Landscape Ecology of Arboviruses in Southern California: Patterns in the Epizootic Dissemination of Western Equine Encephalomyelitis and St. Louis Encephalitis Viruses in Coachella Valley, 1991–1992

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ABSTRACT Temporal and spatial patterns in the initiation and dissemination of western equine encephalomyelitis and St. Louis encephalitis virus activity in Coachella Valley during 1991 and 1992 were detected by testing pools of host-seeking *Culex tarsalis* Coquillett for virus infection and sentinel chickens for seroconversions. Both viruses repeatedly were detected first at a salt marsh adjacent to the Salton Sea in the southeastern corner of the study area and then disseminated to the northwest to freshwater marsh, agricultural, and residential habitats. Virus dissemination was relatively slow (<1 km/d) and may have been accomplished by dispersive host-seeking mosquitoes. Repeated early-season recovery of virus activity indicated that both viruses may persist interseasonally in salt marsh habitat.

KEY WORDS western equine encephalomyelitis virus, St. Louis encephalitis virus, interseasonal persistence

THE GEOGRAPHICAL DISTRIBUTION of most vector-borne pathogens expands during conditions that are favorable to their host populations and contracts during conditions that are unfavorable. Refugia that permit host survival during unfavorable periods are essential to pathogen persistence and may be identified by examining the distribution of host infection rates over time and space. This approach to the recognition of enzootic foci may be especially applicable to the North American mosquito-borne encephalitis viruses, which spend much of their life history in infected mosquito vectors, vary markedly in occurrence over time and space, and sporadically produce human disease.

Enzootic activity of western equine encephalomyelitis (WEE) and St. Louis encephalitis (SLE) viruses is detected during most summers in southern California by testing either pools of the primary mosquito vector, *Culex tarsalis* Coquillett, for infectious virus or sera from sentinel chickens for antibody (Meylan et al. 1989, Reisen et al. 1992a). However, the mechanism(s) that allow the interseasonal persistence of these viruses remains obscure. Studies along the Colorado drainage and in the Imperial and Coachella valleys of California failed to document a gradual south-to-north progression in the initiation of virus activity (Work et al. 1977, Reisen et al. 1992a), indicating that virus may not be introduced from Mexico as a slow-moving epizootic. Positive mosquito pools and sentinel seroconversions have been detected sporadically during winter; however, a marked change in vector age structure (Reisen et al. 1995a) frequently is associated with an interruption of horizontal virus transmission.

An intensive examination of virus activity in Coachella Valley delineated consistent patterns of virus initiation, amplification, and dissemination that provided insight into the refugia responsible for virus interseasonal persistence. The purpose of the current study was to describe these patterns and grossly estimate the rate of WEE and SLE virus dissemination in Coachella Valley, Riverside County, California. These analyses extended the data interpretation of Reisen et al. (1995b) and attempted to identify the enzootic focus of annual virus initiation.

Materials and Methods

A description of the southern Coachella Valley, the juxtaposition of sampling sites to terrain features including the Salton Sea and Whitewater Channel, and the seasonality of temperature and precipitation were described previously (Reisen et al. 1995b). A map of the southern Coachella Valley showing the location of our 19 study sites was presented in Fig. 1 of Reisen et al. (1995b). Sites 16–19 positioned along an east-to-west transect ≈10 km north of site 13 remained negative for virus activity and are not shown in Figs. 1–4. The study area encompassed 1,250 km^2 and was demarcated by the Salton Sea to the south, the Mecca Hills to the east, the Santa Rosa and San Jacinto mountains to the west, and desert to the north of Palm Springs. The maximum distance between sites 7 and 19 was 62.5 km.

Virus activity was monitored at each site by testing pools of host-seeking *Cx. tarsalis* females for

