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ANNUAL SURVIVAL OF HOUSE FINCHES IN RELATION TO WEST NILE VIRUS

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Abstract. From 2001 to 2008, we estimated probabilities of survival and encounter of adult House Finches (*Carpodacus mexicanus*) breeding at Stone Lakes National Wildlife Refuge, Sacramento County, California, from capture–recapture data on birds trapped with mist nets and ground traps. Our primary objectives were to determine when West Nile virus (WNV, Flaviviridae, *Flavivirus*) arrived at the site and if survivorship changed after this arrival. We monitored viral activity by screening blood samples from House Finches for WNV antibodies with an enzyme immunoassay and by testing mosquito pools for viral RNA. WNV arrived after the breeding season in late 2004, so we compared data from 2001–2004 (pre-WNV) to that from 2005–2008 (post-WNV). We found a decrease in annual survival following the arrival of WNV (pre-WNV, 0.59; post WNV, 0.47), which, if representative, may have contributed to the reported decline in the abundance of this species in northern California.

Key words: adult survival, Carpodacus mexicanus, *House Finch*, mark–recapture, *West Nile virus.*

Supervivencia Anual de *Carpodacus mexicanus* con Relación al Virus del Oeste del Nilo

Resumen. Desde 2001 a 2008, estimamos las probabilidades de supervivencia y encuentro de adultos de *Carpodacus mexicanus* que anidan en el Refugio Nacional de Vida Silvestre Stone Lakes, condado de Sacramento, California, a partir de datos de captura-recaptura de aves atrapadas con redes de niebla y trampas terrestres. Nuestros objetivos principales fueron determinar cuándo el virus del Oeste del Nilo (VON, Flaviviridae, *Flavivirus*) llegó al sitio y si la supervivencia cambió luego de este arribo. Seguimos la actividad del virus analizando muestras de sangre de *C. mexicanus* para anticuerpos de VON con un ensayo inmunológico enzimático y evaluando la presencia de ARN viral en charcas de mosquitos. El VON llegó luego de la estación de cría a fines de 2004; comparamos los datos de 2001–2004 (pre-VON) con los de 2005–2008 (post-VON). Encontramos una disminución en la supervivencia anual luego del arribo del VON (pre-VON, 0.59; post-VON, 0.47), la cual, si es representativa, puede haber contribuido a la disminución reportada en la abundancia de esta especie en el norte de California.

INTRODUCTION

West Nile virus (WNV, Flaviviridae, *Flavivirus*) is an emergent zoonotic pathogen that was introduced into the United States at New York City in 1999 and has rapidly invaded the New World (Kramer et al. 2008). Infections have been detected in a wide variety of vertebrates, although a relatively few species of birds serve as maintenance and amplifying hosts (Kilpatrick et al. 2007, Allan et al. 2009). Transmission occurs primarily through *Culex* mosquitoes as vectors, although evidence of transmission by soft-bodied ticks (Hutcheson et al. 2005), direct transmission through fecal material (Kipp et al. 2006), allopreening (Ward et al. 2006), and ingestion of infected carrion or prey (Peterson et al. 2004) has been documented, even during winter (Dawson et al. 2007). In both laboratory experiments (Komar et al. 2003a, Reisen et al. 2005a) and wild populations (Komar et al. 2003b) WNV causes mortality in multiple species of birds, and it appears to have reduced many wild populations (Koenig et al. 2007, LaDeau et al. 2007, Crosbie et al. 2008, Wheeler et al. 2009).

The House Finch (*Carpodacus mexicanus*) is a competent host of WNV (Reisen et al. 2005a, Kilpatrick et al. 2007) that is fed upon frequently by *Culex* vector mosquitoes (Tempelis et al. 1976) and is considered to be important in WNV transmission (Reisen et al. 2005a). It has also been listed, through analysis of trends in population abundance, as a species reduced by WNV (Wheeler et al. 2009), though the exact mechanisms behind the population declines remain unknown and not quantified.

