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Research Paper

Prediction of Equine Risk of West Nile Virus Infection Based on Dead Bird Surveillance

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ABSTRACT

Since the introduction of West Nile Virus (WNV) to the United States in 1999, the efficacy of dead bird surveillance for the prediction of human and veterinary WNV infection has been an issue of debate. We utilized South Carolina's Department of Health and Environmental Control surveillance data from 2003 to determine whether dead bird surveillance accurately predicts equine WNV infection on a county level. We adjusted for human population density as a potential confounder of an association between WNV-positive dead bird counts and mammalian WNV risk. We found a strong positive association between avian risk of WNV death and subsequent equine mortality due to WNV in South Carolina even after adjusting for human population density. Sensitivity of dead bird surveillance as a predictor of future equine WNV risk was far superior to mosquito surveillance (95% vs. 9.5%, respectively). A Poisson regression model of the equine WNV rate as a function of WNV-positive dead bird rate, adjusting for population density and taking into account effect modification by population density shows a good fit with the data. Unlike most previous studies, we control for potential confounding of the dead, WNV-positive bird-equine WNV infection association by human population density. Yet, the positive association between dead bird surveillance and equine WNV risk remains strong and statistically significant, indicating that dead bird surveillance remains a valuable tool of WNV surveillance. Key Words: West Nile virus—prediction—risk—surveillance. Vector-Borne Zoonotic Dis. 6, 1–6.

INTRODUCTION

SINCE THE INTRODUCTION of West Nile Virus (WNV) into the United States in 1999, the efficacy of various surveillance methods for prediction and prevention of human and veterinary illness from WNV infection has been an issue of debate (Campbell et al. 2002, Garmendia et al. 2001, Marfin et al. 2001, Petersen 2001). The high susceptibility of some North American bird species for fatal WNV infection and the fact that bird mortality typically precedes human or equine WNV infection has made dead bird surveillance a focus of particular interest. Several papers evaluating the efficacy of dead bird surveillance (Eidson 2001, Eidson et al. 2005, Guptill et al. 2003, Mostashari et al. 2003, Blackmore et al. 2003, Watson et al. 2004) suggested that dead bird counts are in fact predictive of subsequent local human WNV illness. However, a recent report by Brownstein et al. (2004) concluded that mosquito surveillance is superior to dead bird surveillance as a basis for human WNV risk prediction and some have called for abandoning dead bird surveillance as a primary tool of WNV surveillance (NCDENR 2004).

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Most of the studies mentioned above, including Brownstein et al. (2004) did not directly account for human population density as a potential confounder and/or effect modifier of the association between dead bird counts and human WNV risk. Areas with greater human population densities are more likely to have dead birds reported (due to an increased probability of people finding them) and are more likely to have an increased number of human cases (due to a larger number of people at risk of infection) (Eidson 2001), which might confound the association between dead bird counts and WNV risk. Although Blackmore et al. (2003) correlated overall dead bird rates per human population with WNV activity in Florida, 2001, bird mortality due to other causes may lead to an underestimation of the true association.

Horses and other equids are highly susceptible and vulnerable to WNV infection and carry the largest mammalian burden of morbidity (South Carolina DHEC 2003). Human and equine risk follow similar temporal and spatial patterns (Blackmore et al. 2003, CDC 2003), likely due to shared bridge vectors (A. Spielman, personal communication). In this paper, we examine the relationship between WNV-positive dead bird counts and equine WNV risk based on data from South Carolina, using a simple method of adjustment for human population density.

METHODS

We obtained South Carolina dead-bird and veterinary surveillance data for the year 2003 from the South Carolina Department of Health and Environmental Control. Although data were also available for 2002, we decided to only use the 2003 data, because criteria for testing dead birds for WNV infection changed from one year to the next. Information on population and county area was taken from public web pages (U.S. Census Bureau 2000). Dead birds of four species (American and Fish Crow, Blue Jay, Northern Cardinal) were accepted from the public for testing if there had not been a WNV-positive bird less than 15 days within a 5-mile radius from the site where the bird had been found. Mosquitoes were mostly collected with CO_2-baited CDC light traps.

The number of farms, which was obtained from the United States Department of Agriculture's Census of Agriculture statistics, 1997, was used as a proxy for horse density, which is not directly recorded in South Carolina (NASS 1997). The complete data are displayed in Table 1.

