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Source: Journal of Wildlife Diseases, 22(3) : 326-334

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-22.3.326

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Journal of Wildlife Diseases, 22(3), 1986, pp. 326–334

VIREMIC ENHANCEMENT DUE TO TRANSOVARIALLY ACQUIRED ANTIBODIES TO ST. LOUIS ENCEPHALITIS VIRUS IN BIRDS[1]

G. V. Ludwig,[2,4] R. S. Cook,[2] R. G. McLean,[3] and D. B. Francy[3]

ABSTRACT: Adult house sparrows (*Passer domesticus*) were captured and experimentally inoculated with St. Louis encephalitis (SLE) virus to produce high concentrations of circulating antiviral antibody. Nestlings, 5–7 and 14–16 days of age, from SLE immune adult females and challenged with SLE virus, exhibited viremic enhancement by producing viremias of greater duration and magnitude than did controls. Nestlings possessing maternal antibody and challenged with SLE virus between 8 and 13 days of age did not produce viremias differing significantly from controls in magnitude, duration, or temporal appearance. Experimental nestling sparrows possessed detectable amounts of maternally derived passive antibody to SLE virus prior to challenge with this virus. Passive geometric mean antibody titers ranged from a high of 1:34.5 in nestlings tested 5–7 days posthatching, to a low of 1:11.2 in 14–16-day-old birds. Results presented imply that enhancement of SLE virus infections could lead to increased viral amplification and dissemination rates during natural disease cycles.

INTRODUCTION

St. Louis encephalitis (SLE) is a mosquito-borne flavivirus that has been responsible for extensive human epidemics in North America (Centers for Disease Control, 1982, 1983). Vertebrate hosts for SLE virus include passeriform and columbiform birds (McLean and Scott, 1979). When SLE virus is present under favorable conditions, a rapid epizootic may be triggered in urban bird populations which may in turn lead to human epidemics (McLean and Bowen, 1980).

House sparrows (*Passer domesticus*) are the major host species for SLE virus transmission cycles in several areas of North America (McLean and Bowen, 1980). In many of these areas, house sparrows are the predominant bird species and are probably the major contributors to the amplification of SLE virus activity (McLean et al., 1983).

Once SLE virus appears in house sparrow populations, it often spreads rapidly through a major portion of the population. Antibody-dependent enhancement (ADE) of in vitro flaviviral infectivity in the presence of low titers of flavivirus antibody (Hawkes, 1964; Hawkes and Lafferty, 1967; Halstead et al., 1980) and presence of antibody transmitted transovarially to nestling birds from their immune mothers (Bond et al., 1965) have suggested new possibilities for studying the mechanisms behind the rapid annual recrudescence of SLE virus.

ADE of arboviral infectivity was first observed in vitro by Miura and Scherer (1962). Work by Hawkes (1964) showed that the infectivity [in plaque-forming units (PFU)] of Murray Valley encephalitis (MVE) virus, West Nile encephalitis virus, and Japanese encephalitis virus was increased by as much as 12 times over controls by the action of antiviral antisera.

The most widely accepted mechanism for the occurrence of ADE of arbovirus infections suggests that immune complexes are formed between virus and antiviral IgG molecules. The Fc termini of these antibody–virus complexes then at-

Received for publication 5 November 1985.

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tach to complementary cell receptors on mononuclear phagocytes, leading to internalization of the complexes which facilitates enhanced viral infections. Enhancement of infections occurs in the presence of antibodies diluted beyond neutralizing titers (Halstead and O'Rourke, 1977; Halstead et al., 1977).

The transfer of maternal neutralizing (Nt) antibody to a wide variety of viruses, including SLE virus, from adult female to offspring has been demonstrated in many animals (Kissling et al., 1954; Reeves et al., 1954; Sooter et al., 1954; Buescher et al., 1959; Bond et al., 1965; Jennings, 1969).

Between 1955 and 1971, nearly 50% of the human SLE virus cases in the United States occurred in a 1-mo period between July and August (Monath, 1980). This period corresponds to the period just following maximum house sparrow reproduction (Summers-Smith, 1963). Since human infections are closely related to the rate of vertebrate host infection, then ADE of SLE virus infections in nestling birds possessing maternal antibody might facilitate early virus transmission through increased amplification, i.e., an increased mosquito infection rate from feeding upon hosts with enhanced magnitude and duration of viremia. Such amplification could help explain the beginning of epizootics, or enzootics which occur in a given location every year.