We examined change in survival probability as one of the potential mechanisms behind the decline of the House Finch

population documented in northern California (Wheeler et al. 2009). Few estimates of survival probability have been published for the House Finch, and no estimates of survival in relation to WNV have been reported. Existing studies include seasonal estimates of survival in relation to *Mycoplasma gallisepticum* (Faustino et al. 2004) and regional annual estimates (DeSante and Kaschube 2007). Our goals were to determine the arrival date of WNV at our study area and to estimate the effect of WNV on the annual survival of adult House Finches.

METHODS

STUDY AREA AND FIELD METHODS

We recorded data at Stone Lakes National Wildlife Refuge starting in 2001. The refuge borders housing developments from the city of Elk Grove, California, to the east and is surrounded by agriculture (small grains, vineyards, and orchards) to the north, south, and west. Although House Finches nest in low densities throughout the refuge, we selected one site as a focal area (38° 22′ N, 121° 29′ W) because of its high density of breeding birds.

We determined the date of arrival of WNV in the study area by looking for the presence of antibodies in juvenile birds as evidence of new infections during the corresponding calendar year. We also looked for the presence of viral RNA in host-seeking mosquitoes trapped within the refuge for the first records of viral transmission.

We sampled host-seeking female mosquitoes with three dry-ice-baited traps (Sudia and Chamberlain 1962) and gravid female mosquitoes with one "gravid" trap (Cummings 1992) placed at permanent locations around the perimeter of the study site. Attracted by water in the "gravid" trap, female mosquitoes attempt to land on water to lay eggs and are drawn into a collection chamber. Collected mosquitoes were anesthetized with triethylamine (Kramer et al. 1990), counted, and separated by species. Groups of ≤50 female *Culex* mosquitoes were frozen at −80 °C and later tested for RNA of WNV, St. Louis encephalitis, and western equine encephalomyelitis with a multiplex real-time reverse-transcriptase polymerase chain reaction (RT-PCR). We carried out RT-PCR with ABI TaqMan One-Step RT-PCR Master Mix with primers and probe designed at the Center for Vectorborne Diseases, University of California, Davis (CVEC-UCD; A. Brault, unpubl. data).

Prior to 2006 we sampled mosquitoes at two other locations within the refuge, one 6.29 km to the north, the other 3.62 km to the south. Mosquito sampling at these sites differed in that we deployed four traps baited with dry ice every other week and used no gravid trap. In 2007 and 2008, we increased the frequency of sampling from every other week to once per week at the site of the House Finch study and added the use of a gravid trap.

We determined antibody presence in House Finches through testing serum samples. Blood samples (0.1 mL) were collected via jugular puncture with a 28-gauge needle and syringe and immediately expressed into 0.9 mL of 0.5% bovine albumen/phosphate-buffered saline. No individual was bled more than once in 10 days. Samples were kept cold on Blue Ice until centrifuged that day at 8000 revolutions min^{-1} for 5 min. Sera were stored at −80°C until screened for the flavivirus antibodies with an enzyme immunoassay (Chiles and Reisen 1998). We confirmed samples positive by this assay and identified the virus with a plaque-reduction neutralization test.

We estimated survival from mark–recapture data. From March to July we captured birds in mist nets (30-mm mesh, 2.6×12 m) and a funnel-style ground trap baited with grain. Captured House Finches were aged and sexed according to Pyle (1997), banded with U.S. Fish and Wildlife Service aluminum bands, bled, and released at the site of capture.

Capture effort was not continuous across the entire study period. Prior to 2007, capture effort was limited to a maximum of 2 net-days (10 nets for 4 hr) per month. Starting in 2005, one ground trap was operated weekly on a separate field day. In 2007 and 2008, we increased capture effort with the intent of increasing the probability of an encounter in the face of decreasing numbers of birds, adding one further day of netting and one further day of ground-trapping per week during both of these seasons.

STATISTICAL ANALYSES

We calculated return rate as the proportion of individuals from a given year's cohort that were recaptured in at least one year following the initial capture and release. We differentiated between annual return rate (the proportion of individuals encountered in the second year of being marked) and overall return rate (the proportion of individuals encountered in a year following the capture year). If an individual was encountered in more than one subsequent year, it was still counted as only one individual. Same-season recaptures were excluded from analysis.

