



*Suffolk County  
Vector Control &  
Wetlands Management  
Long Term Plan &  
Environmental Impact  
Statement*

**Task 3: Literature Search  
Part 4: Serosurveys for West Nile Virus**

*Prepared for:*

**Suffolk County Department of Public Works  
Suffolk County Department of Health Services  
Suffolk County, New York**

**CASHIN ASSOCIATES, P.C.**  
1200 Veterans Memorial Highway, Hauppauge, NY

*January 2005*

**SUFFOLK COUNTY VECTOR CONTROL AND WETLANDS MANAGEMENT  
LONG - TERM PLAN AND ENVIRONMENTAL IMPACT STATEMENT**

**PROJECT SPONSOR**

**Steve Levy**  
**Suffolk County Executive**



**Department of Public Works**

Charles J. Bartha, P.E.  
*Commissioner*  
Richard LaValle, P.E.  
*Chief Deputy Commissioner*  
Leslie A. Mitchel  
*Deputy Commissioner*

**Department of Health Services**

Brian L. Harper, M.D., M.P.H.  
*Commissioner*  
Vito Minei, P.E.  
*Director, Division of Environmental Quality*

**PROJECT MANAGEMENT**

Project Manager: Walter Dawydiak, P.E., J.D.  
Chief Engineer, Division of Environmental Quality, Suffolk County Department of Health Services

**Suffolk County Department of Public  
Works, Division of Vector Control**

Dominick V. Ninivaggi  
*Superintendent*  
Tom Iwanejko  
*Entomologist*  
Mary E. Dempsey  
*Biologist*

**Suffolk County Department of  
Health Services, Office of Ecology**

Martin Trent  
*Acting Chief*  
Kim Shaw  
*Bureau Supervisor*  
Robert M. Waters  
*Bureau Supervisor*  
Laura Bavaro  
*Senior Environmental Analyst*  
Erin Duffy  
*Environmental Analyst*  
Phil DeBlasi  
*Environmental Analyst*  
Jeanine Schlosser  
*Principal Clerk*

## **SUFFOLK COUNTY LONG TERM PLAN CONSULTANT TEAM**

<b>Cashin Associates, P.C.</b>	<b>Hauppauge, NY</b>
<b>Subconsultants</b>	
Cameron Engineering, L.L.P.	Syosset, NY
Integral Consulting	Annapolis, MD
Bowne Management Systems, Inc.	Mineola, NY
Kamazima Lwiza, PhD	Stony Brook University, Stony Brook, NY
Ducks Unlimited	Stony Brook, NY
Steven Goodbred, PhD & Laboratory	Stony Brook University, Stony Brook, NY
RTP Environmental	Westbury, NY
Sinnreich, Safar & Kosakoff	Central Islip, NY
Bruce Brownawell, PhD & Laboratory	Stony Brook University, Stony Brook, NY
Anne McElroy, PhD & Laboratory	Stony Brook University, Stony Brook, NY
Andrew Spielman, PhD	Harvard School of Public Health, Boston, MA
Richard Pollack, PhD	Harvard School of Public Health, Boston, MA
Wayne Crans, PhD	Rutgers University, New Brunswick, NJ
Susan Teitelbaum, PhD	Mount Sinai School of Medicine, NY
Zawicki Vector Management Consultants	Freehold, NJ
Michael Bottini, Turtle Researcher	East Hampton, NY
Robert Turner, PhD & Laboratory	Southampton College, NY
Christopher Gobler, PhD & Laboratory	Southampton College, NY
Jerome Goddard, PhD	Mississippi Department of Health, Jackson, MS
Sergio Sanudo, PhD & Laboratory	Stony Brook University, Stony Brook, NY
Suffolk County Department of Health Services, Division of Environmental Quality	Hauppauge, NY

Primary research for this report was conducted by Cashin Associates (personnel including David J. Tonjes, PhD, and Michael Tumbarello). It was edited and revised in response to comments by Cashin Associates (personnel including David Tonjes, PhD). Review was provided by Suffolk County Department of Public Works, Division of Vector Control, and Suffolk County Department of Health Services (personnel including Phil DeBlasi and Erin Duffy). Additional comments have been received from \_\_\_\_\_.