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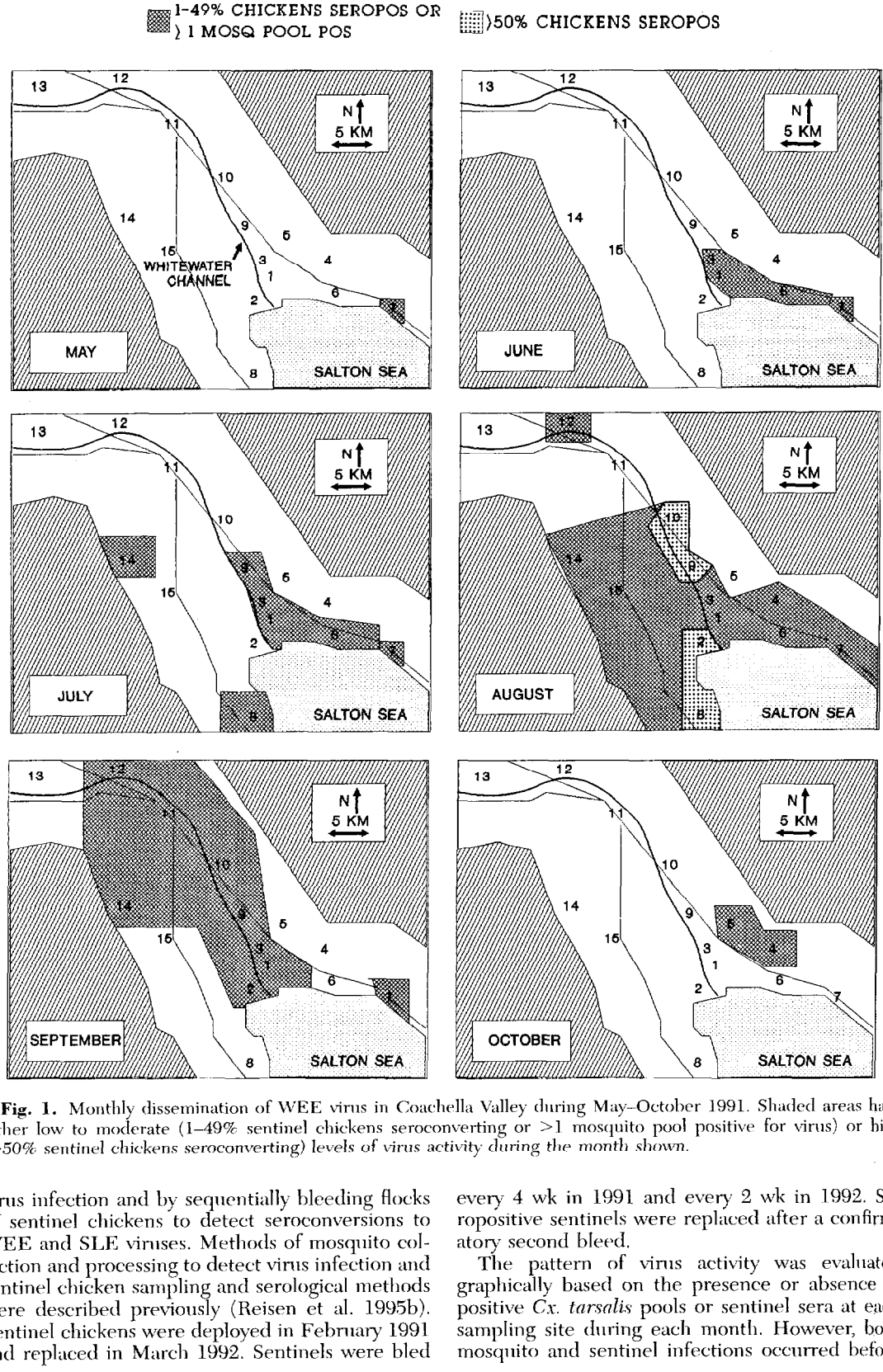

Fig. 1. Monthly dissemination of WEE virus in Coachella Valley during May–October 1991. Shaded areas have either low to moderate (1–49% sentinel chickens seroconverting or >1 mosquito pool positive for virus) or high (>50% sentinel chickens seroconverting) levels of virus activity during the month shown.

virus infection and by sequentially bleeding flocks of sentinel chickens to detect seroconversions to WEE and SLE viruses. Methods of mosquito collection and processing to detect virus infection and sentinel chicken sampling and serological methods were described previously (Reisen et al. 1995b). Sentinel chickens were deployed in February 1991 and replaced in March 1992. Sentinels were bled every 4 wk in 1991 and every 2 wk in 1992. Seropositive sentinels were replaced after a confirmatory second bleed.

The pattern of virus activity was evaluated graphically based on the presence or absence of positive *Cx. tarsalis* pools or sentinel sera at each sampling site during each month. However, both mosquito and sentinel infections occurred before

Table 1. WEE and SLE virus dissemination in Coachella Valley, 1991–1992

Virus	Dates[a]	Sites[b]	Distance, km	Rate, km/d
	From–to	From–to		
WEE, 1991	29 May–25 June	7–3	16.3	0.6
	25 June–23 July	3–14	12.5	0.4
	23 July–20 Aug.	14–11	12.5	0.4
WEE, 1992	27 July–10 Aug.[c]	7(8)–3	16.3 (8.8)	1.2 (0.6)
SLE, 1991	23 July–23 Aug.	7–3	16.3	0.5
	20 Aug.–17 Sept.	3–10	6.3	0.2
SLE, 1992	1 June–15 June	8–7	18.0	1.3
	15 June–24 June	7–6	7.5	0.8
	15 June–29 June	8–2	5.0	0.4
	27 July–5 Aug.	2–15	11.2	1.2
	27 July–10 Aug.	2–12	30.0	2.1