We used the Cormack–Jolly–Seber model in program MARK (White and Burnham 1999) to estimate probabilities of annual survival (φ) and encounter (ρ) (Lebreton et al. 1992). On the basis of our knowledge of the site and the hypothesis that survival probability changed after the arrival of WNV, we created a candidate set of nine models a priori. Unseen factors can influence φ from year to year, so models allowed φ to vary with time. To address the question of the effects of WNV on φ, we looked first at antibody presence directly. The data were too sparse, however, with only 10.7 % of the captured birds being antibody positive. The resulting models had large standard errors and β confidence limits, indicating high uncertainty, which led us to ultimately discard them. Instead, we assessed the effect on φ of time period, defined as years before (pre-WNV, 2001–2004) and after (post-WNV; 2005–2008) the arrival of WNV. We did not address the influence of sex on φ, as there is no evidence in the literature of mortality differing by sex. For encounter probability, we examined capture effort

by year and time-varying ρ. We defined capture effort as the sum of all hours of ground trapping and mist-netting. We did not include sex or capture method in the models for ρ as they were not central to our hypothesis. We included models with constant φ and ρ as null hypotheses. Because of sparseness of data we did not include juveniles in the survival analysis.

We built the models by using the design matrix and logit-link function. The input file was structured into one group with additional constraints added in the design matrix. We restricted data to the breeding season, which, from observations of breeding characteristics, we defined as March through July. Ten individuals were originally captured outside the breeding season but subsequently encountered in at least one breeding season, so we included them in the analysis to increase the number of recaptures. We tested assumptions of global model fit by the bootstrap goodness-of-fit method (1000 simulations) then calculated the variance-inflation factor ($\hat{c}$) as the global model deviance divided by the mean bootstrap deviance. We corrected for overdispersion by using $\hat{c}$ to adjust model selection and parameter estimates. We then used quasi-Akaike's information criterion ($QAIC_c$) to select the best model and derive support for the importance of each variable (Burnham and Anderson 2002). Because of uncertainty about the identity of the best model, we used model averaging to derive the reported estimates of φ and ρ.

RESULTS

We detected the first WNV-positive mosquitoes on the refuge on 22 September 2004, when a pool of *Culex erythrothorax* tested positive. The two most abundant bird-feeding species tested for virus at the House Finch study site were *C. tarsalis* (116 pools; 3776 mosquitoes) and *C. pipiens* (94 pools; 2401 mosquitoes), both of which tested positive. WNV-positive mosquitoes were detected at this site in 2006 (one pool) and 2007 (three pools). No positive mosquitoes were detected at the House Finch site in 2008, though positive pools were collected at the two alternate mosquito sites.

WNV antibody was first detected at the site in 2005 in blood samples from both adult and juvenile House Finches. Antibody-positive adults were detected each year from 2005 to 2008, whereas antibody-positive juveniles were detected only in 2005 and 2006. Between 2001 and 2008, 48 of 448 blood-sampled adults and four of 127 blood-sampled juveniles were antibody positive. Three individuals, all male, were antibody positive in two consecutive years of capture. As in 2008 the antibody-positive birds were all adults; we could not determine the date of infection. With no positive mosquitoes or juvenile birds detected, there appeared to be no viral activity at the site in 2008.

From 2001 to 2008 we captured 468 adult House Finches, the blood of 20 of which we did not sample. Of the 376 individuals caught between 2001 and 2007, we recaptured 49, for an overall return rate of 13%. Twenty-four (6%) of these were
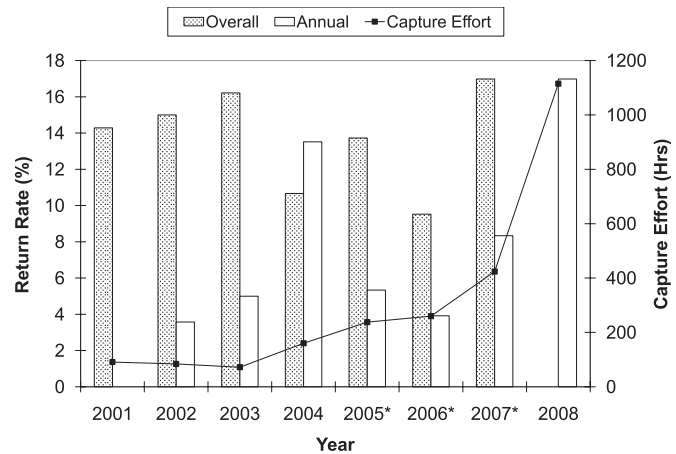