## TABLE OF CONTENTS

<b>EXECUTIVE SUMMARY .....</b>	<b>1</b>
<b>1. INTRODUCTION .....</b>	<b>2</b>
<b>2. WEST NILE VIRUS .....</b>	<b>3</b>
2.1 History in the United States .....	3
2.2 Standard Medical Reporting .....	4
<b>3. WNV SEROLOGICAL SURVEYS .....</b>	<b>7</b>
3.1 Douglaston, NY, 1999 .....	7
3.2 Staten Island, NY, 2001 .....	8
3.3 Suffolk County, NY, 2001 .....	8
3.4 Fairfield County, CT, 2001 .....	9
3.5 Cuyahoga County, OH, 2002 .....	9
3.6 South Oakville, Ontario, Canada, 2003 .....	10
3.7 unidentified location, Wisconsin, 2002 .....	10
3.8 Bucharest, Romania, 1996 .....	11
3.9 Hashimiah, Jordan, 1998 .....	11
3.10 WNV in the Blood Supply .....	11
<b>REFERENCES .....</b>	<b>13</b>
<b>List of Tables</b>	
Table 2-1. Human Cases of WNV in the US, 1999-2003 .....	6

## **List of Abbreviations and Acronyms**

CDC	Centers for Disease Control and Prevention
ELISA	enzyme-linked immunoabsorbent assay
IgM	immunoglobulin M
km <sup>2</sup>	square kilometer
mi <sup>2</sup>	square mile
NYCDOH	New York City Department of Health
NYSDOH	New York State Department of Health
PRNT	plaque reduction neutralization
SCDHS	Suffolk County Department of Health Services
SLE	St. Louis encephalitis
WHO	World Health Organization
WNV	West Nile virus

## **EXECUTIVE SUMMARY**

West Nile virus (WNV) infections can range from asymptomatic to fatal illnesses. Human serologic surveys have been conducted to determine the rate of infections in humans. Most of the surveys found approximately two percent of humans in highly exposed areas may seroconvert. The highest infection rate, found in Jordan, was eight percent; however, this was apparently from a strain of the virus that does not cause virulent disease. In areas with the more virulent form of WNV, infection rates ranged from zero in Connecticut in 2000 to four percent in Romania in 1996.

Data on symptomatic illness appeared to show that approximately 20 percent of those infected might experience measurable impacts. However, in approximately one out of every 150 cases, serious disease, such as encephalitis or meningitis requiring hospitalization, can occur. Nationwide fatality rates of those with the most severe symptoms have ranged from six percent in 2000 to 14 percent in 2001. The most recent data suggests the fatality rate for these individuals is slightly less than ten percent. This suggests that one out of every 1500 cases may result in a fatality in any given year.

These data can be used to make a very simplistic estimate of the annual risks for a population exposed to WNV infection. An infection rate of two percent would imply that 2,000 out of 100,000 exposed people would become infected. Approximately 20 percent of those infected exhibit fever or other flu-like symptoms. Thus, approximately 400 people out of 100,000 might have such symptoms. Estimates also show that one out of 150 infected people have severe neurological effects requiring hospitalization. This suggests that 10 to 15 people out of the hypothetically-exposed population of 100,000 might require hospitalization. Furthermore, data on fatalities from the disease suggests that one or two those hospitalized people will die from the disease. Thus, the serological data on West Nile virus infections and associated illnesses suggests that, through a very simplistic and 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.

## **1.0 INTRODUCTION**

Serology is the study of analyzing blood to detect the presence of antibodies against a microorganism. A serological survey is when samples are taken from a population to determine exposure to a microorganism.

To perform a serological test, blood is drawn and then analyzed in a laboratory. Certain microorganisms, known as antigens, stimulate the body to produce antibodies during an active infection. In the laboratory, the antibodies react with antigens in specific ways that can be used to confirm the identity of the specific microorganism (WHO, 2003).