Diagnostic testing of birds, equids, and mosquitoes was performed according to the CDC guidelines for laboratory diagnosis of WNV (CDC 2003). All bird and mosquito samples were tested at South Carolina Department of Health and Environmental Control, Columbia, South Carolina, while equine samples were tested at Clemson University, Clemson, South Carolina.

Statistical analysis

The main outcome variable was the number of positive WNV equine cases, and the predictor variable of main interest was the number of WNV-positive dead birds. To quantify the association between county-specific numbers of WNV-positive dead birds per human population (positive bird rate [PBR]) and of WNV-positive equids per farm (positive equine rate [PER], a surrogate for WNV-positive equine density due to lack of number of equids reported in South Carolina), a Poisson regression analysis was used. The number of WNV-positive equids per county was modeled as a function of PBR, with the log of the number of farms per county as offset variable. The rate modeled therefore is the county-specific PER. To capture effect modification of the association of interest by the level of urbanization, we included an interaction term for PBR*human population density in the model. We evaluated model fit by sorting the 45 counties by population density and partitioning them into five equally sized groups of similar population density. The expected number of equids per group was then compared with the observed number using a Pearson chi-square statistic (4 df). A significant result indicates poor model fit. For all statistical analyses, we used SAS (SAS Institute 1999).

TABLE 1. COUNTY-SPECIFIC WEST NILE VIRUS (WNV) SURVEILLANCE DATA, SOUTH CAROLINA, 2003

County	*No. of WNV-positive birds*	*No. of WNV-positive equids*	*No. of farms*	*Area (in square miles)*	*Population*
Abbeville	0	0	471	508	26,164
Aiken	14	6	729	1073	142,557
Allendale	2	0	114	408	11,226
Anderson	9	1	1271	718	165,719
Bamberg	1	2	254	393	16,674
Barnwell	9	1	325	548	23,472
Beaufort	9	0	99	587	120,952
Berkeley	15	2	292	1098	142,704
Calhoun	0	2	261	380	15,171
Charleston	19	2	266	919	309,997
Cherokee	2	0	412	393	52,542
Chester	1	0	340	581	34,077
Chesterfield	4	1	537	799	42,805
Clarendon	0	0	304	607	32,486
Colleton	15	7	416	1056	38,240
Darlington	5	0	346	561	67,393
Dillon	4	0	199	405	30,727
Dorchester	21	3	314	575	96,440
Edgefield	0	0	271	502	24,593
Fairfield	1	0	172	687	23,481
Florence	1	1	615	800	125,735
Georgetown	7	0	206	814	55,816
Greenville	22	5	761	790	379,633
Greenwood	4	0	377	456	66,278
Hampton	6	1	207	560	21,384
Horry	52	10	896	1134	196,580
Jasper	2	1	123	656	20,668
Kershaw	5	0	324	726	52,654
Lancaster	2	0	500	549	61,377
Laurens	0	0	686	715	69,580
Lee	1	1	222	410	20,105
Lexington	4	0	799	699	215,998
Marion	6	3	200	489	35,457
Marlboro	2	0	180	480	28,831
McCormick	0	0	92	360	9,960
Newberry	0	0	499	631	36,080
Oconee	7	1	611	625	66,231
Orangeburg	2	3	965	1106	91,590
Pickens	4	0	532	497	110,757
Richland	6	0	350	756	320,642
Saluda	0	0	556	452	19,185
Spartanburg	6	1	1067	811	253,821
Sumter	3	0	396	665	104,669
Union	2	0	255	514	29,870
Williamsburg	1	0	602	934	37,263
York	5	0	726	682	164,607

RESULTS

The sensitivity of WNV-positive bird reports for equine case prediction was high—20 of the 21 counties (95%) with equine WNV cases also had WNV-positive dead birds that occurred at least 1 week prior to any equine cases reported. Specificity, on the other hand, was low—only seven of the 25 counties (28%) without equine cases had zero positive birds from those tested. This is not surprising, however, as the risk for equine infection becomes only substantial when enzootic transmission is intense. In comparison, based on only three WNV-positive mosquito pools that occurred in three different counties among 89,349 mosquitoes, sensitivity

