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Title Page

Estimates of Worst Case Baseline West Nile Virus Disease Effects in a Suburban New York County

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ABSTRACT: Serosurveys conducted where West Nile Virus (WNV) caused health impacts were used to construct a model of potential worst case health impacts in a suburban setting. This model addressed two common public perceptions regarding mosquito control activities and WNV disease: it is not a disease of major consequence, and exposed populations quickly become immune. Comparisons to blood bank infection and serious disease incidence data were similar to some of the serosurvey model results. Accounting for theoretical increasing immunity, even over a 20-year horizon, did not substantially reduce the potential impacts. The model results were approximately an order of magnitude greater than those actually experienced in Suffolk County, New York; differences in mosquito populations and/or the degree of mosquito control between Suffolk County and serosurvey sites seem to be the cause of the differences.

Text

INTRODUCTION

Suffolk County, New York, occupies the eastern two thirds of Long Island, has a population of 1.4 million, and has conducted organized mosquito control for more than 75 years (Glasgow 1938). West Nile Virus (WNV), an exotic mosquito-borne disease that was first detected in North America in New York City in 1999 (Petersen and Roehrig 2000), also affected Suffolk County then, causing horse mortalities (Ostlund et al. 2001) but no human cases (Nasci et al. 2001). Under New York State law, government actions (including the adoption of plans that may lead to action) that have a potential for environmental impacts require formal environmental review (Lopez and Miller 2002). Two nearby municipalities, New York City and Westchester County, conducted comprehensive environmental reviews in 2001 (Lopez and Miller 2002, Shapiro and Micucci 2003) on plans that were developed to address the new situation. Suffolk County followed suit and began a complete re-evaluation of all aspects of its mosquito control operations.

Public comments received at the start of the re-evaluation project included claims that there was so little risk associated with WNV disease in Suffolk County that perhaps no mosquito control was needed (e.g., see the Draft Task 1 Report from the Long-Term project, <u>http://www.suffolkcountyny.gov/health/suffolkvectorplan/tasks/task1_download.htm</u>, where Scoping comments are summarized). A second public concern was based on the course of the disease as it spread nationwide, where first-year high disease impacts have often been followed by greatly reduced second-year effects (see Lopez and Miller 2002, for instance), and generally low Old World disease incidence despite decades of exposure to the virus (see Weinberger et al. 2001, for example). These observations suggested to some that human populations rapidly

become immune to the disease, thereby obviating the need for control measures. In fact, some researchers suggest in areas where the virus is endemic that adult populations are immune to the disease (Ben-Nathan et al. 2003), at rates as high as 90 percent (Campbell et al. 2002).

Risks associated with WNV disease are generally understood to be critical or neuroinvasive illness or death, but not simple infection as most WNV infections are asymptomatic (Hayes 2001). Accurately determining the "natural" risk from the disease therefore depends on computing the incidence of critical or neuroinvasive illness and death, in the absence of an effective control program. Determining whether sufficient immunity develops in a population to reduce risks requires understanding the infection rate in the population. If these data were to be developed, it could be possible to estimate the current risk from the virus and to forecast risks to exposed populations in subsequent seasons. These calculations could also allow for comparisons of risks from disease to risks associated with disease control (as computed in Peterson et al. 2006, for instance).

Infection rates in six geographically distinct populations have been determined through serosurveys (Mostashari et al. 2001a, Mostashari et al. 2001b, Hadler et al. 2001, McCarthy et al. 2001, Mandalakas et al. 2005, Loeb et al. 2005). More recently, a nationwide analysis of blood donations was conducted to also determine infection rates and critical illness risks (Busch et al. 2006).

The two New York City serosurveys are widely cited in reports of human infection rates. In 1999, a survey was conducted in a small area of Douglaston, Queens, the apparent epicenter of the initial outbreak of WNV disease (Mostashari et al. 2001a). The second widely cited study was conducted in 2000 on Staten Island, part of a wider survey of the New York metropolitan area (Mostashari et al. 2001b). Other areas surveyed in the 2000 effort were Suffolk County

(Mostashari et al. 2001b) and Connecticut (Mostashari et al. 2001b, Hadler et al. 2001, McCarthy et al. 2001); the failure of the latter two surveys to detect infection is probably why they are not often discussed (Campbell et al. 2002). In 2002, a serosurvey was made in Cuyahoga County, Ohio, after disease impacts were detected there (Mandalakas et al. 2005). In early 2003, another serosurvey was made to capture impacts from the 2002 season in suburbs of Toronto, Ontario (Loeb et al. 2005). The infection rate and ratio of undiagnosed infections to hospitalized cases determined in those studies is presented in Table 1.