[a] Chickens bled every 4 wk in 1991 and every 2 wk in 1992.
[b] Movement between closest sites.
[c] Direction of movement uncertain.

detection by our surveillance. During summer, *Cx. tarsalis* has a minimum gonotrophic cycle duration of 4 d (Reisen et al. 1983), and therefore positive pools contained >1 female that had fed on a viremic host >4 d before collection. Adult chickens were tested by enzyme immunoassay for IgG antibodies that were first detectable 8 and 10 d after infection with WEE and SLE viruses, respectively (Reisen et al. 1993a). With a 28-d bleeding schedule, sentinels therefore may have been infected with WEE and SLE viruses during the previous 8–36 or 10–38 d, respectively; with a 14-d bleeding schedule, this period was shortened to 8–22 or 10–24 d, respectively. Mosquito pools were less sensitive than sentinel seroconversions in the detection of virus activity (Reisen et al. 1995b), and therefore the current analysis emphasized sentinel seroconversion rates. Virus activity was considered to be elevated when seroconversion rates exceeded 50% per flock per sampling interval. Virus dissemination or dispersal rates were calculated as the distance between the farthest previous and new positive sites divided by days between sampling dates.

Results

Western equine encephalomyelitis virus was active in Coachella Valley during 1991 and 1992. Of 581 (25,466 females) and 819 (34,805 females) pools of *Cx. tarsalis* tested for virus, 15 and 4 pools were positive for WEE virus in 1991 and 1992, respectively. In addition 87 (5.3%, n = 1,653 sera) and 16 (0.6%, n = 2,671 sera) sentinel chicken sera were positive for WEE virus antibody in 1991 and 1992, respectively. Activity commenced at site 7 on 29 May 1991 when one of nine pools was positive for WEE virus (Fig. 1). Virus then spread west along the shore of the Salton Sea to duck club habitat along the Whitewater Channel floodplain, with sentinel seroconversions detected at sites 1, 3, and 6 on 25 June. Maximum distance of virus dissemination was 16.3 km between sites 7 and 3 (Table 1). Virus activity continued at these sites during July and also spread 12.5 km to the west to site 14 and perhaps 7.5 km south to site 8. *Cx. tarsalis* infection rates were especially high at site 8 during July, when six of six pools collected on 3 July and one of one pools collected on 17 July were positive for WEE virus (minimum infection rate [MIR] = 20.6 infected females per 1,000 tested). By August, virus was active at all sites except site 5 within the town of Mecca and at northern sites 11 and 13, and was transmitted intensively at sites 8, 2, 9, and 10, where >50% of the sentinels seroconverted (Fig. 1, August). The farthest dispersal distance was 17.5 km from site 14 to site 12. By September, WEE activity had begun to subside and was not detected at formerly positive sites 8, 15, 4, and 6. WEE virus was detected for the first time at site 11 on 17 September. By October all sites were negative except for one seroconversion at site 5 and one positive pool at site 4 in citrus and grape agricultural habitat. A final seroconversion occurred at site 5 in November.

Although one of 10 pools collected on 27 February 1992 was positive for WEE virus (MIR = 2 per 1,000) at site 9 (5 km west of site 5), amplification apparently did not commence until late May, when single seroconversions were detected at sites 5 and 6 (Fig. 2). During June, transmission was most intense at site 7, where six of nine sentinels seroconverted on 29 June; however, activity remained confined to sites 7 and 8 during both June and July 1992. Activity then spread to sites 1–4 and 10 during August, but was not detected during September, even though *Cx. tarsalis* abundance was elevated in this area at this time (Reisen et al. 1995a). All sites north of site 10 remained negative during 1992. Final activity was detected at site 2 when one of 10 pools were WEE virus positive on 14 October (MIR = 2 per 1,000).