FIGURE 1.   Return rates (overall, shaded bars; annual, white bars) and capture effort (solid line, in combined net- and trap-hours) for adult House Finches on the Stone Lakes National Wildlife Refuge, Sacramento County, California. The rates are specific to a cohort of birds captured in a given year with overall return rate denoting how many individuals returned over the remainder of the study and annual rate denoting how many individuals returned from the previous year. Overall return rates are reported in the cohort year to which they correspond. Years with WNV activity are denoted with an asterisk (*).

recaptured only in the year immediately following the year of capture. Seven individuals (2%) were captured the year after they were marked and in at least one additional year. Eighteen individuals (5%) were recaptured in at least two different years but not in the year after they were captured. Return rates varied by annual cohort, and annual (returns the following year) and overall (returns sometime during the study period) return rates also varied (Fig. 1).

We used the global model [φ(t)ρ(t)] to run the bootstrap goodness-of-fit test in MARK then used the resulting estimate of overdispersion ($\hat{c}$ = 1.44) to correct for lack of model fit in subsequent analyses. The top model included the time-period effect (Table 1) and indicated a decrease in survival probability after the arrival of WNV. In the top model, survival estimates were 0.64 ± 0.10 SE (pre-WNV) and 0.43 ± 0.07 SE (post-WNV). In the second model survival probability was constant at 0.51 ± 0.06. The model-averaged estimates (pre-WNV, 0.59; post-WNV, 0.47) also demonstrated a decrease, though not as large as that of the top model (Table 2). Encounter probability increased with increasing capture effort (Table 2).

DISCUSSION

WNV arrived at the Stone Lakes National Wildlife Refuge in late 2004. Because the virus arrived after the breeding season, we treated 2004 as a pre-WNV year. Provided that antibodies of maternal origin had waned by the time of capture (Reisen et al. 2005b, King et al. 2010), the presence of antibodies in juvenile birds in 2005 and 2006 confirmed transmission to House Finches during those years. Transmission could not be

TABLE 1.   Models used in program MARK to assess the effect of period pre- or post-arrival of WNV (p/p) and time (t) or lack of effect (.) on apparent survival (φ) and to assess the effect of capture effort (CE) and time or lack of effect on recapture (ρ) of adult House Finches captured on the Stone Lakes NWR, Sacramento County, California. K is the number of parameters in the model, $QAIC_c$ is quasi-Akaike's information criterion adjusted for overdispersion, Δ_i is the $QAIC_c$ value relative to the top model (i.e., the lowest $QAIC_c$), and w_i is the model weight; $n = 468$ individuals.

Model	QDeviance	K	Δ_i	w_i
$\varphi(p/p)\rho(CE)$[a]	44.94	4	0	0.373
$\varphi(.)\rho(CE)$	47.10	3	0.12	0.352
$\varphi(.)\rho(.)$	51.16	2	2.15	0.128
$\varphi(t)\rho(CE)$	38.17	9	3.57	0.063
$\varphi(p/p)\rho(.)$	51.15	3	4.17	0.046
$\varphi(p/p)\rho(t)$	40.25	9	5.65	0.022
$\varphi(.)\rho(t)$	43.76	8	7.08	0.011
$\varphi(t)\rho(t)$	35.55	13	9.41	0.003
$\varphi(t)\rho(.)$	46.89	8	10.20	0.002

[a] Top model; $QAIC_c = 320.42$.

confirmed in 2007 or 2008, but the presence of virus-positive host-seeking mosquitoes in 2007 indicated viral activity and possible transmission.

