3.3. Mosquito-borne Disease

3.3.1. Mosquito-borne Disease Overview

Certain mosquito-borne diseases are important in a historical context, or because they pose a potential ongoing health threat. The most important and common of these diseases will be briefly discussed.

3.3.1.1. Malaria

Malaria is a parasitic disease that can cause an array of symptoms and complications ranging from flu-like symptoms to anemia, jaundice, kidney failure, coma, and death (CDC, 2000). Malaria is spread by one of four species of protozoa, *Plasmodium falciparum*, *P. malariae*, *P. ovale*, and *P. vivax that* infect mosquitoes and are transmitted to people. *P. falciparum* is the highly fatal species found in tropical areas, and *P. vivax* is the species that infects temperate areas, including the US (Eldridge and Edman, 2000).

Malaria is found in over 100 countries, and more than 40 percent of the world's population is at risk from this disease. The World Health Organization (WHO) estimates that there are 300 to 500 million cases of malaria each year, with more than one million deaths (CDC, 2000). Most of those who die from malaria are young children. For those surviving childhood malaria, a malaria incidence as an adult is unlikely to be fatal (Spielman and D'Antonio, 2001).

Ninety percent of malaria cases occur in sub-Saharan Africa. The disease is also found in Central and South America, the Middle East, Southeast Asia, and Oceania. Areas of limited risk include North America, Europe, and Asia. Approximately 1,200 cases of malaria are diagnosed in the United States each year (CDC, 2000).

3.3.1.2. Yellow Fever

Yellow fever, transmitted primarily between non-human primates and mosquitoes, is caused by a zoonotic virus (that humans share with animals). When a human becomes the host, the disease is considered urban yellow fever (CDC, 2003a).

The yellow fever virus enters the host and incubates from three to six days. After the incubation period, the disease develops in two phases, and may be transmitted by mosquitoes to other

humans. The first, which lasts three to four days, may result in fever, muscle pain, headache, shivers, loss of appetite, and nausea (WHO, 2001).

In 85 percent of those infected, symptoms disappear after the first phase. The remaining 15 percent, however, enter a second phase within 24 hours of the first. The second phase is characterized by fever, jaundice, abdominal pain, and bleeding from the mouth, nose, eyes, and/or stomach, and kidney failure. The disease is fatal for approximately half of those who experience the second phase of the virus (WHO, 2001).

Notable historical outbreaks in the US seem to have been caused by ships carrying barrels of water that contained larvae of the primary vector, *Aedes aeygpti.* Infected sailors leaving the ships in port then transmitted the disease to local populations of *Ae. aeygpti,* a mosquito that is common in the southern US. Once the disease was present in local mosquitoes human epidemic followed. Outbreaks in northern cities such as Philadelphia and Boston depended on summer populations of *Ae. aeygpti,* and so these epidemics ceased when colder weather slowed the mosquitoes (Spielman and D'Antonio, 2001).

WHO estimates that approximately 200,000 cases occur per year, resulting in about 30,000 deaths. As is the case with malaria, the disease is primarily found in sub-Saharan Africa and tropical regions of South America. However, it has occurred in Europe and North America (WHO, 2001).

The disease places infants and children at greatest risk for infection and there is no treatment for the virus. Afflicted people are often treated for the side effects of the disease, such as dehydration and pain (WHO, 2001).

3.3.1.3. Dengue Fever

Dengue fever is a virus nicknamed "bone break fever," which can cause sudden and severe incapacitating illness including headache, high fever, and extreme muscle and joint pain. It can lead to Dengue hemorrhagic fever (DHF), which is frequently fatal (Budiansky, 2002).

The disease is found in approximately 100 countries in tropical and subtropical regions throughout the world and places about 2.5 billion people at risk. WHO estimates approximately 50 million cases of Dengue infection occur worldwide annually. The geographic area affected

by Dengue is increasing with time, and, thus, the incidence of Dengue is increasing each year (WHO, 2002).

The disease is caused by one of four serotypes of *Flavivirus* (DEN-1, DEN-2, DEN-3, and DEN-4), and is transmitted primarily by the *Ae. aegypti* mosquito. Infection from one serotype offers lifetime immunity; however, there is no cross-immunity between serotypes (WHO, 2002). *Aedes albopictus* is the primary vector of the disease in Asia. *Ae. albopictus* was recently introduced into the US (it breeds in discarded tires, and so was shipped from Asia to Texas in a load of waste tires) and it was feared that this mosquito would allow for Dengue transmission to occur in the US. *Ae. albopictus* have been caught in traps in Nassau County, but not in Suffolk County, yet.

Once in the bloodstream, the virus circulates for two to seven days, causing fever and flu-like symptoms in the host. DHF, a complication of Dengue fever, includes hemorrhaging, high fever, enlargement of the liver, and, frequently, failure of the circulatory system. The afflicted either die from shock within 12 to 24 hours, or recover with proper medical treatment (Spielman and D'Antonio, 2001).

The fatality rate is approximately two percent for Dengue fever and over 20 percent for DHF. Treatment can reduce the fatality rate for DHF to approximately one percent (WHO, 2002).

During the two to seven days that the virus circulates throughout the bloodstream, a mosquito feeding on the host may contract the disease. The disease must develop in the mosquito for eight to ten days before the mosquito can transmit the disease. Once infected, the mosquito can transmit the disease for the rest of its life. In addition, the mosquito can pass the virus to its offspring through eggs (CDC, 2003b).

There is no specific medical treatment or vaccine for Dengue fever. Prevention is crucial to fighting the disease. Combating the vector mosquito, *Ae. aegypti*, has been determined to be the most effective means of prevention (CDC, 2003b).

3.3.1.4. Filariasis

Filariasis is caused by the Filaria worm and causes inflammation of the lymph nodes occasionally leading to elephantiasis, a build up of lymphatic fluid which causes swelling in parts of the body, particularly the arms, legs, and genitals (GAELF, 2004).

According to CDC, lymphatic filariasis is a leading cause of permanent and long-term disability worldwide. People with the disease can suffer pain, disfigurement, and sexual disability. The disease affects over 120 million people, in over 80 countries. 1.1 billion people are at risk of contracting the disease. Filariasis is found in Asia, Africa, Oceania, and Central and South America, including parts of Caribbean (CDC, 2003c).

Mosquitoes act as vectors for filariasis when they ingest microfilariae, newly born worms, during a bloodmeal, which then partly develop in the mosquito before being passed to humans. The microfilariae complete development in the human body (GAELF, 2004).

Male and female worms settle in lymph nodes, where they live and reproduce. Eggs are fertilized, and hatch within the female worm's uterus. Microfilariae are then distributed in lymphatic circulation and pass into bloodstream, where they can be ingested by mosquitoes and passed to another host (GAELF, 2004).

The limited function of affected lymph system makes it difficult for the body to fight germs, which leads to bacterial infections in the skin. The preferred course of treatment is to clean affected areas and use anti-bacterial treatments on wounds. In some cases, surgery is necessary to alleviate problems associated with elephantiasis (GAELF, 2004).