There are several serology techniques that can be used depending on the suspected antibodies. Commonly used serology techniques include:

- agglutination
- precipitation
- complement-fixation
- fluorescent antibodies

(WHO, 2003)



## 2.0 WEST NILE VIRUS

### 2.1. History in the United States

An outbreak of human encephalitis, then of unknown etiology, began in New York City in early August 1999. On September 14, 1999, a virus was isolated at the National Veterinary Services Laboratories in Ames, Iowa, from tissues of a crow from the New York City area. This virus was later identified as West Nile virus (WNV) and confirmed as the cause of the human encephalitis outbreak (Lanciotti et al., 1999). A total of 59 human cases were confirmed as exhibiting clinical illness from the virus, and seven deaths resulted (Nash et al., 2001).

Surveillance has continued in the affected area and in regions where WNV has spread. Surveillance consists of investigating suspect cases in horses and humans, capturing and testing wild birds, testing bird carcasses, observation of sentinel chickens, and particularly the collection and testing of mosquitoes (CDC, 2003).

Until 2002, WNV was confined to states in the eastern half of the country. By the summer of 2002, all but six of the continental states reported WNV in birds, mosquitoes, animals or humans (CDC, 2003). In 2004, an epidemic occurred in Arizona, and the virus reached California (Zielinski-Gutierrez, 2004).

WNV was first described in Uganda in 1937. It is endemic in parts of Africa and the Near East, where it generally causes minor human impacts. Some have described its locus as being the Mediterranean basin. The first known virulent outbreak of WNV was in Romania in 1996. Concurrent with its appearance in the US in 1999 was the recognition of a smaller, but similar, outbreak in Russia. These outbreaks were characterized by considerable numbers of human deaths. The change in virulence appears to coincide with the mosquito species *Culex pipiens* becoming a major vector (Hayes, 2001).

Birds appear to be the primary host of WNV. *Cx. pipiens* primarily feeds on birds; therefore, either *Cx. pipiens* also feeds often enough on people to spread the virus, or another species of mosquito, a bridge vector, actually transmits the virus from birds to some other infected host to people. *Cx. quinquefasciatus* and *Cx. tarsalis* appear to be the major vectors of WNV in the

areas where these species are common, which is south and west of the northeast US. If these latter two species are more efficient vectors than *Cx. pipiens* and/or *Cx. pipiens* and the unknown bridge vector, it would explain the greater human toll outside of the northeast US (CDC, 2003).

## 2.2 Standard Medical Reporting

WNV causes several forms of illness in humans. West Nile fever, the least virulent form, is characterized by symptoms such as fever, body aches, headache, and, sometimes, swollen lymph glands and rash. West Nile fever generally lasts only a few days, although in some cases symptoms have been reported to last longer, up to several weeks. West Nile fever does not appear to cause any long-term health effects and there is no specific treatment for West Nile fever – or for any WNV infection, for that matter. People with West Nile fever recover on their own, although symptoms can be relieved through various treatments appropriate for flu and flu-like symptoms (e.g., standard medication for headache, fever, body aches, etc.) (Huhn et al., 2003).

Some people may develop a brief, West Nile fever-like illness before they develop more severe disease, although the percentage of patients in whom this occurs is unknown (Huhn et al., 2003).

Occasionally, an infected person may develop a more severe course of the disease – West Nile encephalitis or West Nile meningitis. Encephalitis is an inflammation of the brain, and meningitis is an inflammation of the membrane around the brain and the spinal cord. Although there is no treatment for WNV infection itself, a person with severe disease often needs to be hospitalized. Care may involve providing intravenous fluids, respiratory support, prevention of secondary infections, and general nursing support of the symptoms (Huhn et al., 2003).

In terms of standard medical reporting, “confirmed cases” of WNV infection result when physicians who have treated patients that exhibited symptoms severe enough to require medical assistance report these cases of human illness. When classifying the severity of illness, a general set of rules is followed. In order to classify a human as being ill from WNV, also known as West Nile fever, the patient must exhibit the typical symptoms associated with the disease. These symptoms include headache, tiredness, and body aches, occasionally with a skin rash on the trunk of the body and swollen lymph glands (CDC, 2001). The Centers for Disease Control and

Prevention (CDC) often suggests that about 20 percent of the people who become infected will develop these symptoms, although there is no clear source for this general rule of thumb. In order to classify a human as being severely ill from WNV, also known as West Nile encephalitis or meningitis, the patient must exhibit more intense neurological symptoms, including headache, high fever, neck stiffness, stupor, disorientation, coma, tremors, convulsions, muscle weakness, and paralysis. These results are confirmed by antigen tests (CDC, 2001).