In 2003, screening of blood donations for WNV was instituted across the US (Busch et al. 2006). The data from these tests were analyzed to determine state-by-state and national seasonal infection rates, and then to project potential state-wide and nation-wide numbers of infections. These data could then be compared to the number of neuro-invasive cases reported to develop ratios of undiagnosed infections to hospitalized cases (Table 2).

The infection rate data in Table 1 represent rates determined for local areas where WNV disease incidence was apparent, based either on dead birds that tested positive for WNV, WNV-positive mosquito pools, or reports of human disease. The infection rate data in Table 2 are for state-wide areas, and therefore are generally not comparable to the serosurvey data sets. Three of the infection rates in Table 1 are much higher than the other three rates. These are results for the onset of WNV disease in particular areas. The lower three infection rates also were calculated for areas that implemented mosquito control programs designed to reduce WNV disease incidence. The serosurvey data therefore suggest WNV infection rates are two to three percent for naïve populations where mosquito control efforts may not be optimal. The only state-wide blood bank data that are this great (or greater) are from Kansas, Nebraska, Colorado, North Dakota, South Dakota, and Wyoming. *Culex tarsalis* is reported to be the primary WNV vector in this general

area (Bell et al. 2005), and may be more efficient than the vectors that transmitted WNV where the serosurveys were made.

Please note a serosurvey from Saskatchewan that reported even rates of infection as high as 10 percent for some sub-populations (Schellenberg et al. 2006), reported after this work was conducted, was not included in the analyses.

The undiagnosed case ratio from Table 1 has a general middle value of approximately 150 to one. Ratios found using the blood donation data tended to be higher, although there was a great deal of scatter in the data set. The overall national ratio was 260 to one (the mean of state ratios was 310 to 1, and the median was 240 to one). Table 3 lists compilations of annual CDC data sets for the number of WNV neuroinvasive cases and deaths through 2005. An approximate ratio of cases with critical illness to deaths is 10 to one.

MATERIALS AND METHODS

Study Setting

The study was conducted in Suffolk County. This suburban county occupies the eastern two-thirds of Long Island, and has a population of approximately 1.4 million. A large mosquito control program, designed to alleviate nuisance impacts and to address disease risks (predominantly but not exclusively from eastern equine encephalitis) (e.g., as reported in Bradley et al. 2000, Peyton et al. 1999) was in place at the time of the initial outbreak of WNV in 1999. Model Premises

Models based on mosquito behavior, prevalence, and infection rates that aim to mirror actual transmission of disease have been constructed for WNV (see Wonham et al. 2004, for instance). These models require accurate data collected at scales that are representative for the activities of interest. Although the Suffolk County Vector Control program has an extensive

surveillance program, researchers from the Harvard School of Public Health (led by Andrew Spielman and Richard Pollack) concluded that not enough relevant information was at hand. They thought that selecting default values to fill missing data sets would determine the overall results of the model, and so suggested another approach be undertaken to estimate potential health impacts from WNV (A. Spielman, HSPH, personal communication, 2004). There have been some recent advances in means to generate appropriate kinds of detail from program surveillance efforts (Diuk-Wasser et al. 2006) but mosquito ecology-grounded models still appear to require the collection of model specific information in order to be most credible.

Eisen and Eisen (2008) addressed a similar situation with an approach similar to that used here. They compared the density of detections of virus in mosquitoes to the density of human cases, across census tracts, to create a spatial risk model. As will be seen, the Suffolk County human disease incidence was not common enough to support that level of analysis, and there did not appear to be as clear a spatial bias in the detections of virus in mosquitoes, either. Therefore, the approach adopted here was slightly more simple.

The serosurvey data were used as a basis for a model of risks for a population exposed to WNV infection. The serological studies defined the area of exposure to the disease as where

- a) dead birds tested positive for WNV, and/or
- b) mosquito pools tested positive for WNV, and/or
- c) people were diagnosed with WNV.