Prevention of the disease can be achieved through the use of drugs (such as mefloquine, which is 90 percent effective) and avoiding mosquito bites (GAELF, 2004).

3.3.1.5. Encephalitis

Encephalitis is due to a virus that causes an inflammation of the brain, which may result in headache and fever, and can progress to paralysis, seizures, coma, and death.

There are many types of encephalitis. The major mosquito transmitted strains are:

- Eastern equine encephalitis (EEE)
- Japanese encephalitis
- LaCrosse encephalitis
- Murray Valley encephalitis
- St. Louis encephalitis

- Venezuelan equine encephalitis
- Western equine encephalitis
- West Nile encephalitis (WNV)

Encephalitis is found in the temperate zones throughout the world. The encephalitides found in North America are:

- St. Louis encephalitis
- EEE
- Western equine encephalitis
- La Crosse encephalitis
- WNV

(CDC, 2003d).

Encephalitis is the most widely transmitted mosquito-borne disease in the US. Between 1964 and 2000, the average number of cases in the US per year was:

- 121 cases of St. Louis encephalitis per year
- 75 cases of La Crosse encephalitis per year
- 18 cases of Western equine encephalitis per year
- 5 cases of EEE per year.

The number of cases follows a somewhat cyclical pattern with highs and lows generally occurring about the same time for the four diseases (CDC, 2003d).

Until the advent of WNV, St. Louis encephalitis was the most common form of mosquito-borne encephalitis in the United States. Table 3-11 shows the distribution of major outbreaks over time (CDC, 2003d).

Table 3-11. Major outbreaks of St. Louis encephalitis

Year	Cases
1964	470
1966	325
1975	1,967
1976	379
1977	161
1990	247

Japanese encephalitis, though typically mild, can cause paralysis, coma, and death. The fatality rate is 30 percent, with 30 percent of survivors suffering permanent neurological damage (CDC, 2003d).

La Crosse encephalitis and St. Louis encephalitis are similar in that they are rarely fatal, with the infection producing mild symptoms that can, however, progress to seizures and coma. WEE can range from mild fever and flu-like symptoms to severe encephalitis and death (CDC, 2003d).

3.3.2. West Nile Virus

3.3.2.1. General Information on WNV

WNV was first isolated from a woman in the West Nile district of Uganda in 1937 (Marra et al., 2004). Although WNV is new to the western hemisphere, it is widely distributed in Africa (Malkinson and Banet, 2002; Hurlbut and Taylor, 1955; Taylor et al., 1956; McIntosh et al., 1968; Work et al., 1953), West Asia (Platonov et al., 2001), and the Middle East (Malkinson et al., 2002), and has been found in southeastern Europe (Tsai et al., 1998; Ernek et al., 1977). Since 1999, from its introduction in the general area of LaGuardia Airport in Queens, New York, WNV has spread throughout the US and North America (Marra et al., 2004). The New World strain is most closely related to a WNV isolated from a dead goose in Israel (Lanciotti et al., 1999; Bin et al., 2001) that may have been infected by white storks (*Ciconia ciconia*) migrating from Europe (Malkinson et al., 2002; Malkinson et al., 2001).

The transmission cycle of WNV requires mosquitoes, the vectors for the disease. At least 40 species of mosquitoes have tested positive for the virus in North America (Cornell, 2004). The major species associated with the spread of WNV belong to the *Culex* species; cases are linked to *Cx. pipiens, Cx. quinquefascatus,* and *Cx. tarsalis* (CDC, 2003e). Mosquitoes feed seeking

essential nutrients for egg production. If an infected host is fed on, the mosquito can become a carrier for the virus, and can transfer the virus when it feeds again. Birds tend to develop measurable virus levels (viremia) shortly after being bitten by infected mosquitoes; therefore, they possess the ability to pass the virus onto other mosquitoes if bitten again. Such species are known as "reservoir hosts" because they can pass the virus back to mosquito vectors. Mosquitoes may also infect other animals, including mammals, which are classified as "dead end" hosts, because they do not support a high enough viremia level to successfully pass the virus back to mosquitoes when bitten (Cashin Associates, 2005a).

Migrating birds play a crucial role in the ecology of WNV in Europe and Africa (Malkinson and Banet, 2002). The likelihood of the potential spread of WNV throughout temperate and tropical regions of the Western Hemisphere by migratory birds depends on the pattern of virus occurrence in light of known migratory routes, and a comparison to WNV dissemination in the Old World that involves "a set of conditions including infectious avian host, numerous ornithophilic mosquito vectors, and cross-species transmission to a numerous avian amplifying host" (Rappole et al., 2000).

In North America, however, the disease has not leap-frogged over long distances, has not been confined to a north-south movement from the New York epicenter (as might be expected in light of the flight patterns of migratory birds that visit the northeast US), and prolonged viremia has been noted only in the relatively non-migratory house sparrow (*Passer domesticus*). Migratory birds thus appear unlikely to be the agents for movement of WNV in its new environment. Alternatives that might account for a steady movement across the continent include less mobile birds such as crows and house sparrows (because of high abundance, seroprevalence, and biological competence [Komar et al., 2000]), arthropod vectors other than mosquitoes (e.g., infected ticks that could be transported by migrants), and "displaced" mosquitoes carried long distances by winds or vehicles. In 2003, this led to a prediction that the virus will "eventually enter the mainland Neotropics," and spread throughout the region (Rappole and Hubálek, 2003). Infected birds have been found in Mexico (Estrada-Franco et al., 2003), the Dominican Republic (Komar et al., 2003b), Jamaica (Dupuis et al., 2003), Guadalupe (Quirin et al., 2004), El Salvador (Pro-MED Mail, 2004a), Puerto Rico (Pro-MED Mail, 2004b), Belize (Pro-MED Mail, 2004c), Trinidad, and Cuba (Cornell, 2005) (as compiled by Audubon, 2005). A report from

Guadeloupe in 2003 (Lefrancois et al., 2005) linked seropositive equines and chickens to mangroves containing many species of wild birds and mosquitoes.

WNV causes several forms of illness in humans, which must run their course as there is no effective treatment for the disease. Symptoms can be relieved through various treatments appropriate for flu and flu-like effects (e.g., standard medication for headache, fever, body aches, etc.). 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 up to several weeks. West Nile fever does not appear to cause any long-term health effects and most patients recover fully with no sequelae (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. 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 (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 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 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 person as being severely ill from WNV (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, 2003e).

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). A minor difficulty with this form of reporting is that 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 distribution of encephalitis cases from 1996 to 1998, in the areas where the disease broke out in 1999 appeared to include anomalous cases over that time period. They were anomalous in that most encephalitis prior to WNV occurred in these areas in winter, whereas additional cases occurred in the summers of 1996 to 1998, which were not readily explicable (Platonov, 2001). Similarly, it has been suggested that many cases of meningitis or encephalitis in the US that are classified as of unknown or suspect origins may be due to WNV.

Nonetheless, ArboNET has collected reports on WNV since its introduction in 1999. A summary of the data is included in Table 3-12.