Local health departments are generally tasked with the collection of local information, and with reporting cases to state health departments. The state health departments are further tasked with reporting disease incidence to ArboNET, a web-based database maintained by 54 state and local health agencies and CDC (O'Leary et al., 2002). Complete WNV statistics, based on this reporting system, are in Table 2-1.

Table 2-1. Human Cases of WNV in the US, 1999-2003 (citation?)

Year	States Reporting	Counties Reporting	Cases	Meningitis-Encephalitis Cases	Deaths
1999	1	6	59	59	7
2000	2	6	18	18	1
2001	10	39	66	64	9
2002	37 + DC	619	3,389	2,354	199
2003	45 + DC	NA	9,862	2,866	264

A minor difficulty with this form of reporting is it is not clear that all disease incidences are correctly classified. In Russia, for example, WNV may have been occurring for up to three years prior to the determination of an epidemic in 1999. The basis for this judgment is the distribution of encephalitis cases from 1996 – 1998 in the areas where the disease broke out in 1999. There appeared to be anomalous cases of encephalitis over that time period, anomalous in that most encephalitis prior to WNV occurred in these areas in winter, whereas additional cases occurred in the summers of 1996-1998 (Platonov, 1999). Similarly, it has been suggested that many cases of meningitis or encephalitis classified as of unknown or suspect origins in the US may have been due to WNV.

In addition, since only a small percentage of WNV infections are symptomatic to any degree, the true infection rate is not realized. The incidence of infection is thus clearly underreported. This makes it more difficult to ascertain focal points of the disease, and may lead to less productive control strategies. However, the accurate data needed to determine true infection rates in any area requires a serological survey, which can be difficult and expensive to perform.

A complication of serosurveys for WNV is that even ill people may take time to seroconvert. Therefore, if a serosurvey is taken while mosquitoes are still infecting people, or immediately after new infections have occurred, the blood tests may not reveal all of the cases that will eventually develop (Campbell et al., 2002). Waiting until after all infected people have seroconverted can lead to undercounting of cases in an area, however, due to the mobility that characterizes modern society. This is especially true for resort or other areas frequented by transient populations.

Another complication of serosurveys for WNV is the potential for false positives. The most efficient test for WNV in blood or cerebrospinal fluid has been said to be the detection of IgM antibody to WNV, using enzyme-linked immunoabsorbent assay (ELISA) antibody-capture. However, tests for WNV, even those using indirect immunofluorescence or hemagglutination inhibition, can produce false positives for people vaccinated for other flaviviruses, those infected with other flaviviruses, and people previously infected by WNV (Petersen and Marfin, 2002). Therefore, it has been advised to use cross-neutralization tests with closely-related viruses (Focus Technologies, undated).

### **3. WNV Serological Surveys**

Most citations of human serosurveys for WNV conducted in the US are from two initial efforts in New York City. One was over a relatively small area in Douglaston, Queens, in 1999, which seemed to be the epicenter of the initial outbreak (Motashari et al, 2001a). The second set of results, for Staten Island in 2000, are usually separated out from a three-area study for the New York metropolitan region (Motashari et al., 2001b). These two studies are often used as basis for calculating WNV incidence for exposed populations.

Several other serosurveys will be discussed below. These include two additional regions discussed in Motashari et al. (2001b), being Suffolk County (Graham and Harper, 2004) and Connecticut (Hadler et al., 2001; McCarthy et al., 2001). Other extensive efforts occurred in Cuyahoga County, Ohio, in 2002 (Alan and Mandalakas, undated), and in Ontario in 2003 (Elliott et al., 2003). An interesting study, unlike these general exposure studies, was conducted on exposed turkey farm workers in Wisconsin in 2003 (Glaser et al., 2003). In addition, two seroprevalence studies have been reviewed, one for Romania in 1996 (Han et al., 1999), and the other in Jordan in 1998 (Batieh et al., 2000). Finally, the presence of WNV in blood bank supplies is reviewed (Montgomery, 2004; Biggerstaff and Petersen, 2002).