"Areas," in the serosurvey studies, were generally defined arbitrarily by some form of political division. For Suffolk County, exposure was defined by zip codes where dead birds or positive mosquito pools occurred in any given year. The zip codes where at least one of the three conditions were recorded were mapped for 2000 through 2004 (a simplifying assumption was

made, absent relevant data for dead birds, positive pools, and with no human cases, that no one in the County was exposed to the disease in 1999) (Figures 1 to 5). All people living in the zip code were assumed to have been exposed, and to have the same degree of exposure, notwithstanding greater or lesser numbers of dead birds or positive pools. Working in or traveling through a zip code did not constitute exposure. This assumption is based on the near universal classification of WNV cases by home address, even if other exposures are plausible. Elements of the Model

The number of people infected by WNV each year (I(t)) was determined according to Equation 1:

$\mathbf{I}(\mathbf{t}) = (\mathbf{EP} - \mathbf{IP}) \mathbf{x} \mathbf{IR} (\mathbf{Eq. 1})$

where I(t) = the number of new infections at time step t, EP(t) = the exposed population at time step t, IP(t) = the immune portion of the exposed population at time step t, and IR = the infection rate.

The exposed population was determined for each year using a Geographical Information System (GIS) analysis of Census block data for 2000 combined with zip code coverages (ESRI, Redlands, CA, accessed 2005) for 2000 to 2004, with the overall population of the County was held constant at the 2000 Census totals. Equation 2 defines the exposed populations for any one year (**EP(t)**):

$\mathbf{EP}(\mathbf{t}) = \mathbf{\Sigma} \mathbf{P}(\mathbf{ZC}) \text{ (Eq. 2)}$

where $\mathbf{EP}(\mathbf{t})$ = the exposed population at time step t and $\mathbf{P}(\mathbf{ZC})$ = the population in zip codes with dead infected birds, positive mosquito pools, or human cases for any year.

The infected population each year was assumed to have seroconverted and to be subsequently immune from the disease, as is generally thought to be the case for WNV (Campbell et al. 2002). Thus, the immune portion of the population in any year (IP(t)) was incapable of becoming infected, and so was subtracted from the exposed population in the first five years (2000 to 2004) per Equation 3:

$$IP(t) = ((CP - (I(1) + ... + (I(t-1))/CP) \times EP(t) (Eq. 3))$$

where IP(t) = the immune portion of the exposed population at time step t, CP = County population, I(t) = the number of new infections at time step t, and EP(t) = the exposed population at time step t.

Although the number of potential infections each year was determined for particular sets of zip codes based on the surveillance data, the resulting immune population was set so that it was distributed evenly throughout the County. This simplification was made to make the computations less complicated, and has little effect on the overall results because of the widespread degree of exposure across the County most years.

The percentage of the population with immunity (**PI**) in any of the first five years was determined by Equation 4:

$$PI = ((\Sigma I(t))/CP) \times 100 (Eq. 4)$$

where \mathbf{PI} = the percent infected, $\mathbf{I}(\mathbf{t})$ = the number of new infections at time step t, and \mathbf{CP} = County population. This percentage increased each year, and thus seroconversion decreased the number of people in each area that would be available to be infected each year, as it should. Please note that the proportion of seroconverted individuals was consistent with County-wide infection rates, but potentially not for rates that might have been computed for individual zip codes.

Because the model for the first five years showed increasing rates of seroconversion, projections of potential future impacts were determined to be suitable to account for any long-

term effects of increasing immunity rates. The model was modified to address several new factors. The population of the County was allowed to vary. The overall rate of increase was determined from population surveys over the period 2000 to 2004, using data generated by the local electricity utility based on projections from the 2000 Census based on hook-ups and disconnections (LIPA 2003, LIPA 2005). Therefore, the population of the County for each year past 2004 (**CP(t)**) was assumed to be determined per the recursive Equation 5:

$CP(t) = CP(t-1) \times 1.00836$ (Eq. 5)

where CP(t) = County population at time t, CP(1) = LIPA estimate for 2004 x 1.00836, and 0.0836 = the population increase for the County from the 2000 census to the estimate of 2004 population, divided by the five year period, and then divided by the estimated 2004 population (based on data in LIPA 2005).

The number of seropositive individuals (immune people) in the population in any year $(\mathbf{IP}(\mathbf{t}))$ was determined per Equation 6:

$$IP(t) = IP(t-1) - (IP(t-1) \times D) + (IP(t-1) \times NER) + I(t) (Eq. 6)$$

where IP(t) = the immune portion of the exposed population at time step t, **D** = County-wide mortality rate, **NER** = the net emigration rate, and **I**(t) = the number infected at time step t.