This report describes the infection with SLE virus, of nestling house sparrows from both immune and nonimmune parents and the effect of passive neutralizing antibody upon their viremic response.

MATERIALS AND METHODS

Seventy-two house sparrows were captured in Japanese mist nets from two locations in Fort Collins, Colorado in October of 1982. Birds were chosen for experimentation based on sex and age. A ratio of 54% (38) males to 46% (34) females was used. This ratio was established as optimal in nature by Summers-Smith (1963) and in captive birds by Mitchell and Hayes (1973). Birds were aged by the pattern of cranial ossification (Nero, 1951). Birds greater than 1 yr of age were released.

Birds were transported to the Centers for Disease Control animal care facilities in Fort Collins. There, the birds were banded for identification and randomly assigned to cages. Cages were equipped with roosts, feed and water, and maintained at about 25 C and 70% relative humidity.

Initial blood samples from each bird were tested for the presence of Nt antibody and/or virus. Only birds tested and confirmed serologically negative for SLE virus were used.

Infection of adult sparrows

Birds were separated into equal experimental and control groups. Each bird from the experimental group was inoculated subcutaneously with 3.6 $\log_{10}$plaque-forming units (PFU) in LLC-MK_2 cells [4.0 $\log_{10}$50% suckling mouse intracerebral lethal doses ($SMICLD_{50}$)] of SLE virus, MSI-7 strain. Birds were bled daily for 10 days, then again at 29 days postinoculation (PI) to establish viremic and antibody responses. Virus (4.0 $\log_{10}$PFU) was again administered to the experimental birds on day 114 PI and day 367 PI.

Viremic enhancement studies

Following the 30-day acute infection study, adult birds were released into a mosquito-proof aviary. The SLE-infected (experimental) birds were separated from uninfected (control) birds for the duration of the experiment. Adult birds were bled at 60, 90, 164, 227, 367, 449, and 497 days PI.

During experimentally-induced breeding periods, birds were provided with nesting material and given a high protein diet. Nests were observed daily and egg laying, incubation, and hatching were recorded. Details on diets and procedures are given elsewhere (Ludwig, 1984).

Upon hatching, each brood was assigned randomly to an age group representing the age at which nestlings were to be challenged with virus. Age groups of 5–7, 8–10, 11–13, and 14–16 days were used. Upon reaching the proper age, all nestlings from each brood were bled and inoculated subcutaneously with 3.6 $\log_{10}$PFU of SLE virus, MSI-7 strain. Nestlings were bled daily for 7 days to determine viremic responses and measure antibody. Surviving hatchlings were bled twice monthly for 3 mo to follow Nt antibody development and persistence.

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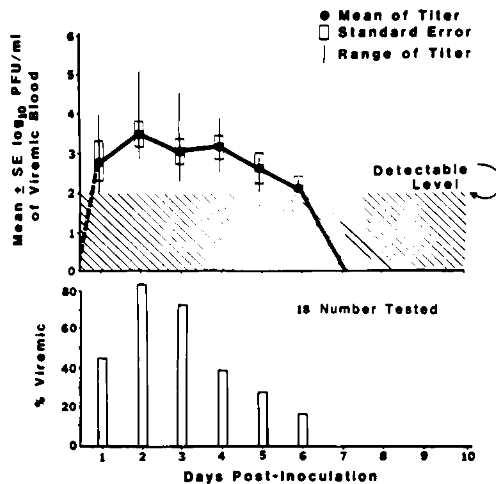

FIGURE 1. Viremia and percentage virus-positive results of adult female house sparrows after inoculation with St. Louis encephalitis virus.

Laboratory procedures

The MSI-7 strain of SLE virus utilized in this research was first isolated from the blood of a nestling house sparrow from Mississippi in 1975 (Bowen et al., 1980). Virus harvested from the fourth mouse brain passage was used in this research. The virus was prepared as a 10% clarified mouse brain suspension diluted in 4% bovine albumin in phosphate buffered saline (BAPBS). Virus stock was titrated in both cell culture and suckling mice with techniques described by Calisher and Poland (1980).

Virus isolations and assays from serum samples were carried out in monolayers of cell cultures (DETC, primary Pekin duck embryo cells; or LLC-MK_2, rhesus monkey kidney cells) according to procedures described by Scott et al. (1983). Some virus isolations were confirmed by mouse inoculation. Virus identification was confirmed by virus-dilution constant-serum protection tests (Calisher and Poland, 1980), and indirect fluorescent antibody tests (Monath et al., 1981).