#### **3.1 Motashari et al., 2001a (Douglaston, NY, 1999)**

The survey was carried out in early October, 1999, approximately six weeks after the peak of encephalitis cases diagnosed in New York City. The study site was in a 7.3 km<sup>2</sup> area in Queens, New York, surrounding a five km<sup>2</sup> area believed to be the center of the most intense virus activity. An experimental design by the World Health Organization (WHO) was followed. Households were randomly identified based on census tract data, and residents in the households age five and up were invited to donate blood.

Six hundred sixty-seven individuals from 459 households were sampled. 19 were seropositive. Interviews of the respondents found that six of the 19 seropositive individuals (32 percent) reported recent febrile illness, compared to 70 of the 648 non-seropositive individuals (11 percent). The infection rate was determined to be 2.6 percent, with a 95 percent confidence interval of 1.2 to 4.1 percent. There were nine reported hospitalizations of people from in this

area. Therefore, the data were extrapolated, assuming constant ratios. The 59 hospitalizations from WNV in New York City in 1999 probably resulted from 8,200 infections, within a conservative range of 3,500 to 13,000, with some 1,700 of the infections resulting in some detectable illness. This suggests that there may be approximately 140 undiagnosed infections for every hospitalization.

### **3.2 Motashari et al., 2001b (Staten Island, NY, 2001)**

In 2000, the second year of the WNV outbreak in the New York City region, there were 14 cases, including one death. To better estimate the public health impact of the outbreaks, household-based seroprevalence surveys were conducted on Staten Island, New York, in Suffolk County, New York, and in Connecticut. In 2000, Staten Island was the locus of WNV in New York City. Most reports on the serosurvey work tend to focus on the Staten Island data.

CDC and the New York City Department of Health (NYCDOH) conducted a door-to-door WNV survey on Staten Island in October. The CDC analysis of 871 blood samples, all from persons 12 years or older, found that four tested positive for an antibody against WNV. This is considered indicative of a recent infection, likely during the summer of 2000. This rate equals an infection rate of 0.46 percent of the tested population, with a 95 percent confidence interval of 0.18 to 1.17 percent. The data suggested that 1,574 residents of Staten Island were infected by WNV in 2000. There were nine hospitalizations for WNV among Staten Island residents, suggesting that 156 undiagnosed cases may have occurred for every hospitalization.

### **3.3 Motashari et al., 2001b; Graham and Harper, 2004 (Suffolk County, NY, 2001)**

In November of 2000, the Division of Public Health of the Suffolk County Department of Health Services (SCDHS) conducted a WNV serosurvey in conjunction with the New York State Department of Health (NYSDOH) and CDC, near the Suffolk County disease epicenter, Babylon, NY. The epicenter was determined due to the high number of dead crows and coincidental detections of virus in mosquitoes. However, there were no known human cases in the vicinity of these indicators of WNV.

Blood was collected from 836 residents from 703 randomized households over a 27 mi<sup>2</sup> study area of Babylon. The blood was tested for WNV-antibody activity by NYSDOH, with

confirmatory testing completed by CDC. One positive result was determined, confirming the presence of WNV infection in humans in Suffolk County prior to any hospitalizations. The computed infection rate was 0.12 percent, with a 95 percent confidence interval of 0.01 to 0.67 percent.

### **3.4 Motashari et al., 2001b; Hadler et al., 2001; McCarthy et al., 2001 (Fairfield County, CT, 2001)**

In 1999, Connecticut was one of three states in which WNV actively circulated prior to its recognition. In 2000, prospective surveillance was established, including monitoring bird deaths, testing dead crows, trapping and testing mosquitoes, testing horses and hospitalized humans with neurological illness, and conducting a human seroprevalence survey. WNV was first detected in a dead crow found on July 5 in Fairfield County. Ultimately, 1,095 dead crows, 14 mosquito pools, seven horses, and one mildly symptomatic person were documented with WNV infection. None of 86 hospitalized persons with neurological illness, meningitis or encephalitis, and no-one of 731 individuals tested in the seroprevalence survey, were infected. These results were interpreted by the disease transmission experts as suggesting that the infection rate, in an area of intense viral activity, was 0 percent, because of the lack of detections in the serosurvey. The presence of one human requiring hospitalization from West Nile fever was not factored into this quantitative analysis.