The number of new infections at any time step was determined by the number of susceptible, exposed individuals times the infection rate, per equation 1. Since the entire County was assumed to be exposed to WNV agents for this part of the exercise, the population that could be infected in any one year was based on the infection rate and the number of naïve people in the County. The naïve population was the total population minus the number that was immune, per Equation 7:

$$\mathbf{I}(\mathbf{t}) = (\mathbf{CP}(\mathbf{t}) - \mathbf{IP}(\mathbf{t}-\mathbf{1}) + (\mathbf{CP} \times \mathbf{D}) - (\mathbf{CP} \times \mathbf{NER})) \times \mathbf{IR} (\text{Eq. 7})$$

where I(t) = the number infected at time step t, CP(t) = County population at time t, IP(t) = the immune portion of the exposed population at time step t, D = County-wide mortality rate, NER = the net emigration rate, and IR = the infection rate. Note that the seroconverted population lags by a time step.

At this stage, the percentage of the population that was immune could be calculated per Equation 4 for any time stage.

Model Input Values

The first iteration of the model (Model A) assumed a constant increase in population for the County through 2025. Model A' assumed that the County's population was capped at 2010 levels to reduce the "dilution" of immune populations by continual in-migration. This is based on an assumption that growth in Suffolk County is somewhat limited by extensive open-space and farmland development rights acquisitions (Daniels 2001), although the date selected for a population cap is arbitarary.

For the first model iteration, an infection rate of two percent was used, based on the Table 1 information. This implies that 2,000 out of every 100,000 exposed people would become infected, at least in the first year of exposure. Table 1 data sets also suggest that one out of 150 infected people have severe neurological effects requiring hospitalization, so that 15 people out of the hypothetically-exposed population of 100,000 might require hospitalization. Furthermore, data on fatalities suggest that one or two of those hospitalized people will die from the disease. Thus, the serological data on WNV infections and associated illnesses suggest that, through a very generalized estimation, out of 100,000 exposed individuals in any year, one or two people might be expected to die from the effects of the disease.

The blood donation testing suggested that a much higher ratio of undetected illness than the 150 to one ratio extrapolated from serosurveys might be appropriate to consider. Although other rates are possible to consider, based Busch et al. (2006), the national average of 260 undiagnosed infections per critical illness was selected. The Staten Island, Cleveland, and Ontario studies defined an area within which infections were limited (based on detections of illnesses, positive mosquito pools, and positive dead bird tests), and within that area determined the number of cases of critical illness that had occurred. If the blood donation testing results are accurate, they would indicate that the serosurvey data sets underestimated the number of undiagnosed infections (assuming there were no undiagnosed critical illness cases of WNV). The higher undiagnosed case ratio would suggest that the infection rate should be approximately 60 percent higher than the rate associated with a ratio of 150 undiagnosed cases per critical illness. If the Ontario infection rate (3.1 percent) had been 60 percent greater, then the infection rate there would have actually been 5 percent. The state blood bank data suggest 5 percent may be a reasonable maximum infection rate, as this is the greatest amount detected across any one state (although data from Saskatchewan indicate even higher infection rates are possible (Schellenberg et al. 2006)).

Mortality was assumed to be 831 deaths per 100,000 (the overall US mortality rate for 2003) (Hoyert et al. 2005), removing some proportion of the immune population from the County. The mean County birth rate for 2000 to 2004 (1,393 per 100,000) (LIPA 2003, LIPA 2005) was used for projected population increases post 2004, to account for additional naïve residents. Migration into the County (the increase in population above net births over deaths, a rate of 0.274 percent) was assumed to have seropositive rates similar to the County as a whole at

the time of migration into the County. It was also assumed that everyone in the County was exposed to WNV (lacking surveillance data to exclude some portion of the County).

Therefore, several variations on the basic models were made. Model B used a 3 percent infection rate, Model C used a 4 percent infection rate, and Model D used a 5 percent infection rate.

All models were run using 2000 to 2004 exposure definitions, and also for the 2005 to 2025 period. Models B', C', and D' used the capped 2010 populations, similar to Model A'.

RESULTS

Table 4 presents the model results for the defined exposed populations for 2000 to 2004. Model A results suggest that as many as 64 people might be expected to have been fatally infected in Suffolk County across the five year period, assuming that the infection rate in Suffolk County were similar to the 2 percent value developed from the serosurvey data. The model shows increasing immune rates, as would be expected. Model B, with a lower critical illness to infection ratio but a slightly higher infection rate, shows impacts that are slightly less than Model A (546 critical illnesses in total, and 54 deaths). Model C and Model D show higher projected impacts, but also much higher immune rates.