St. Louis encephalitis virus also was active in Coachella Valley during 1991 and 1992. Overall, four (MIR = 0.16 per 1,000) and four (MIR =

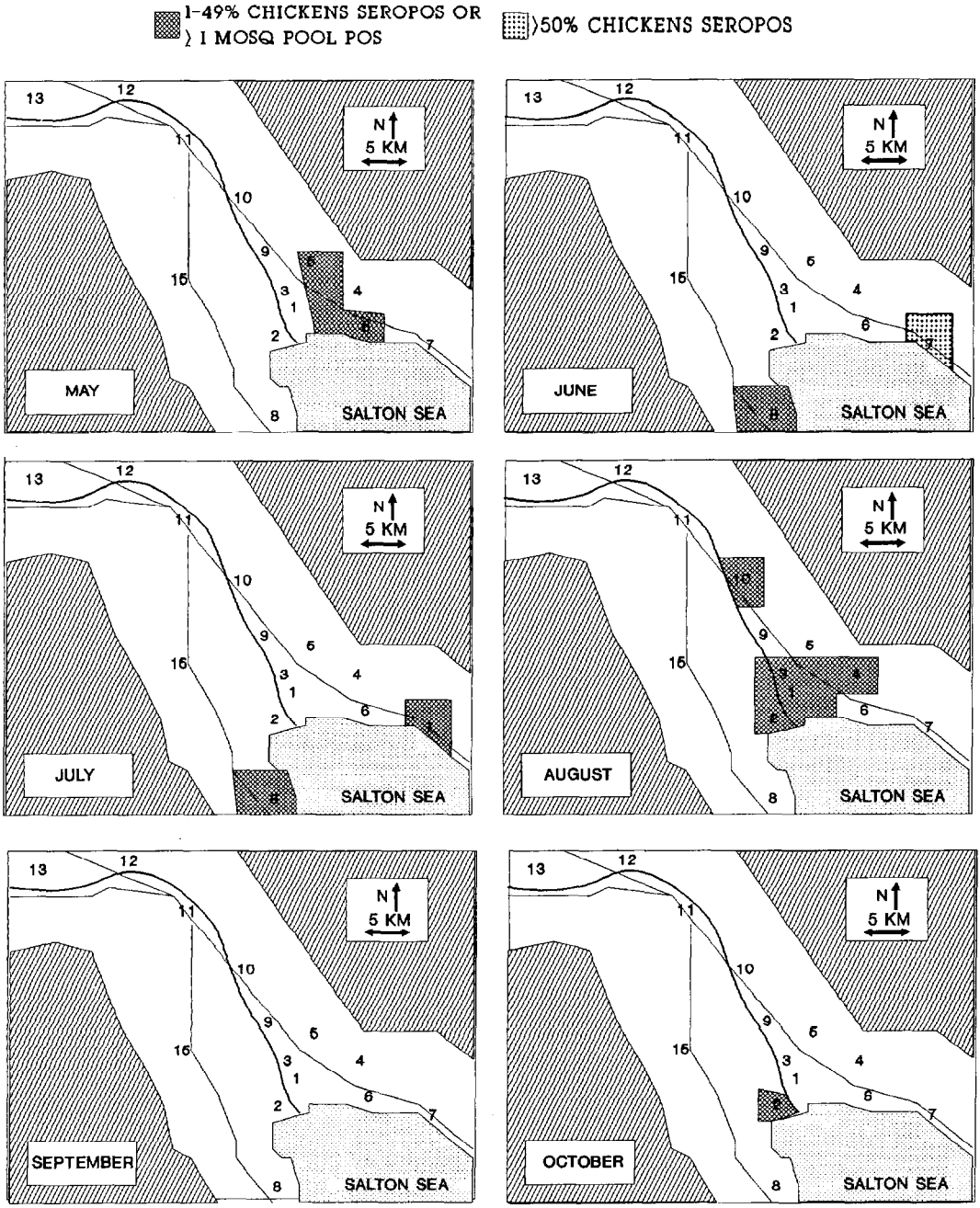

Fig. 2. Monthly dissemination of WEE virus in Coachella Valley during May–October 1992. Shaded areas have either low to moderate (1–49% sentinel chickens seroconverting or >1 mosquito pool positive for virus) or high (>50% sentinel chickens seroconverting) levels of virus activity during the month shown.

0.11 per 1,000) pools of *Cx. tarsalis* were positive and 77 (4.6%, n = 1,669 sera) and 49 (1.9%, n = 2,610) sentinels seroconverted to SLE virus during 1991 and 1992, respectively. Although SLE virus first appeared 6 wk later than WEE virus, activity commenced at site 7, where three of 10 sentinels bled on 23 July were seropositive (Fig. 3). By August, virus disseminated westward to sites 1, 3, 4, and 6, and perhaps to the southwest to site 8. The maximum distance dispersed was 16.3 km between sites 7 and 3 (Table 1). Virus activity spread to sites 2, 9, and 10 during September and was most intense at sites 3, 4, 8, and 9, where >50% of the sentinels seroconverted. Although the intensity of transmission declined in October, seroconversions were detected at several sites in the southeastern

