now widespread (McLean and Bowen, 1980) occupying both rural and urban habitats. At least four introductions were made in Illinois (1868–76) and by 1886 nearly the entire state of Illinois, as well as a large part of the rest of the United States, was occupied (Graber and Graber, 1963). In the Los Angeles Basin, the introduced house sparrow is nearly, if not entirely, an ecologic equivalent of the house finch (Vanderpool, 1993) and has intruded into the natural endemic SLE cycle. When triggering conditions develop, apparently during the summer months (Fig. 7), certain viriferous immune house sparrows may exhibit SLE virus recrudescence (Fig. 8) often in synchrony with optimal levels of mosquito activity. If these triggering conditions are limited geographically, then only one or a few locations are activated into becoming SLE virus foci. Alternatively, if the stimulating conditions are widespread, then many foci may become active sources of SLE virus for in-

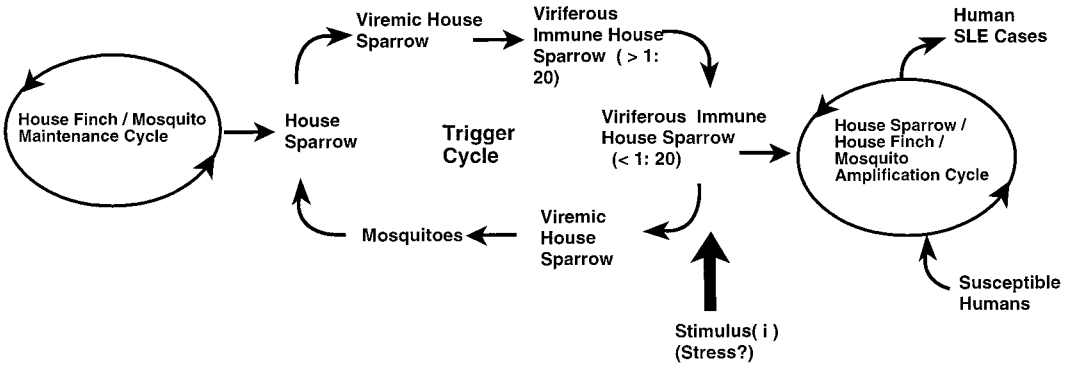


FIGURE 8. House Sparrow Trigger Cycle (Summer/Fall) at SLE Foci.

fective vector mosquitoes possibly setting the stage for a human SLE epidemic.

House sparrows seem to have been important components in various urban SLE epidemics primarily because of their abundance (McLean, 1991). Smith et al. (1983) implicated house sparrows in northwestern Mississippi as the major amplifying hosts for SLE virus with other passerine species involved in enzootic maintenance of SLE virus. McLean et al. (1993) found a similar situation in Arkansas. Other investigators implicated the house sparrow as being significant in SLE epidemics in Corpus Christi, Houston, and Dallas (Texas, USA) (Lord et al., 1973, 1974), McLeansboro (Illinois, USA) (Kokernot et al., 1967), and Danville (Kentucky, USA) (Mack et al., 1967). Spence (1980) quoted Monath's suggestion that the relative scarcity of sparrows in some urban localities of tropical America may be important in restricting SLE epidemics. This keystone importance as a principal factor in the SLE virus enzootic cycle is interesting because *P. domesticus* is a relatively recent arrival to SLE virus endemic localities. It may be that the house sparrow is preadapted as a competent reservoir host for SLE virus (a flavivirus) because of its known association with Old World flavivirus. Examples include records of West Nile virus (a flavivirus) infection in *P. domesticus* from Egypt (Hayes, 1989; Taylor et al., 1956; Work et al., 1955), Israel (Nir et al., 1969), and South Africa (Hayes, 1989).