Our estimates of survival varied with time, showing a distinct decrease following the arrival of WNV. The pre-WNV estimate was very similar to another published estimate (DeSante and Kaschube 2007) that was calculated from data collected from 1992 to 2003 at 40 stations in the southwestern United States. Survival estimates are influenced by multiple factors including emigration (Cilimburg et al. 2002), the proportion of transients included in the sample (Pradel et al. 1997), and mortality. Permanent emigration mimics mortality when individual fates are unknown. Although there is no way to determine the rate of emigration from our data, it is likely that

TABLE 2.   Model-averaged estimates of apparent survival (φ) and recapture probabilities (ρ) of adult House Finches captured on the Stone Lakes National Wildlife Refuge, Sacramento County, California before and after arrival of West Nile virus. Estimates for ρ are based primarily on models containing capture effort. Unconditional standard errors (SE) are reported.

$\varphi \pm SE$	$\rho \pm SE$
Pre: 0.59 ± 0.15	2001–2002: 0.13 ± 0.05
Post: 0.47 ± 0.11	2002–2003: 0.13 ± 0.05
	2003–2004: 0.14 ± 0.05
	2004–2005: 0.16 ± 0.04
	2005–2006: 0.16 ± 0.04
	2006–2007: 0.21 ± 0.11
	2007–2008: 0.37 ± 0.23

some of our birds did emigrate, as there is suitable breeding habitat within flight range of the study site. Although emigration undoubtedly decreased the overall survival estimate, we assumed it was relatively constant during our study because we did not observe any obvious change in the local habitat or food availability. Transiency of birds also mimics death or permanent emigration, with the effect that as the proportion of transients in the sample increases, the probability of apparent survival decreases. Our capture effort was not constant across the study period, with post-WNV sampling more frequent and later into the breeding season (July), when transients became more abundant, than pre-WNV sampling. We did not control completely for transients in our analysis. Therefore, some of the decrease in survival probability post-WNV may have been due to an increase in the proportion of transients associated with the increase in capture effort late in the breeding season.

Mortality rate is influenced by a variety of factors, including disease. The evidence for WNV at the study site in 2005, 2006, and 2007 implied some mortality due to WNV. Laboratory infection experiments with WNV have demonstrated mortality rates of >60% in House Finches (Komar et al. 2003a, Reisen et al. 2005a). The presence of antibodies in adults and juveniles starting in 2005 demonstrated that some birds survived natural infection and that mortality due to WNV at our study site was <100%. Furthermore, laboratory experiments have demonstrated that birds surviving infection with WNV or the closely related St. Louis encephalitis virus have immunity against future infection (Fang and Reisen 2006), suggesting that the mortality rate within a population might decrease over time as acquired immunity increases.

In addition to WNV, we also anecdotally noted the presence of lesions similar to those produced by pox on some of our birds. Avian pox was first documented in House Finches in California in the 1970s (Power and Human 1976). Although it was never positively identified at our site by histopathology or other diagnostic tests, the frequency of poxlike lesions on the heads and feet of captured birds seemed to increase concurrently with the arrival of WNV. We captured some individuals frequently enough to document a possible recovery and survival from apparent pox infection, but we captured many only once, leaving their fate unknown. It is possible that pox could have also contributed to the decrease in survival probability, though we have no data to substantiate this hypothesis.

The slight reduction in return rate for the 2004, 2005, and 2006 cohorts despite increased capture effort supports the hypothesis that there may have been mortality due to WNV. The increased return rate for the 2007 cohort might be due to the strongly increased capture effort, to the lack of viral activity at the site, or to a combination of both. House Finches leave their nest sites and form foraging flocks nearby. It is possible that some birds became infected during the nonbreeding season at an alternate location, making survival difficult to correlate with viral activity at the study site.

In summary, WNV has been linked with population declines in multiple species of birds, though the exact mechanisms behind the declines remain unknown. Our findings provide field evidence of reduced survival probability associated with the arrival of WNV. Further research is needed to determine the role that other mechanisms, such as fecundity and dispersal probability, play in these declines. Additionally, more research is needed to determine how stable survivorship will be in the future and how these changes will affect the House Finch's population dynamics.