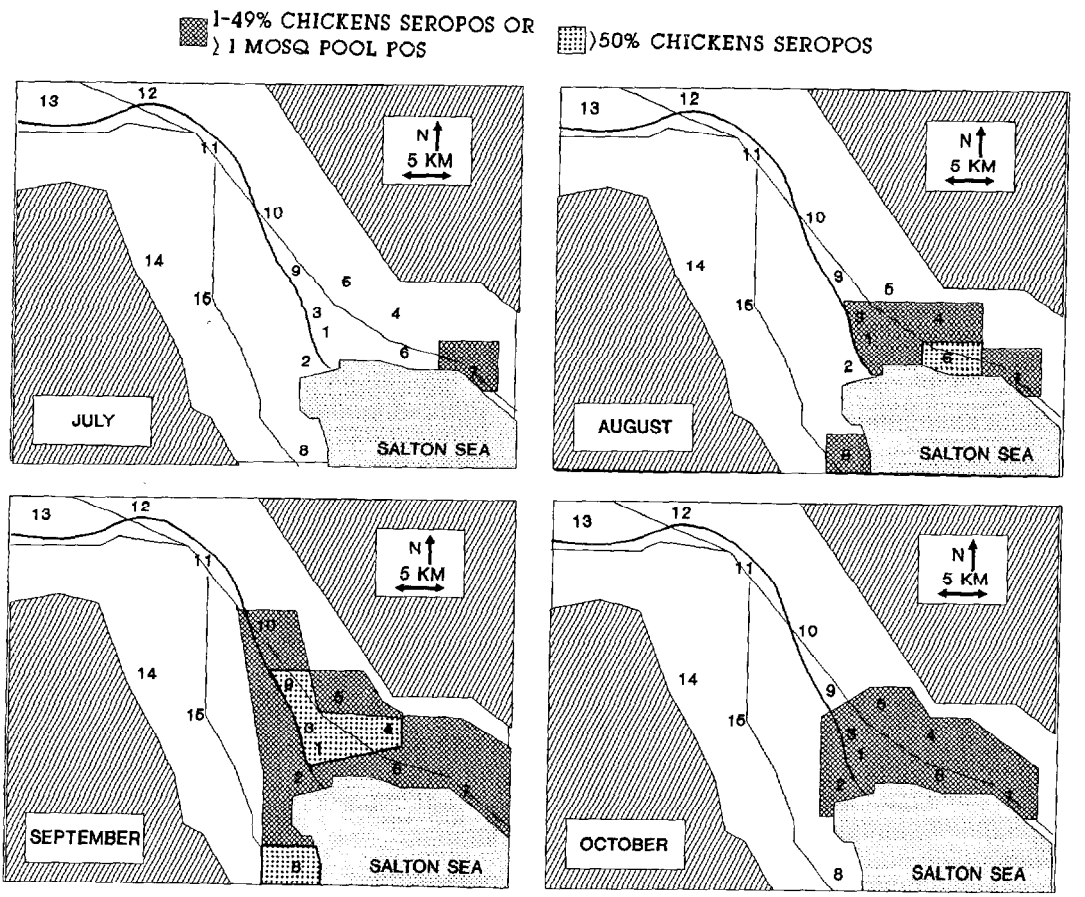

Fig. 3. Monthly dissemination of SLE virus in Coachella Valley during July–October 1991. Shaded areas have either low to moderate (1–49% sentinel chickens seroconverting or >1 mosquito pool positive for virus) or high (>50% sentinel chickens seroconverting) levels of virus activity during the month shown.

portion of the study area (Fig. 3). Single seroconversions occurred at site 6 in November and at site 12 in December.

Although single seroconversions to SLE virus were detected on 6 January 1992 at site 7 and site 14, virus was not detected again until late May, when two chickens at site 8 had seroconverted when bled on 1 June 1992 (Fig. 4). Low-level virus activity then was detected by additional seroconversions at sites 7 and 8 on 15 June and at sites 2, 7, and 8 on 29 June; a single pool of *Cx. tarsalis* collected on 24 June from site 6 was positive for SLE virus. Movement from site 8 to site 7 along the margin of the Salton Sea was 18 km or 1.3 km/d (Table 1). Low-level virus activity continued along the margin of the Salton Sea in July. By August, virus had spread 11.2 km to the northwest to site 15 and 30.0 km to site 12 near Indio. *Cx. tarsalis* abundance at site 15 remained low throughout 1992; however, a single pool of nine females collected on 5 August was positive for SLE virus. By September, virus had spread 5.5 km to site 9 and 3 km to site 5 in the town of Mecca. SLE virus activity decreased along the margin of the Salton Sea during October and was not detected at sites 1, 7, and 8, whereas activity continued at site 12 near Indio. SLE virus was not detected at sites 16–19 north of site 12. Final single seroconversions were detected on 2 November at sites 4 and 6.