Heat inactivated (56 C for 30 min) serum samples were screened for Nt antibody and positive serum titrated by constant-virus serum-dilution plaque reduction assays in Vero cells as described by McLean et al. (1983). We did not distinguish, serologically, passive from active antibody in individual serum samples. Procedures for the bleeding and handling of blood samples have been described (Scott et al., 1983; McLean et al., 1985).

Statistical analysis

Data were analyzed by Student's t-test or analysis of variance and Student-Newman-Keuls' multiple range test, depending on the means to be compared. Antibody data from experimental nestlings gathered prior to challenge with SLE virus were used to calculate a regression line and correlation coefficient (r) then tested by analysis of variance. All proportions were compared using the binomial expansion and distribution. Results were considered significant for probabilities less than or equal to 0.05 (alpha = 0.05).

RESULTS

Infection of adult sparrows

By day 2 PI 83% of all adult sparrows were viremic with a peak geometric mean (GM) titer of 3.40 ± 0.47 $\log_{10}$PFU/ml. By day 7 PI, none of the adult birds had detectable viremias (Fig. 1). Control sparrows, i.e., those not inoculated with virus, never had detectable viremias.

There were no significant differences in the temporal appearance, percentage positive, titers, or duration of Nt antibodies between male and female sparrows.

Nt antibody was first detected in adult female birds on day 4 PI, when a GM Nt antibody titer of 1:28.3 was observed. This amount of circulating antibody persisted until day 10 PI and rose to a titer of 1:117.9 on day 29 PI. Titers of antibody then decreased through day 90 PI. On day 164 PI, 50 days following the first SLE virus booster injection, all birds were antibody positive, and the GM antibody titer had increased to 1:175.5. Antibody titers did not change significantly through the remainder of the experiment (Fig. 2). All nestlings utilized in this study were hatched from eggs laid between 164 and 497 days PI.

5–7-day-old nestlings

Differences in GM virus titer and the proportion of virus-positive nestlings between treatment groups were significant. Only on days 1 and 2 did experimental nestlings possess lower viremias than did

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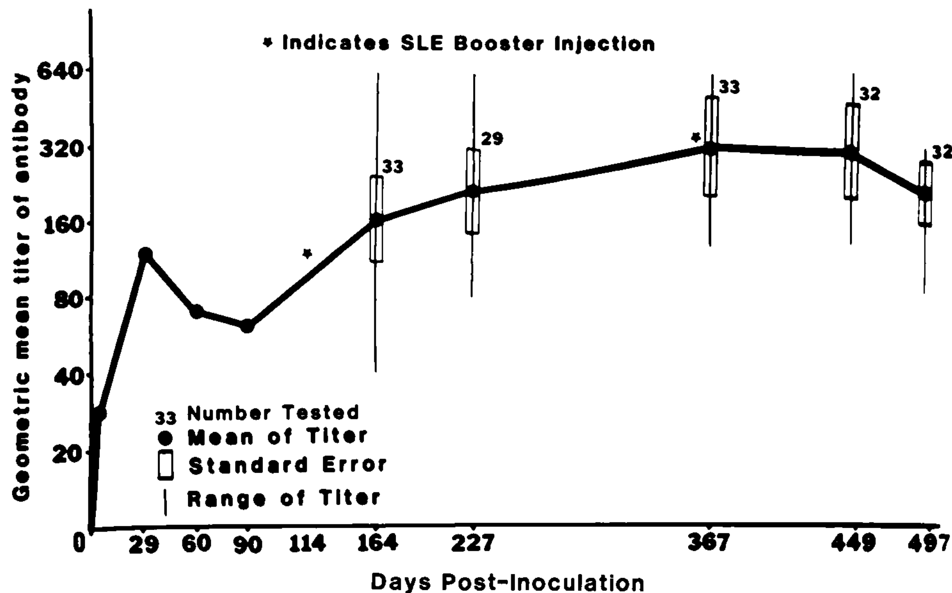

FIGURE 2. Antibody responses in adult female house sparrows for 500 days after inoculation with St. Louis encephalitis virus.

controls. On days 5–7 PI experimental nestlings had significantly greater viral titers than did controls. In addition, the proportion of virus-positive experimental birds was greater than controls on days 5, 6, and 7 PI (Fig. 3).