Table 5 shows selected years' results over the 2005 to 2025 period. Despite much higher immunity rates for Models B, C, and D, impacts are approximately the same as Model A due to the higher underlying infection rates (and so subsequently higher numbers of critical illnesses and deaths). In 2025, impacts associated with Model B are only 28 percent lower than those for Model A.

Capping the population of the County for the years 2005 to 2025 decreased the overall impact of the disease on the County by approximately 15 percent, but did not change the relative

effect of the different scenarios very much (Table 6). Model B' has fewer projected impacts than Model A', by on the order of 30 percent. Even with immunity rates approaching two-thirds of the County as a whole, the naïve population that remains, coupled with the high infection rates, still results in projections of 10 or more deaths a year from WNV in 2025 for all models. The death rates for these scenarios range from 0.58 per 100,000 (for Model B') to 0.90 per 100,000 for Model A. Predicted total deaths over the 21 year modeling runs range from 265 to 377, with thousands of neuro-invasive cases.

DISCUSSION

The modeling exercise was intended to address two concerns raised by the public regarding WNV in Suffolk County. They were:

- 1) is WNV a serious health threat requiring active mosquito control?
- 2) might residents quickly become immune to the disease, and so reduce the need for mosquito control to prevent disease transmission?

The modeling suggests that if WNV transmission were to occur in Suffolk County in much the same fashion as it did in Queens in 1999, and in Ohio and Ontario in 2002, then more than a hundred people would become critically ill and more than ten people might die each year. To put such mortality into perspective, the homicide rate for Suffolk County was 1.5 to 2.5 per 100,000 for 2000 to 2002 (22 to 37 deaths per year) (per data from New York State Department of Health, updated July 2004,

http://www.nyhealth.gov/nysdoh/chac/cha/plots/violence/homici47.htm). WNV infections occur across a six month period of the year, but peak in only several weeks in August and September (Petersen and Marfin 2002). Assuming 75 percent of the infections occur during those two months, WNV appears to constitute a greater mortality threat than homicide for residents of Suffolk County, at least during part of the year. Traffic fatalities for 2001 (183 deaths) and 2002 (169 deaths) (per county-by-county compilations of data by the US Department of Transportation, www-nrd.nhsta.dot.gov/pdf/nrd-30/NCSA/AlcRpt/2002/New%20York.pdf) were approximately an order of magnitude higher than the projected WNV deaths. So, even factoring in the seasonal restrictions on WNV impacts, car accidents are approximately twice the risk to local residents. Therefore, the maximal seasonally adjusted risk of fatality for Suffolk County residents from WNV is greater than that posed by homicide, but less than that posed by driving. The higher risk associated with automobile use is generally accepted as a part of modern life, but risks associated with violence generally are not, although they are lower.

Perhaps a more interesting comparison is to influenza. In 2002, there were 272 deaths across the County from influenza and pneumonia (according to the Annual Report of the Health Department, <u>http://www.co.suffolk.ny.us/Health%20Services/AnnRpt2003.pdf</u>), which is more than an order of magnitude above the projected West Nile virus disease death rate for the same year. Substantial County resources are expended to try to minimize flu impacts, primarily through flu vaccination drives. There are few protests in the County regarding the relatively high number of deaths from the flu (as compared to the projected effects from West Nile virus disease). Perhaps because there is no vaccine to help ward off impacts, there is a readily identifiable, noxious vector of the disease, and the vector is perceived as being controllable, it seems probable that there would be great public out cry if hundreds of people died from West Nile virus disease. Differences in the perception of risk have been discussed for over 20 years; identified important elements in increasing risk perceptions include the novelty of the risk factor and lesser degrees of control over the cause of risk (Slovic 1987). Continuing exposure to the disease may reduce the novelty effect; and, if public education succeeds in persuading the public

they have control over disease risks through risk reduction steps (Loeb et al. 2005), the perception of WNV disease risks may decrease with time.

However, absolute reductions in risk over time appear to be small. The notion that exposure to WNV will rapidly lead to an immune population does not seem well-founded. Higher infection rates clearly lead to higher immunity rates over time. However, since people are dead-end hosts for WNV, increases in immunity only lead to proportional decreases in health impacts. A much greater infection rate would be required to result in rapid general immunity. Although blood bank testing found variable rates for the relationship between seroconversion rates and incidences of critical illness, unless these ratios were of a magnitude akin to those measured for West Virginia and California in 2003 (1,300 and 1,600 to one, respectively), higher infection rates appear to lead to predictions of greater human impacts, even when larger infection to critical illness ratios are used.