Table 3-12. Human Cases of WNV in the US, 1999-2004

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	4,008	2,354	284
2003	45 + DC	NA	9,862	2,866	264
2004	48 + DC	NA	2,539		100

(collected from various annual CDC reports)

3.3.2.2. Serosurveys for WNV

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 because so many infected people are either asymptomatic, or symptoms which occur are confused with those associated with other, more common ailments such as allergies, colds, or influenza. Thus, a blood test is often the only means of determining if

infection has actually occurred. Blood tests of a population in an area are called serologic surveys. Serologic surveys 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 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 resorts or other areas frequented by transient populations. Waiting to conduct the survey also reduces its value for human health management.

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 immunofluoresence 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).

Most citations of the results 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 (Mostashari et al., 2001a). The second set of results, for Staten Island in 2000, is usually separated out from a three-area study for the New York metropolitan region (Mostashari 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 Mostashari 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; Mandalakas et al., 2005), and in Ontario in 2003 (Elliott et al., 2003; Loeb et al., 2005).

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^2 area in Queens, New York, surrounding a five km^2 area believed to be the center of the most intense virus activity. An experimental design by 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 (Mostashari et al., 2001a).

667 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). This is the source of the estimate that 20 percent of infected people have only mild effects from the disease, and approximately 80 percent have no symptoms. 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 in this area. Therefore, the data were extrapolated to larger exposed populations, assuming constant ratios. The 59 hospitalizations from WNV in New York City in 1999 were assumed to have resulted from 8,200 infections (ranging from 3,500 to 13,000 infections, given the statistical uncertainties) 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 (Mostashari et al., 2001a).

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, and in Connecticut. In 2000, Staten Island was the locus of WNV in New York City (Mostashari et al., 2001b). Most reports on this serosurvey work tend to focus on the Staten Island data (because the Connecticut and Suffolk County surveys did not find large numbers of infections in the exposed population).

CDC and the New York City Department of Health 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 was 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 (Mostashari et al., 2001b).Suffolk County, NY, 2001

In November, 2000, the Division of Public Health of SCDHS conducted a WNV serosurvey in conjunction with NYSDOH and CDC, near the Suffolk County disease epicenter in Babylon. The epicenter was identified by the high number of dead crows and co-incidental detections of virus in mosquitoes. However, there were no known human cases in the vicinity of these indicators of WNV (Graham and Harper, 2004).

Blood was collected from 836 residents from 703 randomized households over a 27 mi^2 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, but without any hospitalizations. The computed infection rate was 0.12 percent, with a 95 percent confidence interval of 0.01 to 0.67 percent (Mostashari et al., 2001b; Graham and Harper, 2004).

Fairfield County, CT, 2001

In 1999, Connecticut was one of three states in which WNV actively circulated prior to being identified. 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 found to be 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. That one person required hospitalization from West Nile fever during this time

period in the study area was not factored into this quantitative analysis (Mostashari et al., 2001b; Hadler et al., 2001; McCarthy et al., 2001).

Cuyahoga County, OH, 2002

As Table 3-12 showed (above), WNV exploded across the country in 2002. In Ohio, there were 441 cases, and 31 fatalities. Cuyohoga 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 surveys cited above, as it was encouraged through the use of $10 gift certificates. 1,209 participants 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 or other unidentified flaviviruses, 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 St. Louis encephalitis or WNV could not be clearly differentiated. This reduced the initial estimate of County-wide sero-prevalence 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 when demographics were statistically adjusted for. 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 (Allan and Mandalakas, undated; Mandalakas et al., 2005).

Overall, these infection rates and serious illness incidence statistics are similar to those found for New York City in 1999, and the serious illness incidence rate is similar to that for Staten Island in 2000.

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 (Elliott et al., 2003).

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. A demographic 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, with a 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 (Elliott et al., 2003; Loeb et al., 2005).

3.3.2.3. WNV Serosurvey Summary

The serosurveys discussed in detail above have been summarized in Table 3-13.

Table 3-13. Serosurvey Results

Year	Location	Infection Rate (percent)	Undiagnosed Infections per Case
1999	Douglaston	2.6	140
2000	Staten Island	0.5	156
2000	Babylon	0.2	NA
2000	Connecticut	0	NA
2002	Cuyahoga County	1.9	171
2002	Ontario	3.1	157

In areas where there was no significant mosquito control (Douglaston, Cuyahoga County, and Ontario), infection rates appear to be between two and three percent. The data on symptomatic illness appeared to show that approximately 20 percent of those infected might experience measurable impacts (CDC, 2004). However, in approximately one out of every 150 cases, serious disease can occur, such as encephalitis or meningitis requiring hospitalization (see Table

3-13). Nationwide, fatality rates of those with the most severe symptoms have ranged from a low of six percent in 2000 to a high of 14 percent in 2001. The most recent data suggests the fatality rate for these individuals is slightly less than 10 percent (see Table 3-12). This suggests that one out of every 1500 cases may result in a fatality in any given year.

3.3.2.4. Ecological Impacts from WNV

3.3.2.4.1. Mammals

A variety of mammals and domestic animals are also experiencing the effects of WNV. Horses had highly visible mortalities from the disease, and are discussed separately, below. CDC (2004) has reported infections in 29 species of mammals as of 2004, including:

- chipmunks
- skunks
- squirrels
- wolves
- sheep
- goats
- bats
- seals and some other marine mammals
- monkeys
- domestic cats and dogs

Most mammals are considered dead-end hosts, whereby the infected host species are unable to pass the disease back to mosquitoes, and so are not considered part of the transmission cycle. In addition, the majority of mammals are able to produce resistance to the virus; consequently mammal deaths in previously exposed populations from WNV are rare. One notable exception is horses, which have experienced significant mortality rates.

The majority of mammalian research has focused on horse infections and deaths. Impacts from WNV to non-human primates, and domestic dogs and cats have been documented. In rodents,

WNV has been identified in several gray squirrels in Illinois and reports of mass die-offs in the gray squirrel populations were observed in Wyoming (Noler, 2002). Wild raccoons and bats have also seen significant infection and mortality from WNV. Captive species affected include wolves, reindeer, and camels (Graham, 2003).

Household Pets

Dogs, cats, and other domestic animals have been monitored for WNV due to their close physical relationship with humans. In 1999, testing on 189 dogs in New York City and Nassau County, Long Island, revealed that 5.3 percent had WNV antibodies. None of 12 cats tested had antibodies for WNV. Dogs are most likely considered dead-end hosts for WNV (Komar et al., 2001). In 2002, an eight-year-old Irish Setter-Golden retriever mix died from WNV (Lichtensteiger et al., 2003).

Rodents

Rodents are often carriers of disease in urban areas. They also are commonly used in scientific research as surrogates for humans. Several experiments involving WNV and rodents have been performed. Most studies reveal that rodents are highly susceptible to WNV, with most test subjects developing severe symptoms and mortalities occurring at a high rate (Mashimo et al., 2002; Xiao et al., 2001). However, it has also been suggested, based on limited data, that wild-bred mice may have the ability to develop complete resistance to the virus (Xiao et al., 2001). Squirrels may also be susceptible to the disease, as studies in Illinois appear to show impacts to several species (Noler, 2002). Additional rodents affected by WNV in the US include the eastern chipmunk and the black-tailed prairie dog (Marra et al., 2004).