ACKNOWLEDGMENTS

LITERATURE CITED

ALLAN, B. F., R. B. LANGERHANS, W. A. RYBERG, W. J. LANDESMAN, N. W. GRIFFIN, R. S. KATZ, B. J. OBERLE, M. R. SCHUTZENHOFER, K. N. SMYTH, A. DE ST. MAURICE, L. CLARK, K. R. CROOKS, D. E. HERNANDEZ, R. G. MCLEAN, R. S. OSTFELD, AND J. M. CHASE. 2009. Ecological correlates of risk and incidence of West Nile virus in the United States. Oecologia 158:699–708.

BURNHAM, K. P., AND D. R. ANDERSON. 2002. Model selection and multimodel inference: a practical information-theoretic approach. 2nd ed. Springer-Verlag, New York.

CHILES, R. E., AND W. K. REISEN. 1998. A new enzyme immunoassay to detect antibodies to arboviruses in the blood of wild birds. Journal of Vector Ecology 23:123–135.

CILIMBURG, A. B., M. S. LINDBERG, J. J. TEWKSBURY, AND S. J. HEJL. 2002. Effects of dispersal on survival probability of adult Yellow Warblers (*Dendroica petechia*). Auk 119:778–789.

CROSBIE, S. P., W. D. KOENIG, W. K. REISEN, V. L. KRAMER, L. MARCUS, R. CARNEY, E. PANDOLFINO, G. M. BOLEN, L. R. CROSBIE, D. A. BELL, AND H. B. ERNEST. 2008. Early impact of West Nile virus on the Yellow-billed Magpie (*Pica nuttalli*). Auk 125:542–550.

CUMMINGS, R. F. 1992. Design and use of a modified Reiter gravid mosquito trap for mosquito-borne encephalitis surveillance in Los Angeles County, California. Proceedings of the Mosquito and Vector Control Association of California 60:170–176.

DAWSON, J. R., W. B. STONE, G. D. EBEL, D. S. YOUNG, D. S. GALINSKI, J. P. PENSABENE, M. A. FRANKE, M. EIDSON, AND L. D. KRAMER. 2007. Crow deaths caused by West Nile virus during winter. Emerging Infectious Diseases 13:1912–1914.

DESANTE, D. F., AND D. R. KASCHUBE. 2007. The Monitoring Avian Productivity and Survivorship (MAPS) program 2002 and 2003 report. Bird Populations 8:46–115.

FANG, Y., AND W. K. REISEN. 2006. Previous infection with West Nile or St. Louis encephalitis viruses provides cross protection during reinfection in House Finches. American Journal of Tropical Medicine and Hygiene 75:480–485.

FAUSTINO, C. R., C. S. JENNELLE, V. CONNOLLY, A. K. DAVIS, E. C. SWARTHOUT, A. A. DHONDT, AND E. G. COOCH. 2004. *Mycoplasma gallisepticum* infection dynamics in a House Finch population: seasonal variation in survival, encounter and transmission rate. Journal of Animal Ecology 73:651–669.

HUTCHESON, H. J., C. H. GORHAM, C. MACHAIN-WILLIAMS, M. A. LOROÑO-PINO, A. M. JAMES, N. L. MARLENEE, B. WINN, B. J. BEATY, AND C. D. BLAIR. 2005. Experimental transmission of West Nile virus (Flaviviridae: *Flavivirus*) by *Carios capensis* ticks from North America. Vector-Borne and Zoonotic Diseases 5:293–295.

KILPATRICK, A. M., S. L. LADEAU, AND P. P. MARRA. 2007. Ecology of West Nile virus transmission and its impact on birds in the western hemisphere. Auk 124:1121–1136.

KING, M. O., J. P. OWEN, AND H. G. SCHWAB. 2010. Are maternal antibodies really that important? Patterns in the immunologic development of altricial passerine House Sparrows (*Passer domesticus*). PLoS One 5:e9639.

KIPP, A. M., J. A. LEHMAN, R. A. BOWEN, P. E. FOX, M. R. STEPHENS, K. KLENK, N. KOMAR, AND M. L. BUNNING. 2006. West Nile virus quantification in feces of experimentally infected American and Fish Crows. American Journal of Tropical Medicine and Hygiene 75:688–690.