Discussion

Virus Distribution. The pattern of WEE and SLE virus initiation, amplification, and dissemination was similar during both 1991 and 1992. WEE virus in 1991 and 1992 and SLE virus in 1992 were first detected at site 7 by virus-positive *Cx. tarsalis* pools or by sentinel seroconversions. Early-season WEE virus activity also was noted at the adjacent town of Desert Beach in 1987 (Durso & Burguin 1988, Emmons et al. 1988). In 1992, SLE virus appeared first at site 8 on 1 June, but by 15 June sentinels also were seropositive at site 7; these birds may have been infected from 10 to 24 d previously. Both sites 7 and 8 were situated in the southernmost portion of Coachella Valley and were adjacent to salt marsh habitat along the northern

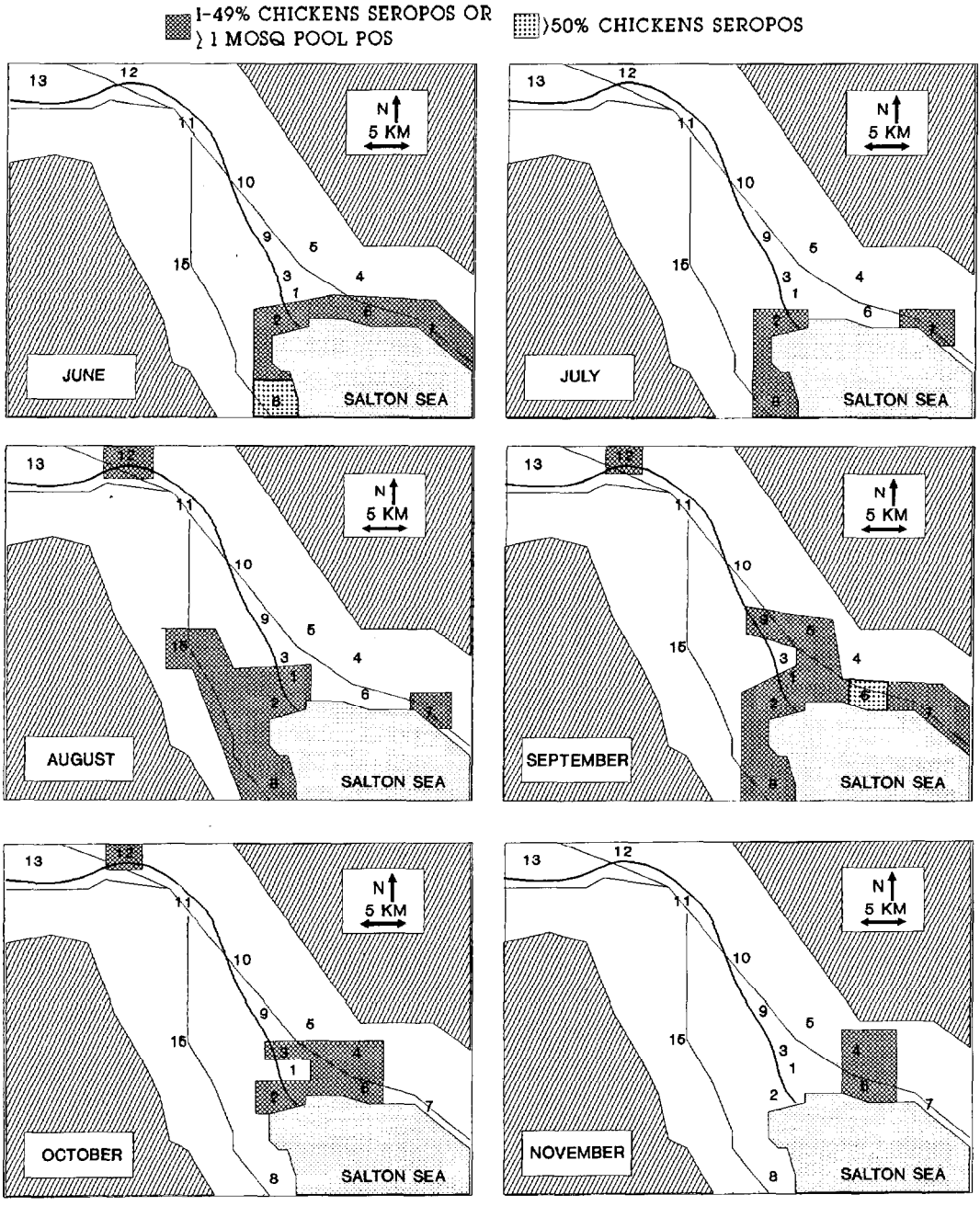

Fig. 4. Monthly dissemination of SLE virus in Coachella Valley during June–November 1992. Shaded areas have either low to moderate (1–49% sentinel chickens seroconverting or >1 mosquito pool positive for virus) or high (>50% sentinel chickens seroconverting) levels of virus activity during the month shown.