Antibody was present in experimental nestlings prior to infection with SLE virus. This antibody was assumed to be maternally acquired because of its appearance in nestlings prior to viral challenge and because nestlings from control groups had no detectable antibodies. Antibody to SLE virus in nestlings from the 5–7-day-old age group declined steadily, and decreased below detectable amounts by day 6 PI. Both control and experimental nestlings began producing antibody to SLE virus by day 7 PI. There was no significant difference in antibody development, persistence, or percentage positive between the treatment groups following day 6 PI.

Nestlings of intermediate age

There was no significant difference between treatment groups in the magnitude, temporal appearance, percentage positive, or duration of viremias in either the 8–10- or 11–13-day-old-age nestling cohorts. Antibody titers developed in a fashion similar to that of the younger age group.

14–16-day-old nestlings

There was no significant difference in viremia magnitude or temporal appearance between treatment groups of the 14–16-day-old age group except on day 2 PI when the GM virus titer of experimental nestlings was greater than that of control nestlings. The percentage of virus-positive nestlings was greater for experimental nestlings following day 2 PI (Fig. 4). Experimental nestlings were, on the average, viremic longer than control nestlings (Table 1).

Transovarially acquired antibody

All experimental nestlings from the 5–7- and 8–10-day-old age groups had Nt antibody prior to challenge with SLE virus. Experimental nestlings challenged 11–

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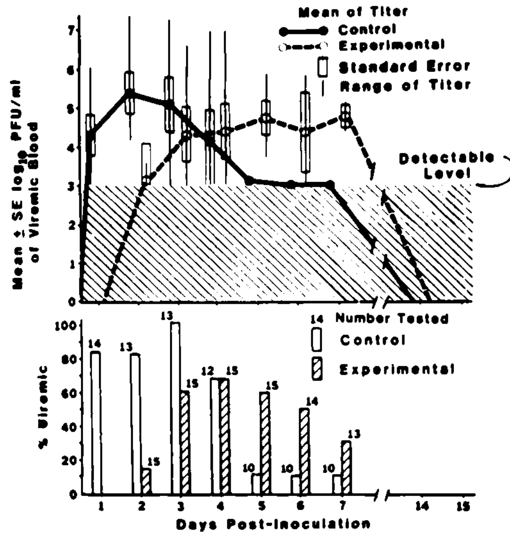

FIGURE 3. Viremia and percentage virus-positive results of 5–7-day-old house sparrows after inoculation with St. Louis encephalitis virus.

13 and 14–16 days posthatching (PH) were 58% and 17% Nt antibody positive on the day of virus challenge, respectively. No antibody was detected in control nestlings

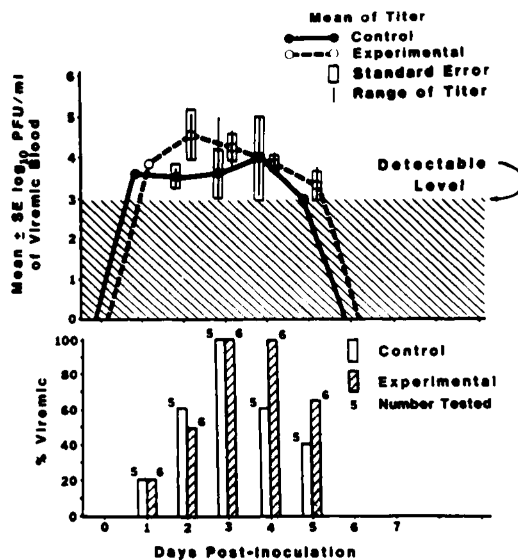

FIGURE 4. Viremia and percentage virus-positive results of 14–16-day-old house sparrows after inoculation with St. Louis encephalitis virus.

TABLE 1. The average duration of SLE viremias in control and experimental nestling house sparrows.

Age group (days)		Average duration of viremia (days ± SE)	Probability
5–7[a]	control	3.6 ± 0.34	>0.2
	experimental	2.85 ± 0.47	
8–10	control	4.38 ± 0.46	<0.001
	experimental	1.44 ± 0.44	
11–13	control	3.82 ± 0.38	<0.001
	experimental	1.75 ± 0.39	
14–16	control	2.5 ± 0.22	<0.01
	experimental	3.5 ± 0.22	

[a] Exact viremia endpoints are unavailable for the 5–7-day age group.

prior to challenge with SLE virus. Maternal antibodies detected in nestlings prior to SLE virus challenge declined below detectable amounts by 16 days PH (estimated by regression).