KOENIG, W. D., L. MARCUS, T. W. SCOTT, AND J. L. DICKINSON. 2007. West Nile virus and California breeding bird declines. EcoHealth 4:18–24.

KOMAR, N. S. LANGEVIN, S. HINTEN, N. NEMETH, E. EDWARDS, D. HETTLER, B. DAVIS, R. BOWEN, AND M. BUNNING. 2003a. Experimental infection of North American birds with the New York 1999 strain of West Nile virus. Emerging Infectious Diseases 9:311–322.

KOMAR, N., K. MARAMOROSCH, F. A. MURPHY, A. J. SHATKIN, T. J. CHAMBERS, AND T. P. MONATH. 2003b. West Nile virus: epidemiology and ecology in North America. Advances in Virus Research 61:185–234.

KRAMER, L. D., S. B. PRESSER, E. J. HOUK, AND J. L. HARDY. 1990. Effect of the anesthetizing agent triethylamine on western equine encephalomyelitis and St. Louis encephalitis viral titers in mosquitoes (Diptera: Culicidae). Journal of Medical Entomology 27:1008–1010.

KRAMER, L. D., L. M. STYER, AND G. D. EBEL. 2008. A global perspective on the epidemiology of West Nile virus. Annual Review of Entomology 53:61–81.

LADEAU, S. L., A. M. KILPATRICK, AND P. P. MARRA. 2007. West Nile virus emergence and large-scale declines of North American bird populations. Nature 447:710–713.

LEBRETON, J. D., K. P. BURNHAM, J. CLOBERT, AND D. R. ANDERSON. 1992. Modeling survival and testing biological hypotheses using

marked animals: a unified approach with case studies. Ecological Monographs 62:67–118.

PETERSON, A. T., N. KOMAR, O. KOMAR, A. NAVARRO-SIGÜENZA, M. B. ROBBINS, AND E. MARTINEZ-MEYER. 2004. West Nile virus in the New World: potential impacts on bird species. Bird Conservation International 14:215–232.

POWER, D. M., AND G. HUMAN. 1976. A local occurrence of avian pox in the House Finch. Condor 78:262–263.

PRADEL, R., J. E. HINES, J. D. LEBRETON, AND J. D. NICHOLS. 1997. Capture–recapture survival models taking account of transients. Biometrics 53:60–72.

PYLE, P. 1997. Identification guide to North American birds: part I. Slate Creek Press, Bolinas, CA.

REISEN, W. K., Y. FANG, AND V. M. MARTINEZ. 2005a. Avian host and mosquito (Diptera: Culicidae) vector competence determine the efficiency of West Nile and St. Louis encephalitis virus transmission. Journal of Medical Entomology 42:367–375.

REISEN, W. K., S. S. WHEELER, S. YAMAMOTO, Y. FANG, AND S. GARCIA. 2005b. Nesting ardeid colonies are not a focus of elevated West Nile virus activity in southern California. Vector Borne Zoonotic Diseases 5:258–266.

SUDIA, W. D., AND R. W. CHAMBERLAIN. 1962. Battery-operated light trap, an improved model. Mosquito News 22:126–129.

TEMPELIS, C. H., W. C. REEVES, AND R. L. NELSON. 1976. Species identification of blood meals from *Culex tarsalis* that had fed on passeriform birds. American Journal of Tropical Medicine and Hygiene 25:744–746.

WARD, M. P., A. RAIM, S. YAREMYCH-HAMER, R. LAMPMAN, AND R. J. NOVAK. 2006. Does the roosting behavior of birds affect transmission dynamics of West Nile virus? American Journal of Tropical Medicine and Hygiene 75:350–355.

WHEELER, S. S., C. M. BARKER, Y. FANG, M. V. ARMIJOS, B. D. CARROLL, S. HUSTED, W. O. JOHNSON, AND W. K. REISEN. 2009. Differential impact of West Nile virus on California birds. Condor 111:1–20.

WHITE, G. C., AND K. P. BURNHAM. 1999. Program MARK: survival estimation from populations of marked animals. Bird Study 46:120–138.