Actual impacts to human health in Suffolk County have been much less. Through 2004, only 19 infections and 4 deaths had occurred (Table 7) (data from S. Campbell, Arthropod-Borne Disease Laboratory, Suffolk County Department of Health Services). There is no reason to assume that general trends regarding undiagnosed infections and serious illnesses are different for Suffolk County than have been found elsewhere, and so it seems likely that the number of infections in Suffolk County is much less than expected, if transmission of the virus occurred in the County as it did elsewhere. It is widely assumed that *Culex pipiens* is the primary human vector for WNV in New York, due to its high infection rate (Lukacik et al 2006), and that it was the primary vector in Queens in 1999 (Apperson et al. 2002), Ohio in 2002 (Mans et al. 2004), and Ontario in 2002 (Shapiro and Micucci 2003), although no contemporary surveillance was made in the exact locations where the serosurveys were made to support these suppositions.

Other information suggests that transmission to people may be more complicated than these simple explanations (see Andreadis et al. 2004 and Kilpatrick et al. 2006, for instance). *Culex pipiens* is a very common mosquito in Suffolk County (see Table 8), so that natural differences in mosquito populations are not obviously the cause of the protection received by Suffolk County residents. Nonetheless, it is not clear that the natural mosquito ecology in the northeast US is capable of transmitting WNV so as to cause large infection rates. *Culex pipiens* and the mosquitoes considered by managers to be the most likely bridge vectors in Suffolk County (*Aedes vexans* and *Ochlerotatus sollicitans*) are not rated as very competent WNV vectors by Turrell et al. (2005). Data from Connecticut (Andreadis et al. 2004) suggest that *Culex salinarius* is probably the greatest transmission risk there. Surveillance in Suffolk County has generally not speciated *Culex restuans/pipiens/salinarius*, and so the general prevalence of *Culex salinarius* in the greater *Culex* population has not been quantified. So the presence of good transmitters of WNV disease to people in Suffolk County has not been clearly demonstrated.

Still, in 1999, a restricted area in Queens suffered fairly concentrated effects, with infection rates on the order of those seen in areas in the Midwest and far West. The mosquitoes that caused this impact are likely to be found in Suffolk County, although Suffolk County is at least 60 km to the east, because of general habitat and environmental features that are shared between the two areas (similar stormwater infrastructures and overall vegetation types, resulting in similar mosquito habitat opportunities). Otherwise, compilations of disease impacts by the Centers for Disease Control and Prevention (CDC) (see

<u>www.cdc.gov/NCIBOD/DVBID/WESTNILE</u>) have consistently shown low impacts in the northeast relative to much of the rest of the country.

It is tempting to suggest that active mosquito control programs cause this difference. The implementation of focused mosquito control in years following the initial outbreaks of WNV in particular places seems to be a reasonable explanation for the generally rapid declines in infections that are measured in the years following initial outbreaks. For Suffolk County, the lack of general impacts in 1999 and subsequent years could be attributable to the comprehensive control program that was in place. However, much of the control effort is focused on Ochlerotatus sollicitans and other salt marsh mosquitoes. Even so, the program has always been responsive to complaints regarding all biting mosquitoes, and ongoing (although unrealized) threats from eastern equine encephalitis mean that all potential bridge vectors are targeted for control in populated areas. Program managers believe that the reason no human cases were found in the serosurvey effort in 2000, despite many detections of infected Culex pipiens pools, was that contemporaneous aerial adulticide treatments had targeted Aedes vexans, and so reduced the transmission risks to people (D. Ninivaggi, Superintendent, SCVC, personal communications, 2004). Suffolk County treats catch basins now to reduce WNV risks and has an active household source reduction program, as do surrounding jurisdictions in New York State and Connecticut. All of these areas have similarly low WNV infection rates. Unlike Connecticut and most other New York jurisdictions, Suffolk County also conducts adulticiding in areas with high numbers of biting adult mosquitoes or in areas with high levels of WNV activity as measured in mosquitoes and/or birds. However, the most likely vectors of WNV are still commonly found in trapping in Suffolk County (Table 8) and other jurisdictions in the northeast (see Andreadis et al. 2004, for instance). It may be that the control programs reduce vector activity to below some threshold level that reduces generally effective disease transmission to humans while not totally interdicting circulation in birds and mosquitoes (so that the positive dead birds and mosquito

pools are still detectable). It is clear, however, that some form of undetected near universal human immunity is not a realistic explanation for the seeming lack of illness, given the results of the model.