### **3.5 Allan and Mandalakas, undated (Cuyahoga County, OH, 2002)**

As Table 2-1 showed, WNV exploded across the country in 2002. In Ohio there were 441 cases, and 31 fatalities. Cuyahoga County, with a population of 1.4 million, had 219 cases (144 meningoencephalitis cases, and 75 WNV fever cases). The sampling design for the serosurvey following the epidemic included stratifying the County into three risk categories, based on hospitalization and mosquito data. Children less than five years old, pregnant women, and those taking anticoagulants were excluded. Participation was not strictly voluntary, as with the other surveys cited above, as it was encouraged through the use of \$10 gift certificates. 1,209 volunteers were recruited for the study, and 96 positive results were found. However, after extensive quality control measures, using plaque reduction neutralization (PRNT), many of the positives were found to be false positives of one sort or another. For example, old St. Louis

Encephalitis (SLE) or other unidentified flavivirus, vaccine reactions, or actual true negative results can illicit false positives. Using PRNT reduced the number of WNV positives to 34 or 35, comprised of 27 confirmations, seven cases noted as “probable,” and one where SLE or WNV could not be clearly differentiated. This reduced the initial estimate of County-wide seroprevalence from 4.0 to 6.4 percent to an unadjusted rate of 2.8 percent. The 2.8 percent rate was statistically adjusted to a 1.9 percent infection rate to accurately reflect demographics. This infection rate implied that 24,764 residents had been infected in 2002, and that there were 113 unrecorded infections for every observed infection. The number of meningoencephalitis cases meant there was one, serious “hospitalization” for every 171 “non-hospitalized” cases. It should be noted that some West Nile fever cases were actually treated in institutional settings, although they were not classified as serious “hospitalizations.”

Overall, these infection rates and serious illness incidence statistics are similar to those found for New York City in 1999 and 2000.

### **3.6 Elliott et al., 2003 (South Oakville, Ontario, Canada, 2003)**

Ontario experienced an outbreak of WNV in 2002, with 319 confirmed cases and 86 probable cases. These cases were tightly clustered near Peel, Halton, and Toronto along Lake Ontario.

The following spring (2003), a serosurvey was conducted in two postal codes near the McMaster University Institute of Environment and Health in the region where most cases occurred. 1,505 out of 30,467 adult residents were sampled. 46 were positive for WNV by ELISA; the Winnipeg National Microbiological Laboratory confirmed all the positives using PRNT. The infection rate was determined to be 3.1 percent, with a 95 percent confidence interval of 2.2 to 4.6 percent infected. An analysis based on the age of the participants compared to 2001 census data resulted in no adjustments to the estimated infection rate of 3.1 percent. This suggests that 944 individuals in the postal codes were infected in 2002, 95 percent confidence interval of 670 to 1,219. In 2002, six residents were hospitalized with WNV-related encephalitis, and five were diagnosed with WNV-related meningitis. Of the five meningitis cases, one was hospitalized. This implies there may have been 157 infections for every person made seriously ill.



### **3.7 Glaser et al., 2003 (unidentified location, Wisconsin, 2002)**

Wisconsin public health officials were notified of two cases of febrile illness in workers at a commercial turkey breeder farm (“Farm A”). The resulting investigation tested workers at Farm A, at five other breeder farms located within ten miles of Farm A, workers at non-breeder turkey farms, and residents who lived within the ten-mile radius. Seroprevalence rates ranged from:

- 55 percent for workers who worked exclusively at Farm A (six of 11)
- 25 percent for Farm A workers who also worked at other company turkey farms (two of eight)
- five percent for workers at other breeder farms
- no positive results for non-breeder farm workers and local residents.

CDC concluded that non-mosquito transmission, as seemed to be the situation here, might be effective if exposure was great enough. Worker protection policies were adopted to minimize risks.