Non-Human Primates

Non-human primates in captive settings in North America have been studied for WNV impacts. Due to the close genetic relationship between humans and other primates, evidence of infection has been sought in some situations. In the summer of 2002, following an outbreak of WNV in Louisiana, blood samples were taken from 1,692 captive Rhesus monkeys (*Macaca mulatto*), pigtail macaques (*M. nemestrina*), and baboons (*Papio spp.*) housed outdoors at a primate breeding facility. Overall, 36 percent of the captive non-human primates had antibodies for

WNV (Ratterree et al., 2003). However, none of the primates showed clinical illness or neurological disease associated with WNV.

Other Mammals

A variety of other mammals have been found to harbor WNV since 1999. In Illinois, a three-month-old female captive wolf (*Canis lupus*) pup died after being infected with WNV (Lichtensteiger, 2003). In northwest New Jersey, three of 51 black bears tested positive in samples taken in February and March of 2002 (Cornell, 2004). During the same study in New Jersey, seven of 689 white-tailed deer were found to be seropositive for WNV. Other confirmed mammalian deaths in North America from WNV include, skunks, raccoons and domestic cattle (CDC, 2004).

Several documented cases of marine mammals testing positive for WNV have been recorded in recent years. A 12-year-old harbor seal at the New Jersey State Aquarium died as a result of WNV infection in 2002, and two harbor seals from New Mexico's Rio Grande Zoo died from WNV in 2003. Two populations of Florida manatees, 27 captive individuals, and 108 wild individuals, were tested in 2003. In the captive population, one tested positive for the virus and five others had strong indications of previous infections. All wild manatees tested negative for the virus (Keller et al., 2004).

3.3.2.4.2. Birds

As of January 5, 2005 CDC (2005) had received reports of West Nile virus mortality in 284 species of native, exotic, and captive birds (CDC provides a disclaimer indicating it "cannot guarantee that the identities of birds reported to its Arbonet surveillance reporting database are correct"). As of September 4, 2003, USGS (2003) listed over 200 species of birds, within 19 orders and 52 families, as being found positive for WNV in surveillance efforts. USGS uses a similar disclaimer as its list is made up of species provided by CDC and other agencies.

Although little is currently known about the effect of WNV on wild bird populations, what is known suggests that certain species, especially corvids (order Passeriformes, family Corvidae: crows, blue jays, and ravens), may be at risk. A number of references showing crows to be highly susceptible to WNV infection (Kramer and Bernard 2001, McLean et al. 2001, Komar et al., 2003a) are provided by Marra et al. (2004) who discuss the degree to which this may be an

anomaly of sampling since the size and abundance of crows in populated areas may make them more likely to be seen than smaller species that may be equally susceptible.

295 of 671 dead birds collected in New York, New Jersey, and Connecticut during the summer and fall of 1999, and tested by NYSDOH were positive for WNV. 89 percent (262) of the birds confirmed to be infected were American crows (Eidson et al., 2001). Of 17,339 reports of dead birds received from May 1 through November 30 by the NYSDOH, 33 percent (5,697) were crows, but WNV was also isolated from 19 other species:

- seven fish crows (*C. ossifragus)*
- four Chilean flamingos (*Phoenicopterus chilensis*)
- four blue jays (*Cyanocitta cristata*)
- two red-tailed hawks (*Buteo jamaicensis*)
- two mallard ducks (*Anas platyrinchos*)
- rock dove(*Columba livia*)
- belted kingfisher (*Ceryle alcyon*)
- laughing gull (*Larus atricilla*)
- herring gull (*L. argentatus*)
- black-crowned night heron (*Nycticorax nictycorax*)
- sandhill crane (*Grus canadensis*)
- guanay cormorant (*Phalacrocorax bougainvillea*)
- Blyth's tragopan (*Tragopan blythi*)
- bald eagle (*Haleaeetus leucocephalus*)
- American kestrel (*Falco sparverius*)
- Merlin (*F. columbarius*)
- broad-winged hawk (*Buteo platypterus*)
- Coopers hawk (*Accipiter cooperii*)

- American robin (*Turdus migratorius*)

In Connecticut 1,040 dead crows were reported, and isolated WNV from 31 dead crows and a Coopers hawk (Anderson et al., 1999).

In 2000, 63 avian species from New York, representing 30 families and 14 orders, tested positive for WNV (Bernard et al., 2001). A total of 1,263 dead crows were confirmed as virus-positive, from 61 of 62 counties (Eidson et al., 2001). Overall, 71,332 avian mortalities were reported to New York State, of which 17,571 (24.6 percent) were crows. For the US as a whole, over 4,000 bird carcasses tested positive for WNV in 2000 (Turell et al., 2003).

Between 1999 and 2003, the NYSDEC Wildlife Pathology Unit examined over 12,500 avian specimens representing 213 bird species (Chu et al., 2003). Of the 5,950 wild American crows tested, 44 percent were diagnosed with WNV. In addition, 34 percent of dead fish crows (N = 116) and 29 percent of blue jays (N = 1,284) tested positive for the virus during the same time period.

Estimates of population decreases in one year for American crows of approximately two-thirds were made for Stillwater, Oklahoma (Caffrey et al., 2005) and east-central Illinois (Yaramych et al., 2004). Laboratory experiments prior to 2004 with American crows have shown 100 percent death rates from WNV infections. These studies have led researchers to extrapolate that infections in endangered corvid species such as the Hawaiian crow (*Corvus hawaiiensis*), the Mariana crow (*Corvus kubaryi*), and the Florida scrub jay (*Aphelocoma coerulescens*) could lead to significant declines of populations (Yaremych et al., 2004).

Another report (Steele et al., 2000) identified WNV as the cause of "clinical disease, severe pathologic changes, and death" in 27 birds (14 species, eight orders):

- eight Passeriformes (five common crows, one fish crow, and two black-billed magpies [*Pica pica*]);
- six Ciconiiformes (five Chilean flamingos and a black-crowned night heron)
- three Pelecaniformes (guanay cormorants)
- two Charadriiformes (laughing gulls)
- three Anseriformes (two bronze-winged ducks [*Anas speculares*] and one mallard duck)

- three Galliformes (two Himalayan Impeyan pheasants [*Lophophorus impeyanus*,] and one Blyth's tragopan)
- one Falconiformes (northern bald eagle [*Haliaeetus leucocephalus alascanus*])
- one Strigiformes (snowy owl [*Nyctea scandiaca*])

All, except for the crows, were captive birds.

Some songbirds can be infected by WNV. The NYSDEC Wildlife Pathology Unit found that between 1999 and 2003, 23 percent of tested house finches (N = 43) and 14 percent of tested house sparrows (N = 427) were positive for WNV (Chu et al., 2003). Aside from corvids such as American crows, fish crows, and blue jays, and the house finches and house sparrows, only three percent of dead songbirds tested positive for WNV. However, other reports suggest cardinals are useful sentinels for WNV, having among the highest seroprevalence rates in areas where the virus is active (CDC, 2003e).