The simple model developed here fails to estimate actual WNV disease effects in Suffolk County. The model clearly shows that herd immunity is not the reason for the underestimation of effects. Thus, it strongly implies that mosquitoes in the County are not capable of causing infections as mosquitoes elsewhere in country can – but the model does not provide information to determine if this is because of control efforts or a lack of vector competence of the local mosquitoes. The model appears to show that WNV presents a potentially serious health threat to residents of the County (and other areas with circulating WNV). The threat is defined as the risks associated with virus transmission as has been documented elsewhere, which would lead to tens of deaths and hundreds of serious illnesses in the County. Because the reason for the lack of local disease impacts cannot be isolated, it seems reasonable to consider the model risks as a baseline measure of the overall WNV disease threat. It is a reasonable maximum estimate of potential effects - a comparison often used in health risk assessments (NRC 2007). Thus, it is also a reasonable level of potential disease impacts to which control impacts can be compared. The comparison is likely to be useful over time, as well, as the model further suggests it is unlikely that continued exposure to WNV will substantially reduce risks through developing population immunity.

The modeling suggests that the determination of baseline risks for this disease can be generated, even with an active control program, even if- the control program appears to contribute to preventing disease transmission. The simplifying assumptions and the failure of the model to mimic real conditions underline that the model cannot be used as a precise tool for

predicting risk. It is useful to bounding risks, and obtains validity because its basis is quantifications of impacts that have actually occurred due to WNV. This basis in real results attributed to the disease makes it difficult to cavalierly dismiss the estimates, as might be the case if a more sophisticated but also more theoretically-based approach were to have been assayed, where appropriate information was not available and so default values that probably constrained the results needed to be selected.

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Tables

Table 1.	Serosurvey	Results
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Year	Location	Infection Rate (percent)	Undiagnosed Infections per Case
1999	Douglaston	2.6	140
2000	Staten Island	0.5	160
2000	Suffolk County	0.2	n.a. ¹
2000	Connecticut	0	n.a. ²
2002	Cuyahoga County	1.9	170
2002	Ontario	3.1	160

¹ No human cases were diagnosed.

² There were no sero-positive sampling results, but one case of WNV was diagnosed.

Table 2. 2003 WNV Blood Donation Testing Results: Infection Rates > 3% or Undiagnosed infections > 1,000 per Case, or States Where Serosurveys Were Conducted (modified from Busch et al. 2003)

State	Infection Rate (percent)	Undiagnosed Infections per Case
California	0.0	1600
Colorado	4.3	320
Kansas	2.1	650
Nebraska	4.9	440
New York	0.1	240
North Dakota	4.1	280
Ohio	0.1	140
South Dakota	4.0	200
West Virginia	0.1	1300
Wyoming	3.5	190
US total	0.3	260
All States median		240
All States mean		310

Year	Meningitis- Encephalitis Cases	Deaths	Ratio of Critical Illnesses to
			Deaths
1999	59	7	8
2000	19	2	10
2001	64	9	7
2002	2,946	284	10
2003	2,860	264	11
2004	1,142	100	11
2005	1,294	119	11

Table 3. Critical WNV Human Cases and Deaths, US, 1999-2005

(collected from http://www.cdc.gov/NCIDOD/DVBID/WESTNILE)

Model	Years	Population Exposed	Hospitalizations	Deaths	Resulting Immune County-wide Percentage (Population of 1,419,369)
Model	2000	1,135,878	152	15	1.5
Α	2001	1,195,260	157	16	3.1
	2002	1,168,088	151	15	4.6
	2003	1,227,931	156	16	6.2
	2004	191,328	24	2	6.5
	Total		640	64	
Model	2000	1,135,878	131	13	2.3
В	2001	1,195,260	135	13	4.7
	2002	1,168,088	128	13	6.9
	2003	1,227,931	132	13	9.2
	2004	191,328	20	2	9.5
	Total		546	54	
Model	2000	1,135,878	175	17	3.1
C	2001	1,195,260	178	18	6.2
	2002	1,168,088	169	17	9.2
	2003	1,227,931	172	17	12.2
	2004	191,328	26	3	12.5
	Total		720	72	
Model	2000	1,135,878	218	22	3.8
D	2001	1,195,260	221	22	7.7
	2002	1,168,088	207	21	11.3
	2003	1,227,931	209	21	15.0
	2004	191,328	31	3	15.4
	Total		886	89	