### **3.8 Han et al., 1999 (Bucharest, Romania, 1996)**

In 1996, 393 cases of WNV were confirmed in southeast Romania; 286 occurred in Bucharest. A serosurvey was conducted from October 2 to October 4 in Bucharest. 959 people were tested, and 39 were positive. Quality assurance associated with the report suggested that the data were biased because the samples were collected only 12 weeks after the epidemic began, meaning some infected people might not yet have seroconverted. The computed infection rate was four percent, meaning more than 90,000 Bucharest residents might have been infected. The ratio of infections to confirmed cases resulting in hospitalizations, reported as serious illnesses in the above studies, appears to have been greater than 300:1.

### **3.9 Batieh et al. 2000 (Hashimiah, Jordan, 1998)**

The presence of a wastewater treatment plant and its associated open-air channels was the impetus for this study in a town of 30,000 inhabitants. 261 of 501 patients at a health center

agreed to be tested for three arboviruses, including WNV. 21 were positive for WNV – a rate of 8.0 percent. No participants showed any evidence of acute infection.

### **3.10 Montgomery, 2004; Biggerstaff and Petersen, 2002 (WNV in the Blood Supply)**

Biggerstaff and Petersen estimated the risk of transmission of WNV through the blood supply from an infected donor to an uninfected recipient at approximately 1.8 cases per 10,000 donations. This estimate was based on the infection rate computed for Douglaston Queens and the estimated viremia load for infected people. A maximum risk, in late August, was 2.7 per 10,000 donations.

In 2002, WNV was detected in certain blood banks. Montgomery reported on steps taken to address the risk by testing minipools of six to 18 donations, as a screening tool. In 2003, 1,027 donations from a total of 6.2 million donations were positive for WNV, yielding a 0.02 percent infection rate. As forecast by Biggerstaff and Petersen, the risk of such donations was greatest in mid- to late-August.

There were 23 investigations of suspected transfusion-induced cases. Six were confirmed, 10 were rejected, two were inconclusive, and five investigations remained open as of February, 2004.

Although blood donations are a biased sample of the general population, infection determinations in the blood supply seemed to track the general location, by county, and the overall timing of other measures of human infection, such as hospitalizations. In fact, there are suggestions that positive results in blood banks precede other evidence of human infection by one to 13 days, on average.

## References

- Batieh, A., EK Saliba, R. Graham, E. Mohareb, Y. Hijazi, and P. Wijeyaratne. 2000. Seroprevalence of West Nile, Rift Valley, and Sandfly arboviruses in Hashimiah, Jordan. *Emerging Infectious Diseases* 6(4):358-362.
- Biggerstaff, BJ, and LR Petersen. 2002. Estimated risk of West Nile virus transmission through blood transfusion during an epidemic in Queens, New York City. *Transfusion* 42:1019-1026.
- CDC. 2003. *Epidemic/Epizootic West Nile Virus in the United States: Guidelines for Surveillance, Prevention, and Control*. 3<sup>rd</sup> Revision. Centers for Disease Control and Prevention, Fort Collins, CO. 75 pp.
- Graham, DG, and BL Harper. 2004. The potential threat of mosquito-borne disease in Suffolk County. *The Suffolk County Medical Society-Suffolk County Academy of Medicine*, July. Public Health Page.
- Elliott, SJ, M. Loeb, J. Eyles, and D. Harrington. 2003. *Results of a West Nile Seroprevalence Survey, South Oakville, Ontario, 2003*. McMaster Institute of Environmental Health, McMaster University, Hamilton, Ontario. 37 pp.
- Focus Technologies. Undated. *Flavivirus (West Nile) ELISA IgG*. Data Sheet. Focus Technologies, Cypress, CA. 4 pp.
- Glaser, LC, MV Wegner, JP Davis, ML Bunning, AA Marfin, GL Campbell, B. Bernard, SW Lenhart, and MJ Sotir. 2003. West Nile Virus infection among turkey breeder farm workers – Wisconsin, 2002. *Morbidity and Mortality Weekly* 52(42):1017-1019.
- Hadler, J. R. Nelson, T. McCarthy, T. Andreadis, MJ Lis, R. French, W. Beckwith, D. Mayo, G. Archambault, and M. Cartter. 2001. West Nile virus surveillance in Connecticut in 2000: an intense epizootic without high risk for severe human disease. *Emerging Infectious Diseases* 7(4):636-642.
- Han, LL, F. Popovici, JP Alexander, Jr., V. Laurentia, LA Tengelsen, C. Cernescu, HE Gary, Jr., N. Ion-Nedelcu, GL Campbell, and TF Tsai. Risk factors for West Nile virus infection and meningoencephalitis, Romania, 1996. *The Journal of Infectious Diseases* 179:230-233.
- Hayes, CG. 2001. West Nile Virus: Uganda, 1937, to New York City, 1999. pp. 25-37. In: White, DJ, and DL Morse (eds.). *West Nile Virus: Detection, Surveillance, and Control*. Annals of the New York Academy of Science, V. 951. New York, NY. 374 pp.
- Huhn, GD, JJ Sejvar, SP Montgomery, and MS Dworkin. 2003. West Nile Virus in the United States: an update on an emerging infectious disease. *American Family Physician* 68(4):653-660.