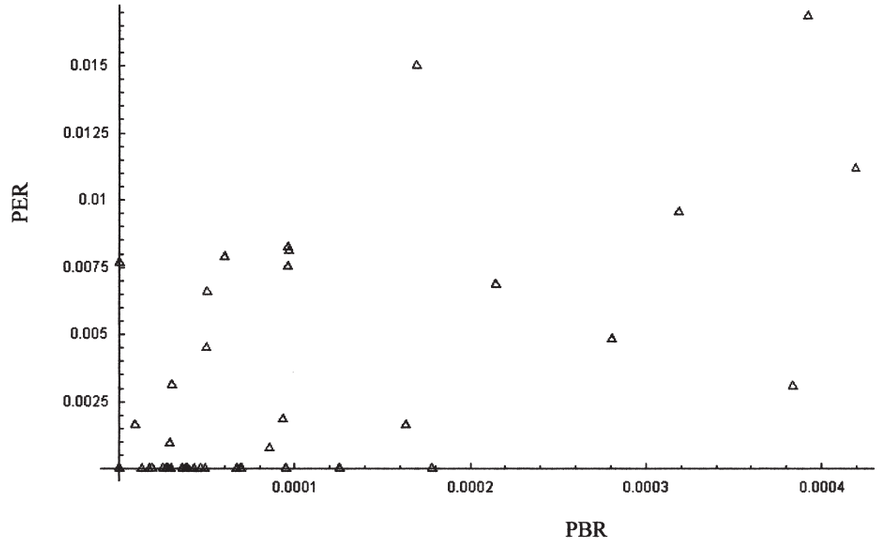

FIG. 1. The crude association between positive bird rate (PBR) and positive equine rate (PER). Each data point corresponds to one county.

of mosquito surveillance for the detection of equine cases was only about 9.5%. On the county level, the PER increased with the PBR (Fig. 1).

When adjusting for potential confounding by population density, the equine risk of acquiring WNV significantly increased with the positive bird rate (Table 2). This increase was roughly linear on the log scale, as indicated by the fact that a quadratic term (PBR*PBR) was not significant (not shown). Although the main effect of population density was not significant, the interaction term PBR*human population density was. The interpretation of this result is facilitated by a graphical representation (Fig. 2). The strength of the association between PBR and PER depends on the population density. The more densely a county is populated, the stronger is the association between the dead bird rate and the equine WNV risk; that is, one dead bird per population translates into a higher equine risk with increasing population density.

The model fit the data reasonably well, especially in the counties with higher population densities. The numbers of equine cases of WNV infection as expected from the model in the five population density groups were 7.901, 7.505, 6.469, 20.712, and 11.311, while the observed numbers were 13, 3, 7, 22, and 9, respectively (Pearson chi-square statistic 12.751, $p = 0.987$).

DISCUSSION

The results from our study suggest that dead bird surveillance remains a valuable tool for the prediction of veterinary WNV infection. In this study, we controlled for the potential confounding effect of human population density on the association between WNV-positive dead

TABLE 2. COEFFICIENT ESTIMATES OF THE FITTED POISSON REGRESSION MODEL OF EQUINE POSITIVE RATE

	Parameter estimate	*SE*	*95% CI*	p-*value*
Intercept	−6.958	0.369	−7.681, −6.235	<0.0001
PBR	5258.018	1602.977	2116.241, 8399.794	0.001
Human population density	0.001	0.002	−0.003, 0.004	0.7608
PBR * human population density	26,180	12.389	1.900, 50.461	0.0346

SE, standard error; CI, confidence interval; PBR, positive bird rate.