 Table 4. Modeled Suffolk County West Nile Virus Incidence 2000-2004

Model	Years	Population	Hospitalizations	Deaths	Resulting Immune County-wide Percentage
Model	2005	1,495,221	187	19	8.2
А	2010	1,558,775	178	18	16.0
	2015	1,625,031	171	17	22.6
	2020	1,694,103	166	17	28.0
	2025	1,766,111	162	16	32.6
	Total		3,619	360	
Model	2005	1,495,221	156	16	12.0
В	2010	1,558,775	142	14	21.1
	2015	1,625,031	132	13	31.8
	2020	1,694,103	123	12	38.8
	2025	1,766,111	116	12	43.2
	Total		2,802	280	
Model	2005	1,495,221	202	20	15.8
С	2010	1,558,775	176	18	29.6
	2015	1,625,031	156	16	40.0
	2020	1,694,103	141	14	47.8
	2025	1,766,111	131	13	53.7
	Total		3,355	336	
Model	2005	1,495,221	244	24	19.4
D	2010	1,558,775	203	20	35.6
	2015	1,625,031	169	17	47.1
	2020	1,694,103	152	15	53.9
	2025	1,766,111	138	14	61.3
	Total		3,775	377	

Table 5. Projected Suffolk County West Nile Virus Incidence 2005-2025

Model	Years	Population	Hospitalizations	Deaths	Resulting Immune County-wide
					Percentage
Model	2005	1,495,221	187	19	8.2
A'	2010	1,558,775	178	18	16.0
	2015	1,625,031	163	16	23.5
	2020	1,694,103	149	15	29.8
	2025	1,766,111	138	14	35.3
	Total		3,420	341	
Model	2005	1,495,221	156	16	12.0
B'	2010	1,558,775	142	14	21.1
	2015	1,625,031	124	12	33.1
	2020	1,694,103	109	11	41.2
	2025	1,766,111	97	10	47.9
	Total		2,635	265	
Model	2005	1,495,221	202	20	15.8
C'	2010	1,558,775	176	18	29.6
	2015	1,625,031	147	15	41.6
	2020	1,694,103	124	12	50.8
	2025	1,766,111	106	11	57.9
	Total		3,144	316	
Model	2005	1,495,221	244	24	19.4
D'	2010	1,558,775	203	20	35.6
	2015	1,625,031	162	16	49.0
	2020	1,694,103	131	13	58.7
	2025	1,766,111	109	11	65.9
	Total		3,525	353	

Table 6. Projected Suffolk County West Nile Virus Incidence 2005-2025, Population Capped at 2010 Levels

Table 7. Actual Suffolk County West Nile Virus Impacts

	1999	2000	2001	2002	2003	2004
Hospitalizations	0	0	1	8	10	0
Deaths	0	0	0	2	2	0

	NJ Light Trap Percent	CDC Light Trap Percent	CDC Gravid Trap Percent
Species	(n = 58,469)	(n = 45,706)	(n = 13,811)
Aedes vexans	7.3	4.8	<0.1
Coquillettidia perturbans	7.8	17.6	<0.1
Culex spp.	12.2		
Culex pipiens-restuans		9.2	95.0
Culiseta melanura	0.7	5.8	<0.1
Ochlerotatus canadensis	0.5	17.7	0.5
Ochlerotatus sollicitans	33.4	12.4	0.6
Ochlerotatus			
taeniorhynchus	7.7	12.0	<0.1

Table 8. Common (>5%) Suffolk County Mosquitoes (2005 Trap Results)

Data provided by the Arthropod-borne Disease Laboratory, Suffolk County Department of

Health Services

Figure Legends

Figure 1. West Nile virus exposure in Suffolk County, NY, 2000, determined by positive dead birds and mosquito pools, defined by zip codes

Figure 2. West Nile virus exposure in Suffolk County, NY, 2001, determined by positive dead birds and mosquito pools, defined by zip codes

Figure 3. West Nile virus exposure in Suffolk County, NY, 2002, determined by positive dead birds and mosquito pools, defined by zip codes

Figure 4. West Nile virus exposure in Suffolk County, NY, 2003, determined by positive dead birds and mosquito pools, defined by zip codes

Figure 5. West Nile virus exposure in Suffolk County, NY, 2004, determined by positive dead birds and mosquito pools, defined by zip codes