Audubon (2005) collected a series of reports of infections:

- captive (at the Toronto Zoo) eastern loggerhead shrike (*Lanius ludovicianus migrans*) with 100% mortality (i.e. all infected individuals died). Results suggested that the virus was likely shed in excreta and oropharyngial fluids, and that contact transmission may occur when the birds are housed together. There seemed to be a potential for these carnivorous birds to be infected through the consumption of infected prey, and, because the shrikes appear to be "exquisitely susceptible to WNV," suggest that "vaccination may be an important tool in the preservation of the species, at least in captivity" (Bertelsen et al., 2004)
- 80 of 98 (81.6 percent) captive owls (at the Owl Foundation in Ontario) of varying species that died of unknown causes were positive for WNV. Antibodies against WNV were found in most of the surviving owls. Differences in susceptibility were noted among species, with death rates greater than 90 percent for the snowy owl, great owl, Northern hawk owl, boreal owl, and Northern saw-whet owl, less than 20 percent for the long and short-eared owls, the great horned owl, flammulated owl, and Northern pygmy owl, and no mortality for the barn owl, burrowing owl, and Eastern screech owl.

Northern native breeding range and large-to-medium body size were found to be significant risk factors (Gancz et al., 2004).

- an average 25 percent reduction in survival of greater sage grouse due to WNV in Canada, where it is listed as endangered, and the western US, where it is under consideration for federal listing (Naugle et al., 2004).
- Citizen monitoring programs, including Christmas Bird Count (CBC), Great Backyard Bird Count, Breeding Bird Survey, and Project Feeder Watch generated preliminary data as suggesting large scale declines in crow populations and possible effects on chickadee, titmouse, blue jay, and great horned owls (Bonter and Hochachka, 2003a; Bonter and Hochachka, 2003b).

Domestic birds, including chickens and turkeys, have been the subject of several WNV studies due to the potential transmission of WNV to humans by eating infected meat. Also, domestic birds have been used in surveillance for bird-transmitted arboviral encephalitis (Langevin et al., 2001).

Research has shown that WNV is not a likely threat to domesticated birds. 11 of 12 chickens inoculated with WNV during experiments showed few, if any, clinical signs of the virus (Senne et al., 2000). In addition, Senne et al. observed no transmission of WNV from chicken to chicken under conditions of intimate contact, including exposure to contaminated feces. Langevin et al. (2001) inoculated 21 chickens, none of which developed the clinical disease, confirming Senne et al. Langevin et al. concluded that flock mates and human handlers are not at risk for WNV infection from chickens.

Due to the chicken's ability to seroconvert without developing clinical symptoms, it is often used as a sentinel species for WNV (Langevin et al., 2001; Cornell, 2004). Currently, several states use chicken flocks for surveillance (CDC, 2003e). However, it should be noted that there are differing opinions on the ability of chickens to re-infect mosquitoes with WNV. Langevin et al. (2001) deemed the magnitude of viremias in chickens insufficient to infect vector mosquitoes, while Senne et al. (2000) suggested that chickens could perpetuate the disease. In addition, sentinel chickens in Suffolk County did not seroconvert although located in areas of high virus activity (McCarthy et al., 2001).

Turkeys are similarly viewed as a potentially important reservoir for WNV due to high density farming techniques. As with chickens, turkeys do not appear to be susceptible to clinical symptoms of WNV when exposed to the disease. Only one of eight inoculated turkeys died after eight days. Control turkeys exposed to the inoculated turkeys showed no WNV-specific antibodies (Swayne et al., 2000). However, CDC testing of turkeys in Wisconsin associated with an outbreak of WNV infections in turkey farm workers found substantial antibody presence (Glaser et al., 2003).

The role of domestic geese (*Anser anser domesticus*) as a WNV reservoir has been supported by observations in Israel and the West Nile region. Infection rates of geese in the Sindbis District of the northern Nile Valley were 27 percent, similar to the rates of buffed-back herons (*Bubulcus ibis ibis*), doves (*Streptopelia senegalensis senegalensis*), and domesticated pigeons (*Columbia livia*), and twice the rate of domesticated chickens and ducks (*Anas platyrhynchos*) (Swayne et al., 2001).

In North America, domestic geese infected with WNV have been found in Manitoba, Canada, where 692 of 2,731 goslings died within a ten-day period at Manitoba Agriculture and Food in Winnipeg (Austin et al., 2004). In addition, a US experiment with goslings resulted in three of four exposed subjects showing signs of depression, weight loss, neck problems including torticollis and opisthotonus, and, eventually, death (Swayne et al., 2001).

The effects of WNV on bird populations appear to be "scattershot," with severe outbreaks confined to localized areas, suggesting that widespread birds such as crows have a good chance of persisting even if local populations are devastated. The greatest threat of WNV ecological impacts appear to be to endangered species whose numbers are so low that they might not be able to survive even a localized WNV outbreak (Chu et al., 2003).

Of 25 species tested in the laboratory, the five most competent reservoirs of WNV, all members of the order Passeriformes, were:

- blue jay
- common grackle (Q*uiscalus quiscula*),
- house finch (*Carpodacus mexicanus*),

- American crow
- house sparrow

Most fatal infections occurred in crows. Birds were shown to be susceptible to oral transmission of WNV, and that cage mates of infected birds may themselves become infected in the absence of mosquitoes. The potential transmission of WNV through ingestion may make carrion feeders, including crows, more susceptible to oral infection; birds that consume infected mosquitoes, including swifts and swallows, might also become infected without being bitten by mosquitoes (Komar et al., 2000, Komar et al., 2003a). Other potential routes of infection for birds include oral-fecal transmission, allopreening, and nestling feeding, all of which are likely to have greater importance within social species (Marra et al., 2004).

However, a study using data generated by CBCs did not find statistically significant effects "warranting conservation concern for any of the ten species examined" (Caffrey and Peterson, 2003). This was noted to be at odds with earlier studies on crow populations (Caffrey et al., 2003).

Acknowledging that little is known about effects on American birds, one report (Male, 2002) listed potential impacts as:

- sufficient immune response, with no species loss;
- taxonomic variation in immune response resulting in substantial mortality or extinction of some species;
- physiologically mediated variation in effects resulting in avian death during stressful conditions, such as long migratory journeys (as apparently occurred to migrating white storks in Israel in 1998, which are thought to have initiated the epizootic of WNV in Israeli geese [Malkinson et al., 2001; Malkinson et al., 2002]);
- insufficient immune response resulting in widespread extinctions.

3.3.2.4.3. Impacts to Bird Populations on Long Island

The CBC is a census of early-winter bird populations conducted by the Audubon Society each year using over 50,000 volunteers. Over 50 years of CBC data were examined to determine if the introduction of WNV in 1999 resulted in significant reductions of certain bird populations.

CBC counts for three susceptible corvids (the American crow, the fish crow, and the blue jay) and two known seropositive songbirds (the northern cardinal and the house sparrow).