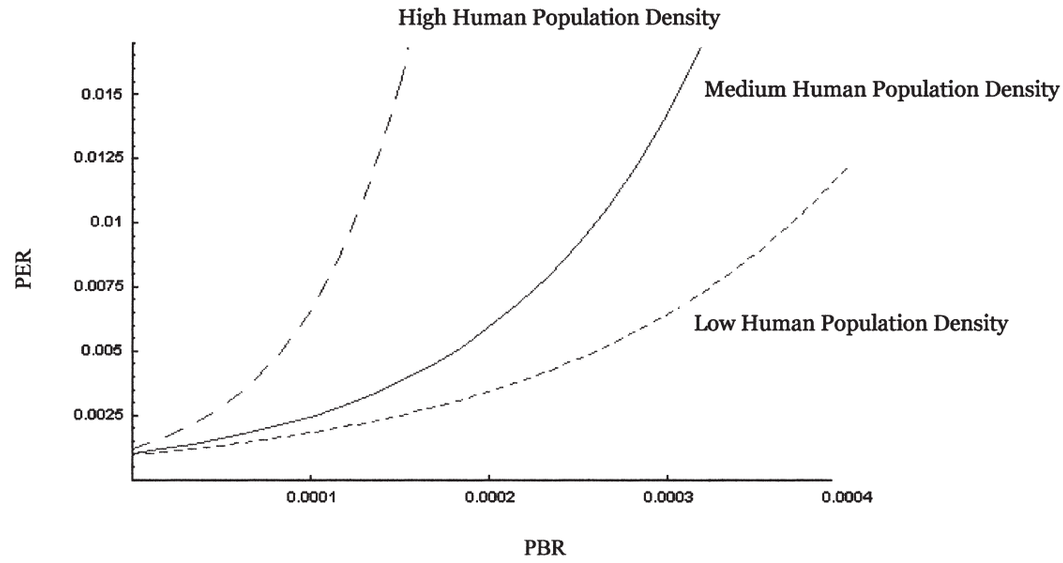

FIG. 2. The modeled association between positive bird rate (PBR) and positive equine rate (PER) by population density. The bottom line (stippled) corresponds to a low population density (≤40 per sq. mi.), the middle line (solid) corresponds to a medium population density (41–134 per sq. mi.), and the upper line (dashed) represents a high population density (≥135 per sq. mi). According to this model, a given PBR corresponds to a higher PER in a more highly versus more sparsely populated county. Similarly, an increase in PBR translates into a more pronounced increase in PER when population density is higher.

bird counts and equine WNV cases, by normalizing WNV-positive dead bird numbers by the county human population. Despite this adjustment, a strong association between avian WNV-associated deaths and equine WNV risk remained.

These results are likely to have public health implications as equids and humans presumably share at least some of their mosquito bridge vectors due to the fact that host specificity of mosquitoes is thought to operate at the taxonomic class level. Although the Poisson regression model presented here fits the data well and therefore strongly supports the notion that WNV-associated avian mortality is a good predictor of ensuing veterinary and therefore human WNV-infection, we need to consider potential sources of bias. First, our approach is an ecological one, with the unit of analysis being counties. Transmission of WNV, however, is likely to happen on a much smaller geographic scale. We can therefore not assert that avian deaths in one county are meaningfully associated with equine cases in the same county. However, the resulting bias would obscure any real association and is therefore of little concern here. Of more potential relevance is a detection bias, whereby people residing in a particular area might be more likely to submit dead birds for WNV testing if they are aware of any WNV-associated illness in horses in that area. Although we cannot ultimately rule out that such mechanism might have affected our results, we point out that in 20 out of 21 counties, WNV-associated bird mortality was noted at least 1 week prior to any equine cases being reported.

Another potential source of error is the dead bird testing strategy used in South Carolina. Birds were only accepted for testing if no WNV-positive bird had been found in a 10-mile diameter within the previous 15 days. However, error resulting from the testing strategy would dilute an existing association rather than inflate it and is of little concern here, because we found a strong association in spite of this potential bias.

The observed association could also be biased if equine immunization levels were to depend on WNV risk. However, the association would only be inflated if immunization levels were specifically higher in areas with lower risk, which is a highly unlikely scenario. In the more likely event that local WNV activity were to go hand in hand with higher immunization

levels in horses, the observed association would be smaller than the true association. As we observed a strong association, this argument is of little concern.

We conclude that the number of WNV-positive birds per population is strongly predictive for the rate of equine WNV morbidity in South Carolina on a county level and far superior to mosquito surveillance for zoonotic WNV prediction, at least in South Carolina, where, in 2003, mosquito surveillance primarily relied on Centers for Disease Control and Prevention (CDC) light traps. The application of this method to human WNV morbidity data as well as a comparison of mosquito surveillance based on gravid traps will further contribute to the discussion of the value of dead-bird surveillance for WNV prediction.

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