Average duration of viremia

There was no significant difference in the average number of viremic days between treatment groups of 5–7-day-old nestlings. However, individual control nestlings were viremic significantly longer than experimental nestlings for the 8–10-day-old and 11–13-day-old age groups. In contrast, experimental nestlings were viremic significantly longer than control nestlings (3.5 days versus 2.5 days) for the 14–16-day-old age group. There was no significant difference in average duration of viremia between 5–7-day-old control, 8–10-day-old control, 11–13-day-old control, or 14–16-day-old experimental nestlings (Table 1). Data on adults were unavailable for this parameter.

Average daily virus titer

Average daily virus titers were greatest in 8–10-day-old control nestlings, and least in adult sparrows. Among the nestling age groups, 14–16-day-old control nestlings had the lowest average GM virus titer (Table 2).

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DISCUSSION

Results from this research indicated that viremic enhancement occurred in two age groups. The 5–7- and 14–16-day-old experimental nestlings exhibited viremic enhancement by producing viremias of greater magnitude and duration than control nestlings of the same age.

Limitations in nestling blood sampling protocols prevented the direct observation of increased total viremia duration in 5–7-day-old nestlings, although results indicated that viremic enhancement in this form was highly probable. Trends in the development and decay of viremias in 5–7-day-old experimental nestlings, and their relatively high percentage of virus positives, even on days 5, 6, and 7 PI, indicated that virus titers would have probably remained high for several days subsequent to day 7 PI (Fig. 3). Virus titers in 5–7-day-old control nestlings, on the other hand, were extremely close to the lower limits of the assay system on days, 5, 6, and 7 PI, and have low percentage virus positives by day 7 PI (Fig. 3). Other nestling age groups with similarly low virus titers and percentages of virus-positive individuals, had their virus titers decline below detectable concentrations by the next day PI (data not given). These observations indicated that virus titers of control nestlings of the 5–7-day-old age group would probably not persist above detectable concentrations for any significant time subsequent to day 7 PI.

Although an increase in the average duration of viremia in 5–7-day-old experimental birds was not observed directly in this report, an increase in the duration of maximum virus titer was noted. An increase in the duration of high virus titers in the blood of infected hosts would appear to be significant for the occurrence of enhanced viral dissemination rates. Research completed by McLean et al. (1985) indicated that SLE virus titers in cardinals (*Richmondena cardinalis*) must reach 3.9 $\log_{10}$PFU/ml before more than 50% of vector mosquitoes feeding on these birds become infected. This report shows that control nestlings from the 5–7-day-old age group produce viremias that reached or exceeded the 50% vector infection threshold for only 4 days PI. In contrast, viremias from 5–7-day-old experimental nestlings reached or exceeded the 50% vector infection threshold for at least 5 days PI. A sparrow that maintains a high titer of virus in circulation for a long period of time would be expected to cause far more SLE virus infections in feeding vector mosquitoes than would a sparrow with a short duration, low magnitude SLE virus infection.

TABLE 2. The average daily SLE virus titer for the control and experimental treatment groups in each nestling age group.

Age group (days)	Average daily virus titer ($\log_{10}$PFU/ml ± SE)	Probability
5–7 control	4.76 ± 0.37	>0.5
experimental	4.66 ± 0.32	
8–10 control	5.17 ± 0.35	<0.001
experimental	3.92 ± 0.34	
11–13 control	4.30 ± 0.24	<0.05
experimental	4.02 ± 0.19	
14–16 control	3.59 ± 0.32	
experimental	3.94 ± 0.25	<0.05
Adult	3.01 ± 0.16	

ADE of virus infections has been shown to occur in the presence of low titers of group specific antibody (Halstead et al., 1980). Klicks and Halstead (1983) showed that in vitro enhancement of MVE virus infections did not occur until infective doses of virus were combined with chicken anti-MVE diluted to 1:3,160. Antibody against MVE virus seemed to exhibit neutralizing activity at high concentrations and enhancement at low concentrations. During an infection in passively protected, naive animals, this threshold antibody concentration could be reached as a result

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of natural decay processes prior to challenge or through viral neutralization following challenge.

During previous studies, passively acquired antibody has been shown to be the driving force behind enhanced viral infections in vertebrate hosts. Halstead (1979) showed that rhesus monkeys (*Macaca mulatta*) passively protected with antibody against dengue 1, 3, or 4 virus, and challenged with dengue 2 virus, always produced viremias of greater magnitude than animals that had received no antibody. Since in vitro studies have shown that ADE of virus infections occurs only when antibody titers against the virus are diluted past some threshold level, the temporal appearance, titer, and decay rate of passive antibody in live animals should play an important role in determining probabilities for the occurrence of ADE in these virus-infected hosts.