Nine CBCs were identified are found on Long Island (see Figure 3-9). They are:

- Captree
- Central Suffolk
- Montauk
- Northern Nassau
- Orient
- Quoque-Watermill
- Sagaponack
- Smithtown
- Southern Nassau

Figure 3.9. Centers Associated with each Christmas Bird Count Circle

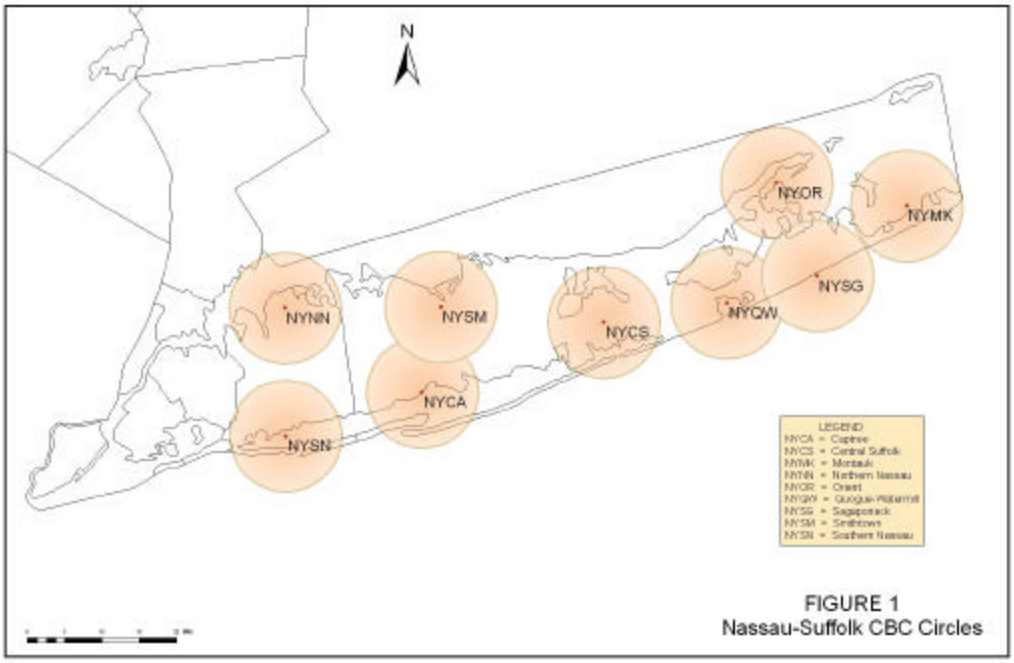
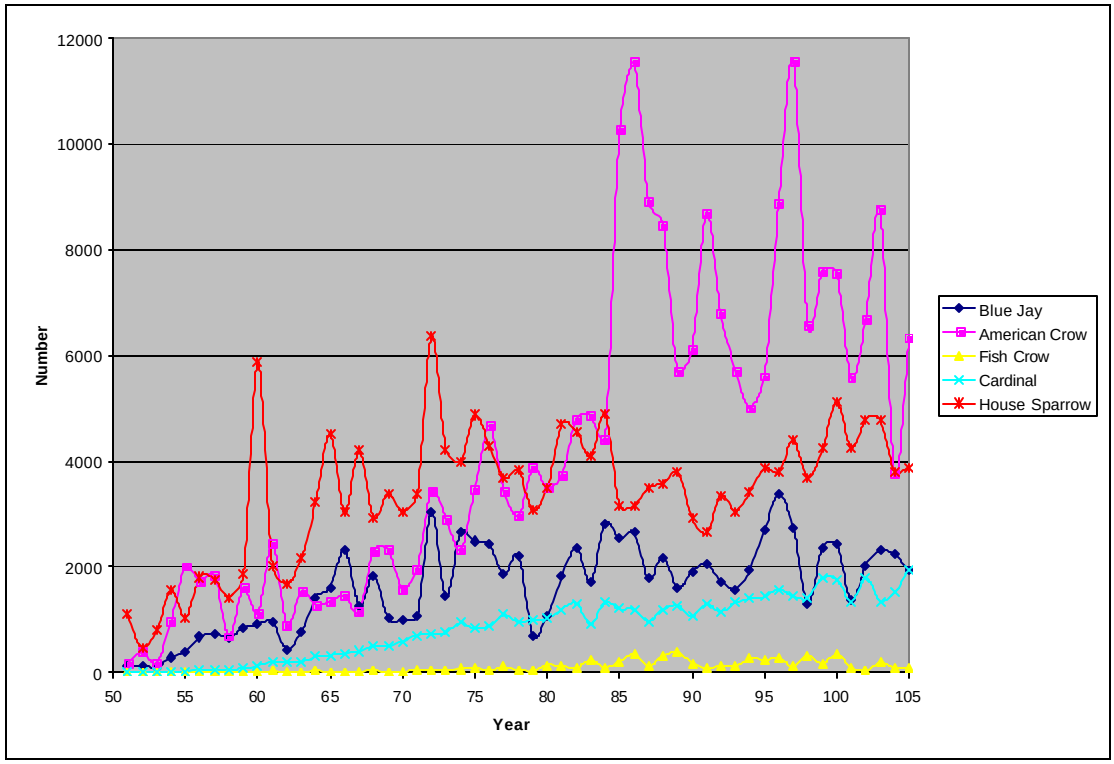


Figure 3-10 graphs summed CDC counts from the nine Long Island circles for over 50 years for the birds of interest.

Figure 3-10. CBCs on Long Island from 1950-2005



A more detailed analysis was conducted over two time periods, 1994 to 1999 (six years prior to the introduction of WNV), and 2000 to 2005 (six years following the introduction of WNV). "Decreases" in populations were determined in one of two ways:

1. the mean over 2000 to 2005 was less than that from 1994 to 1999
2. the linear trend over the time period 2000 to 2005 was negative, whereas the trend over the time period from 1994 to 1999 was upward or neutral.

The large variations in the data sets mean that in no case were any of these distinctions statistically significant at any reasonable probability ($p < 0.1$, for example).

Analysis of American Crow data for the nine CBCs shows that populations have decreased over 2000 to 2005 as compared to 1994 to 1999. Comparing six year population trends and/or means

of these data sets result in eight of the nine areas showing decreases[1]. The only exception is Quoque-Watermill. It is interesting to note that decreases, as measured by means, were not measured in three of the four East End circles. WNV has been characterized as an urban disease, because of its vector potential – chief reservoir combination (*Cx. pipiens*, the house mosquito, and the house sparrow) (Cashin Associates, 2005b). Waning effects on crows with decreasing human population densities tends to support such a characterization.

Therefore, it seems likely that County populations of American crow were impacted by WNV. However, in approximately half of the bird count circles, population numbers rebounded in 2005. In 2004, fewer dead birds were reported to the SCDHS, and there have been no dead crows in 2005 that tested positive for WNV at NYSDOH (one local result was positive). These data suggest that although crows may have been impacted by WNV immediately following its introduction, the effect is waning.