Laird and Hoogstraal (1975) expressed their concern about the introduction of exotic disease agents via imported nonnative bird species into Europe. Apparently the importation of the European house sparrow into North America did not introduce any new human-impacting pathologic agents to new environs. Instead, the house sparrow adapted to cohabiting with the native house finch and in the Los Angeles Basin, at least, integrated itself into the enzootic house finch/SLE virus/mosquito cycle. As a complement of the SLE cycle, house sparrows that are exhibiting summertime SLE virus recrudescence, perturb the endemic enzootic cycle by making SLE virus available during the months of higher level *C. tarsalis* and *C. quinquefasciatus* populations and activity (Fig. 7). These data suggest that prior to the introduction of the house sparrow, the potential for human SLE cases in the Los Angeles Basin was low primarily because of the winter/spring reemergence of SLE virus in house finches. Data from census studies in Illinois (Graber and Graber, 1963) show that urban populations of house sparrows were much greater than rural populations in 1957–58 compared to 1907–09, with the total number remaining about the same (5–6 million) for both census periods. This supports the idea that house sparrows moved into residential habitats as suburbia

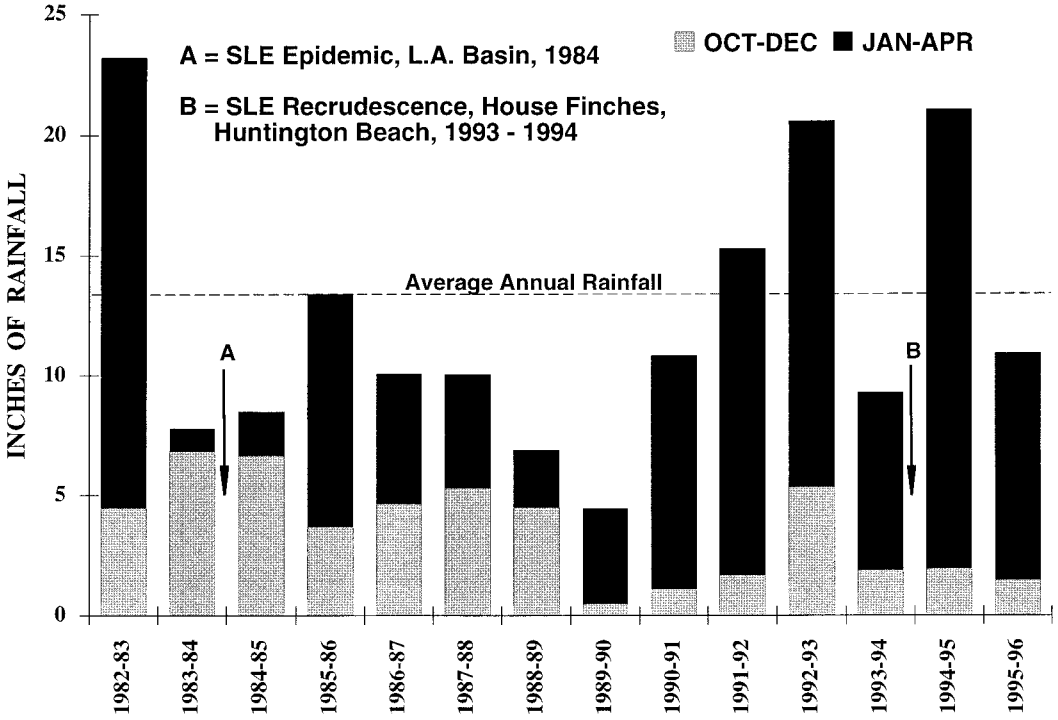


FIGURE 9. Average seasonal rainfall in Orange County, 1982–96; NOAA records.

expanded to accommodate the human population growth and increased the potential for urban SLE epidemics.

Meteorological factors favoring the onset of SLE epidemics (Bowen and Francy, 1980) include warm wet winters, cool springs, and hot dry summers. The weather pattern associated with the 1984 SLE epidemic in the Los Angeles Basin of California generally fits the above description except higher than average winter/spring rainfall occurred a full year before the late summer/fall epidemic (Fig. 9). During the first nine months of 1984, Orange County experienced significant above normal temperatures (Fig. 10). The usual summertime rainless conditions and site-specific above average temperatures (July/August/September) during the Los Angeles Basin SLE outbreak were reported by Webb and Myers (1986). Tsai and Mitchell (1989) proposed that high temperatures may favor SLE virus transmission by decreasing the extrinsic incubation period of the virus in the mosquito vectors. Reiter (1988) reported on bird serology studies that indicated enzootic transmission of urban SLE virus during the spring and autumn of nonepidemic years, whereas, there is a sudden recrudescence of SLE virus during summer (late June or July) of epidemic years. Reiter (1988) also described epidemic years as having above-normal temperatures from May through August, higher than usual rainfall in January and February, and an abnormally dry July. The present results (Figs. 5, 6, 7, 8, 9) parallel and may provide an explanation of the observations by Reiter (1988) of urban SLE transmission during nonepidemic and epidemic years.