In the present study, passively acquired maternal antibody appeared in some of the experimental nestlings of all age groups. The duration of the appearance of maternal antibody in nestlings tended to be relatively short. A least squares regression line ($r = 0.997$, $P = 0.01$) fitted to the data indicated that Nt antibody to SLE virus should be eliminated from nestling circulation between 14 and 18 days PH. These results were consistent with results observed by Bond et al. (1965) in which passively acquired hemagglutination-inhibition (HI) antibody to SLE virus was eliminated by day 22 PH in chicks.

Due to the administration of booster injections to adult birds, we assume that the Nt antibody titer maintained in adult females throughout the experiment represents the maximum obtainable titer for birds from the population utilized and infected as described. Also we assume that the calculated GM titer and duration in days of passively acquired Nt antibody in nestlings recently hatched was also at its maximum. Geometric mean titers and duration of maternal antibody in chicks were directly proportional to the GM titers of the adult females (Reeves et al., 1954; Bond et al., 1965).

The delay in developing viremia observed in experimental nestlings was most likely a result of the presence of passively acquired Nt antibody in these birds. Viremias developed presumably only after antibodies were complexed with viral antigens. Delay of viremia was not observed in older nestlings, probably as a result of the natural decay of passively acquired antibody below protective concentrations prior to virus challenge. Another possible explanation for the delay of viremia in 5–7-day-old nestlings is that immune complexes formed between virus and passive antibody may not have been detectable with the assay system utilized. These undetectable complexes may be infectious to vector mosquitoes, thus increasing chances for viral amplification.

From the results observed during this research, it is difficult to determine the exact mechanism for the observed enhancement of nestling virus infections. Although ADE is implied as a possible mechanism, the observed enhancement of viral infections in the presence of relatively high titers of passive Nt antibody is inconsistent with previous in vitro studies. We have attempted to explain this inconsistency through the discussion of passive antibody decay to enhancing concentrations during the early stages of viral infection as a result of virus neutralization. It may be, however, that the enhancement observed during this research was caused by a different mechanism, such as the mediation by interferon of virus replication and the host's immune response leading to a limited, steady state infection (Mims and White, 1984). Further research is needed to determine the exact mechanism leading to enhanced SLE virus infections in birds.

Nestling house sparrows between 5 and

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10 days old, regardless of the presence or absence of maternal antibody, produce viremic responses to SLE virus infection of greater magnitude and duration, and maintain greater percentage virus positives for longer periods of time than do older nestlings or adults. These observations indicate that younger nestlings present a greater threat as an age-specific amplifying reservoir for SLE virus than do nestlings infected at older ages, and certainly a greater threat than viremic adults. The threat of virus amplification in these younger nestlings is enhanced by the observation that 5–10-day-old house sparrows are nearly featherless and extremely helpless, possibly making them more vulnerable to vector mosquitoes as sources of blood meals. Although viremic enhancement was observed in 14–16-day-old nestlings, the relative importance of such enhancement is probably low. Viremia magnitude and duration was shown to be less in 14–16-day-old experimental nestlings than in any younger age cohort from either experimental or control groups. Results from this study indicate that 14–16-day-old nestlings undergoing an enhanced SLE virus infection, would probably contribute relatively little to virus amplification and dissemination rates in nature.

Young nestlings possessing passively acquired antibody to SLE virus may exhibit viremic enhancement as described previously. In the presence of such enhancement, these nestlings would surely increase the infection rate of vector mosquitoes. SLE antibody prevalence rates in several species of birds from several different serological surveys range from 3.0% to 56.5% (McLean, 1980). The relative contribution of viremic enhancement in the maintenance of enzootics or the beginning of epizootics may be extremely high during periods of high antibody prevalence in bird populations. If the presence of a high density, highly mobile, active, and susceptible mosquito population coincided with SLE virus enhancement in a population of nestling sparrows, the chances of further virus amplification and dissemination would be greatly increased. Increased viral amplification and dissemination rates are two of the most important variables leading to epizootics of SLE virus, and can be major contributors to the infection of aberrant hosts such as humans. Viremic enhancement of SLE virus has the potential to influence the beginning of epizootics/epidemics and the further maintenance of enzootic foci.

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