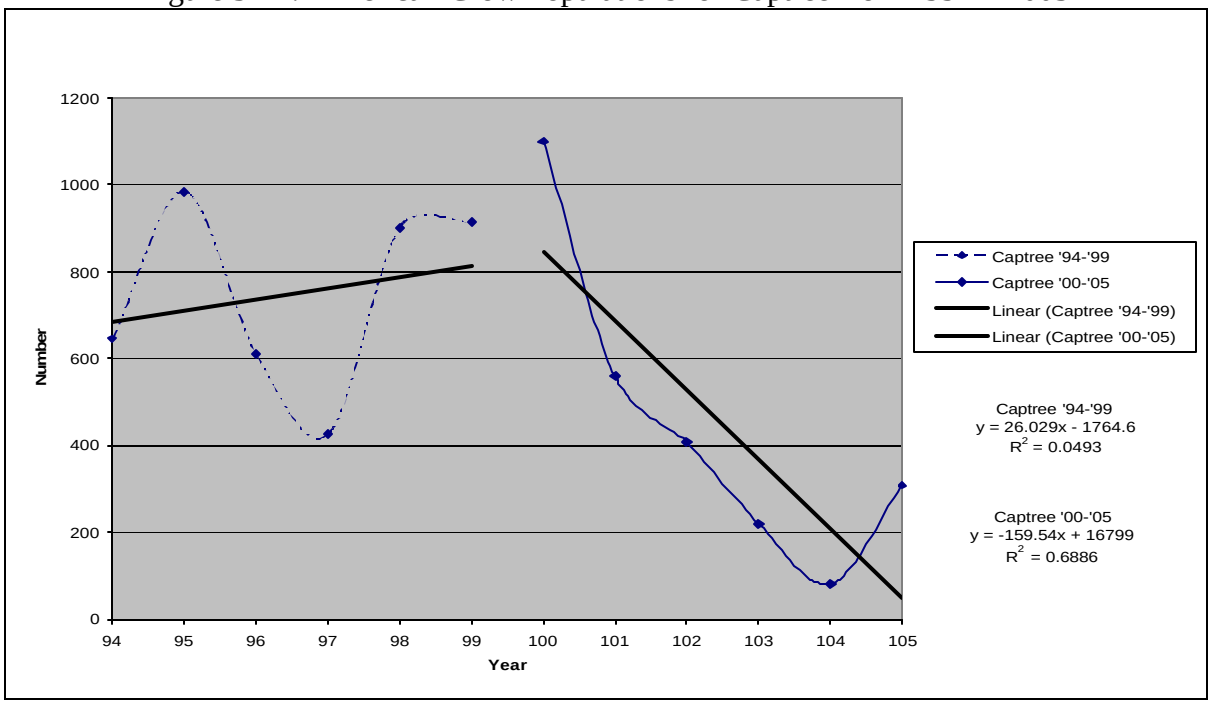
Table 3-14. American crow CBC counts

Area	Year	Mean	Trend (%)
Captree	1994-1999	747.2	3.5
	2000-2005	445.7	-35.8
Central Suffolk	*1994-1999	589.8	-0.4
	2000-2005	344.7	-18.2
Montauk	1994-1999	706.3	14.0
	2000-2005	835.3	-4.6
Northern Nassau	1994-1999	1180.0	0.7
	2000-2005	428.5	-20.0
Orient	1994-1999	800.2	9.0
	2000-2005	735.8	-9.1
Quoque-Watermill	1994-1999	542.3	2.3
	2000-2005	625.7	2.3
Sagaponack	1996-1999	414.8	25.2
	2000-2005	682.3	-1.5
Smithtown	1994-1999	1854.2	10.9
	2000-2005	1555.7	22.3
Southern Nassau	1994-1999	944.7	-6.4
	2000-2005	793.5	-26.4

*1995 data point intentionally disregarded in analysis.

[1] The 1995 data point for Central Suffolk was intentionally left out of the data analysis. Population numbers for that area tended to be between 100 and 800 birds for any given year. In 1995, the database recorded over 10,000 crows.