Once a serosurveillance system comprised of a number of SLE virus active foci has been set up, then predictions of SLE virus activity may be made. Seven of the 10 yr of this study, positive (SLE) bird data anteceded the results from other sentinel systems. For example, in 1990 (Fig. 11) positive birds were detected before positive mosquitoes in April and before posi-

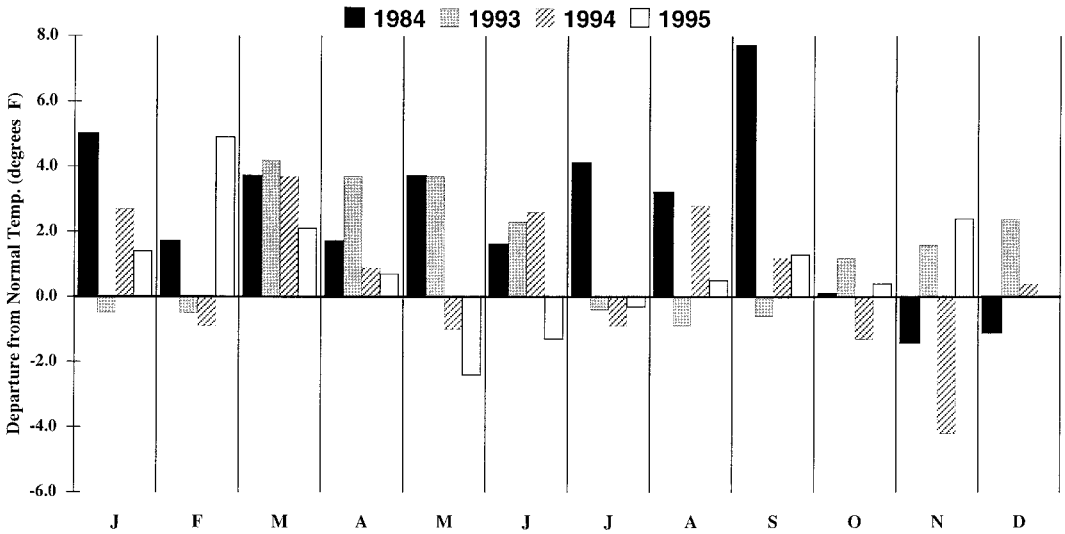


FIGURE 10. Monthly temperatures (1984, 1993, 1994, 1995) expressed as departures of degrees F from the normal temperature; Santa Ana, Orange County, California (NOAA records).

tive sentinel chickens in July; the total number of mosquito pools submitted for testing from the Los Angeles Basin was 1,984 and eight chicken flocks (ca. 20 chickens per flock) were tested monthly for six months (Emmons et al., 1991). Similar results were seen in 1996 (Fig. 11). Thus, monitoring the incidence of antibody positive birds at SLE virus foci could provide "early warning" information about potential high levels of SLE virus activity that possibly could lead to human epidemics.