shore of the Salton Sea. This marsh was inundated by saline seepage from the Salton Sea during late winter and early spring and produced elevated vernal populations of *Cx. tarsalis* (group A [Reisen et al. 1995a]). During 1992, *Cx. tarsalis* populations also were abundant at sites 1 and 2 from January through April, but then crashed dramatically in May, whereas populations at site 7 remained abundant through May and at site 8 through June. Elevated temperature in May and June may be critical for efficient virus transmission by reducing the duration of the extrinsic incubation period (Reisen et al. 1993a) and the gonotrophic cycle (Reisen et al. 1992a).

After amplification at sites 7 and 8, virus then disseminated into the floodplain of the Whitewater

Channel (group B [Reisen et al. 1995a]). Site 2, a managed freshwater marsh adjacent to the Salton Sea, supported large *Cx. tarsalis* and diverse bird populations, but the appearance of WEE and SLE virus frequently was delayed until after being generally disseminated throughout the Whitewater floodplain. Virus then spread northward, but usually remained at sites below sea level within the floodplain and only occasionally involved sites 14 and 15 on the west side and sites 11 and 12 to the north of the valley.

Virus was never detected at sites 13 and 16–19, which were >80 m above sea level and situated in the northern portion of the study area in sandy, well-drained soil (group C [Reisen et al. 1995a]). Even though *Cx. tarsalis* abundance was routinely low (<4 females per CO_2 trap night per season [Reisen et al.1995a]), site 12, situated adjacent to a large citrus orchard, supported the enzootic transmission of WEE virus in 1991 and SLE virus in late 1991 and 1992. This site was ≈7 km northwest of site 11 and ≈7 km east of site 13, neither of which supported comparable levels of virus activity. WEE virus was detected previously in similar flood-irrigated citrus orchard habitat ≈2 km to the south during 1987 (Durso & Burguin 1988). Perhaps large passeriform populations roosting in *Tamarix* windbreaks and citrus supported relatively efficient virus transmission at low vector abundance during summer.

The association of arbovirus activity with specific foci has been demonstrated previously for other mosquito-borne encephalitis viruses, usually because of the habitat requirements of the vector. Foci of eastern equine encephalomyelitis (EEE) virus, for example, usually are associated with bogs within deciduous forest, the preferred habitat for the primary enzootic vector, *Culiseta melanura* (Coquillett) (Morris 1988). Extreme focality, however, is recognized more frequently for transovarially maintained viruses such as LaCrosse encephalitis, which may reappear annually at specific tree holes, the breeding site of the vector *Aedes triseriatus* (Say) (DeFoliart 1983). However, previous attempts to delineate overwintering foci for WEE or SLE viruses have not been successful, because of their sporadic and frequently widespread occurrence. *Cx. tarsalis* typically seeks hosts along riparian vegetation (Meyer et al. 1991), and the distribution of human and equine cases has been associated with irrigated agricultural valleys along riparian habitats (Mitchell 1977).

Identification of Overwintering Foci. The repeated early-season activity of WEE and SLE viruses at site 7 and perhaps site 8 indicated that virus consistently may be reintroduced into or persist in this salt marsh habitat. During 1991 and 1992, WEE virus was first detected during the end of May, whereas SLE virus was detected later, in July 1991 and in mid-June 1992. Consistent reintroduction seems an unlikely mechanism to explain this pattern, because virus usually appears in Coachella Valley earlier than in Imperial Valley to the south (Lothrop et al. 1994) and because most south–north bird migrations are completed by late spring. Although 370 bird species have been recorded from the Salton Sea area, this fauna is reduced by more than half during the hot summer period. Although many water and shore birds are permanent residents, summer passeriform diversity is reduced markedly, but includes the important virus hosts *Passer domestica* L. and *Carpodacus mexicanus* (Say).

Although overwintering along the margin of the Salton Sea seems likely, the mechanism(s) employed and reasons behind the consistent timing of vernal detection of virus remain unclear. Overwintering by continued low-level horizontal transmission among *Cx. tarsalis* and wild birds was supported by the occasional but repeated detection of infected *Cx. tarsalis* or sentinel seroconversions during winter and by the early-season detection of virus at or near sites last positive during the previous season (e.g., WEE virus at sites 5 and 6 after late-season activity near site 5 in 1992). Similar virus carry-over also was detected after the SLE epidemic in the San Joaquin Valley when late-season activity at the Kern National Wildlife Refuge in 1989 was followed by late winter seroconversions in 1990 (Reisen et al. 1992c). Similar to activity at the Kern Refuge, early-season virus activity near site 5 terminated during spring when virus appeared at sites 7 and 8.