- Lanciotti, RS, JT Roehrig, V. Deubel, J. Smith, M. Parker, K. Steele, B. Crise, KE Volpe, MB Crabtree, JH Scherret, RA Hall, JS McKenzie, CB Cropp, B. Panigrahy, E. Ostlund, B. Scmitt, M. Malkinson, C. Banet, J. Weissman, N. Komar, HM Savage, W. Stone, T. McNamara, and DJ Gubler. 1999. Origin of the West Nile Virus responsible for an outbreak of encephalitis in the northeastern United States. *Science* 286:2333-2337.
- McCarthy, TA, JL Hadler, K. Julian, SJ Walsh, BJ Biggerstaff, SR Hinten, C. Baisley, A. Iton, T. Brennan, RS Nelson, G. Archambault, AA Marfin, and LR Petersen. 2001. West Nile serosurvey and assessment of personal prevention efforts in an area with intense epizootic activity: Connecticut, 2000. pp. 307-316. In: White, DJ, and DL Morse (eds.). *West Nile Virus: Detection, Surveillance, and Control*. Annals of the New York Academy of Science, V. 951. New York, NY. 374 pp.
- Montgomery, SP. 2004. Screening of the national blood supply for West Nile Virus – United States, 2003. Power Point presentation. *Fifth National Conference on West Nile Virus in the United States*, Denver, CO. February 3-5, 2004.
- Motashari, F., I Poshni, EM Layton, D. Graham, C. Bradley, M. Kacacia, S. Wong, C. Franchell, D. Morse, B. Wallace, P. Smith, E. Bresnitz, C. Baisley, A. Iton, G. Archambault, D. Mayo, J. Hadler, and EIS officers, CDC. 2001b. Serosurveys for West Nile infection – New York and Connecticut counties, 2000. *Morbidity and Mortality Weekly* 50(3):37-39.
- Motashari, F., ML Bunning, PT Kitsutani, DA Singer, D. Nash, MJ Cooper, N. Katz, KA Liljebjelke, BJ Biggerstaff, AD Fine, MC Layton, SM Mullin, AJ Johnson, DA Martin, EB Hayes, and GL Campbell
- Nash, D., F. Mostashari, A. Fine, J. Miller, D. O’Leary, K. Murray, A. Huang, A. Rosenberg, A. Greenberg, M. Sherman, S. Wong, and M. Layton (for others from the West Nile Outbreak Response Working Group). 2001. The outbreak of West Nile Virus infection in the New York City area in 1999. *New England Journal of Medicine* 344(24):1807-1814.
- Petersen, LR, and AA Marfin. West Nile Virus: a primer for the clinician. *Annals of Internal Medicine*. 137:E173-E179.
- Platonov, AE. 2001. West Nile Virus in Russia, 1999-2001. Were we ready? Are we ready? pp. 102-116. In: White, DJ, and DL Morse (eds.). *West Nile Virus: Detection, Surveillance, and Control*. Annals of the New York Academy of Science, V. 951. New York, NY. 374 pp.
- WHO. 2003. *Manual of Basic Techniques for a Health Laboratory*. 2<sup>nd</sup> Ed. World Health Organization, Geneva, Switzerland. 384 pp.
- Zielinski-Gutierrez, EC. 2004. *Clinician Briefing: West Nile Virus: June 29, 2004*. Clinician Outreach and Communication Activity, Centers for Disease Control and Prevention. Unpaginated.