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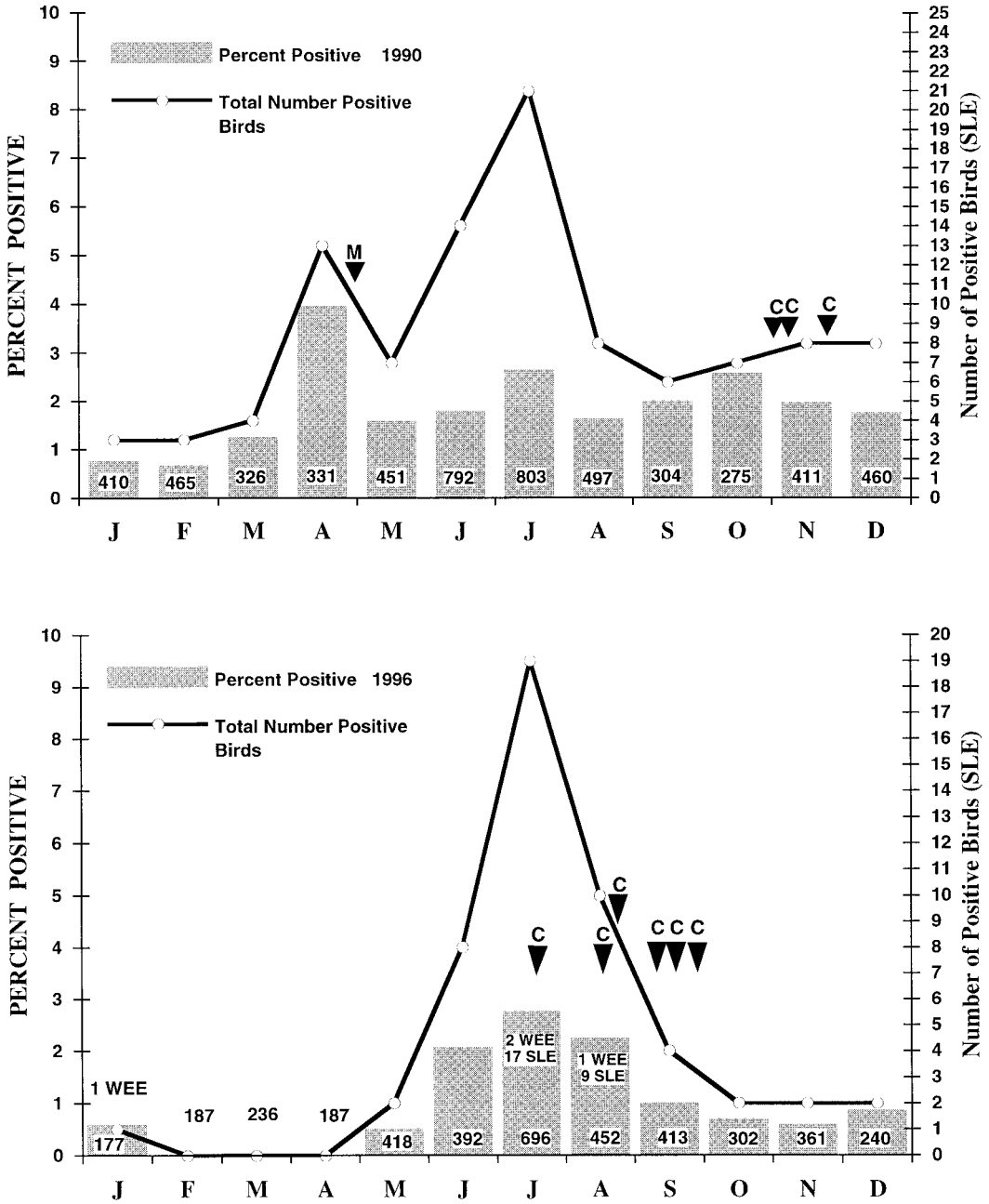


FIGURE 11. SLE (and WEE) virus activity as indicated by antibody (HAI) positive house sparrows and house finches (Orange County) in the Los Angeles Basin, 1990 and 1996; N = Number of birds collected; C = Chicken seroconversions for SLE; M = Mosquito pools positive for SLE.

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This paper is dedicated to Dr. John A. Gruwell who passed away shortly after the 1996 bird collection season. During this 10 yr sero-

surveillance study, John exhibited his skills as a diligent and intuitive researcher, traits rarely found today among scientists in our field, that resulted in a large body of valuable data. His passing is a great loss to us, his family, and the scientific community. Many of the ideas and conclusions expressed in this paper, fortunately, had been formulated by or discussed with John during the course of the study.

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