The appearance of WEE virus in *Cx. tarsalis* and sentinels in late May–early June was well after the onset of *Cx. tarsalis* gonotrophic activity in January (Reisen et al. 1995b) or the renewal of passeriform reproductive activity in February–March. Detection of WEE virus in this *Cx. tarsalis*-bird cycle could be delayed if WEE virus were to overwinter in *Aedes dorsalis* (Meigen) eggs. NJ light trap records of the Coachella Valley Mosquito Abatement District indicate that there is a consistent focal increase in *Ae. dorsalis* females near site 7 during late March–early April. Vernal amplification of WEE virus in an *Ae. dorsalis*-mammal cycle at that time and the subsequent spread to the *Cx. tarsalis*-bird cycle may take several generations. This delay could result in the first detection of WEE virus in sentinel chickens in June. A similar overwintering mechanism for WEE virus was suggested previously to explain the appearance of WEE virus activity in Canada before the termination of *Cx. tarsalis* diapause (McLintock et al. 1970). Recently, Fulhorst et al. (1994) isolated WEE virus from *Ae. dorsalis* collected as immatures at a salt marsh in Morro Bay, CA. These were the first field isolations of vertically transmitted WEE virus and the first isolation of WEE virus from *Ae. dorsalis* in California. Infected adult *Ae. dorsalis* have been collected previously from Saskatchewan (McLintock et al. 1970), Utah (Smart et al. 1972), and Arizona (Hayes et al. 1976). Although rarely abundant, inland populations of *Ae.*

dorsalis persist in Coachella and Imperial valleys. If WEE virus overwinters in diapausing *Ae. dorsalis* eggs, this may explain the consistent reappearance of WEE virus at salt marsh habitat in late spring.

The mechanism responsible for the persistence of SLE virus seems to be the slow, but continuous, horizontal transmission of virus among *Cx. tarsalis* and birds. Continued transmission was supported by repeated winter seroconversions by sentinels. SLE virus requires considerably warmer temperatures for replication than WEE virus and does not replicate in *Cx. tarsalis* below 16°C (Reisen et al. 1993b). Bellamy et al. (1968) demonstrated that quiescent *Cx. tarsalis* remained infective with SLE virus for life and could transmit virus after an enlonged winter period. Warm periods in Coachella Valley during February and March could initiate virus replication in overwintering infected vectors. In addition, Hardy et al. (1980) demonstrated low-level vertical transmission of SLE virus by *Cx. tarsalis* at cool (18°C) water temperatures. Possibly, low-level vertical transmission in *Cx.tarsalis* developing during winter followed by slow vernal amplification may result in the June-to-July appearance of SLE virus in Coachella Valley.

Virus Dissemination. The spread of virus along the shore of the Salton Sea to the rest of Coachella Valley proceeded gradually (<1.3 km/d). With the exception of SLE virus moving ≈30 km in 14 d from site 2 to site 12 in July 1992, dissemination usually was <18 km to adjacent sites. This gradual spread could be accomplished by infected host-seeking *Cx. tarsalis*, because marked host-seeking females were recaptured after dispersing from site 2 to sites 8, 3, and a site ≈3 km west of site 6 within 3 d of release (W.K.R. & H.D.L., unpublished data). This 3-d period was well within the duration of a single gonotrophic cycle, the first opportunity for virus transmission. Presumably bird populations during summer utilize the same nocturnal roosts, thereby reducing virus dissemination by infected avian hosts. In contrast, virus movement within the San Joaquin Valley during summer epizootics appeared to be more rapid than in Coachella Valley, with WEE virus appearing widespread within 3 wk of initial detection (Reisen 1984) and with SLE virus detected at three sites spread over 50 km within 1 wk of initial detection (Reisen et al. 1992c). Virus dissemination in the San Joaquin Valley followed years of low or no virus activity and presumably required single or multiple introductions.

Additional research is required to investigate more fully the mechanisms by which viruses persist in and around the salt marsh habitat near site 7 in Coachella Valley. Detailed studies are needed on the bionomics and population dynamics of *Ae. dorsalis* and the ability of this inland population to transovarily transmit WEE virus. In-depth studies also are needed on the ecology of the bird fauna of the Salton Sea and the possible role of relapsing chronic infections in virus persistence and dissemination.

Acknowledgments

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