Figure 3-11. American Crow Populations for Captree from 1994 – 2005



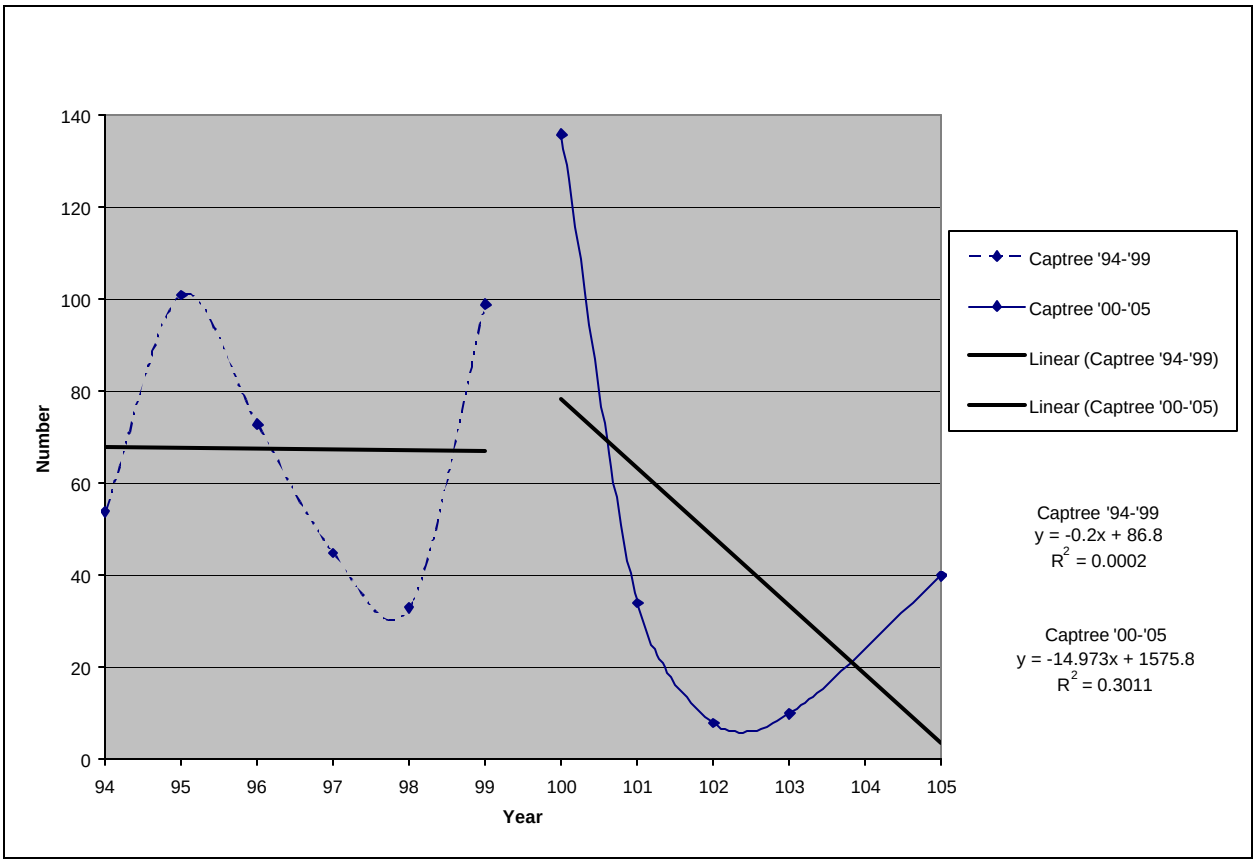
Fish Crow

Fish crow data for Long Island were generally incomplete. Only two of the nine Christmas bird count circles had data recorded for all 12 years of the study. The other seven sites had at least one missing data point (some as many as ten). Certain areas like Montauk, Quoque-Watermill, and Sagaponack were missing nearly all data from one or the other time period, making it impossible to compare the two periods. However, for the other six Christmas bird count circles that comparisons could be made on, population numbers decreased from 2000 to 2005. From what can be determined for most of the county, fish crow populations have decreased in number since 1999. This suggests WNV affected fish crow populations. Data are insufficient to determine whether or not fish crow populations have rebounded in 2005.

Table 3-15. Fish crow CBC counts

Area	Year	Mean	Trend (%)
Captree	1994-1999	67.5	-0.3
	2000-2005	45.6	-32.8
Central Suffolk	1994-1999	9.8	-10.2
	2002-2005	2.5	-40
Northern Nassau	1994-1999	7	19.6
	2000-2005	10.3	-29.3
Orient	1995-1999	1.8	-22.9
	2002-2005	1.3	-8
Smithtown	1994-1999	23.3	22.0
	2000-2005	16.7	1.0
Southern Nassau	1994-1999	120.2	-12.9
	2000-2005	76.3	-15.1

Figure 3-12. Fish Crow Populations for Captree from 1994-2005



Blue Jay

Analysis of data show blue jay populations decreased for six of these nine areas. The three areas of Long Island that did not show a decrease in population number were Montauk, Sagaponack, and Smithtown.

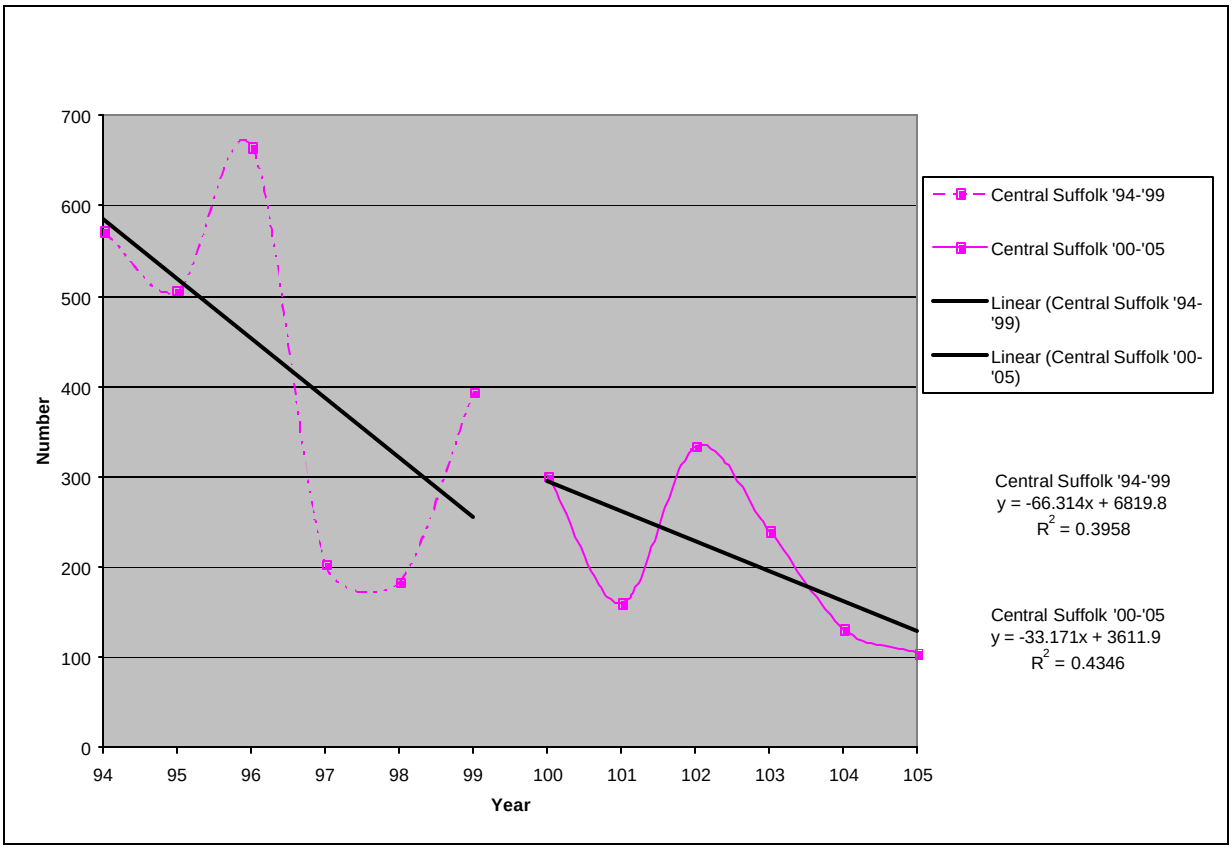
Although Montauk showed a slight decrease in the mean populations, and the five year trend for 2000 to 2005 declined slightly, the blue jay population for that area essentially was the same for the two time periods. Blue jay populations in Sagaponack suffered a decline from 2000 to 2001 immediately following the introduction of WNV; however, the population was able to rebound greatly so that the mean for 2000 to 2005 was about double of what it was for 1994 to 1999. Mean blue jay populations in Smithown before and after WNV remained essentially the same.

In all but three areas of Long Island, blue jay populations continue to decrease into 2005. Therefore, in the majority of Long Island, blue jay populations seem to have been affected by WNV, and most areas are still being affected.

Table 3-16. Blue jay CBC counts

Area	Year	Mean	Trend (%)
Captree	1994-1999	103.8	2.6
	2000-2005	66.7	-1.3
Central Suffolk	1994-1999	420.5	-15.8
	2000-2005	211.8	-15.7
Montauk	1994-1999	476.7	-5.3
	2000-2005	462.8	-5.8
Northern Nassau	1994-1999	201.5	-1.6
	2000-2005	140.2	-1.3
Orient	1994-1999	747.2	-5.7
	2000-2005	461.8	3.0
Quoque-Watermill	1994-1999	157.5	-10.0
	2000-2005	133	-5.2
Sagaponack	1996-1999	198	50.5
	2000-2005	410.8	18.5
Smithtown	1994-1999	102	-6.8
	2000-2005	102	-1.3
Southern Nassau	1994-1999	63.5	8.9
	2000-2005	53.8	-11.3

Figure 3-13. Blue Jay Populations for Central Suffolk from 1994-2005



Northern Cardinal

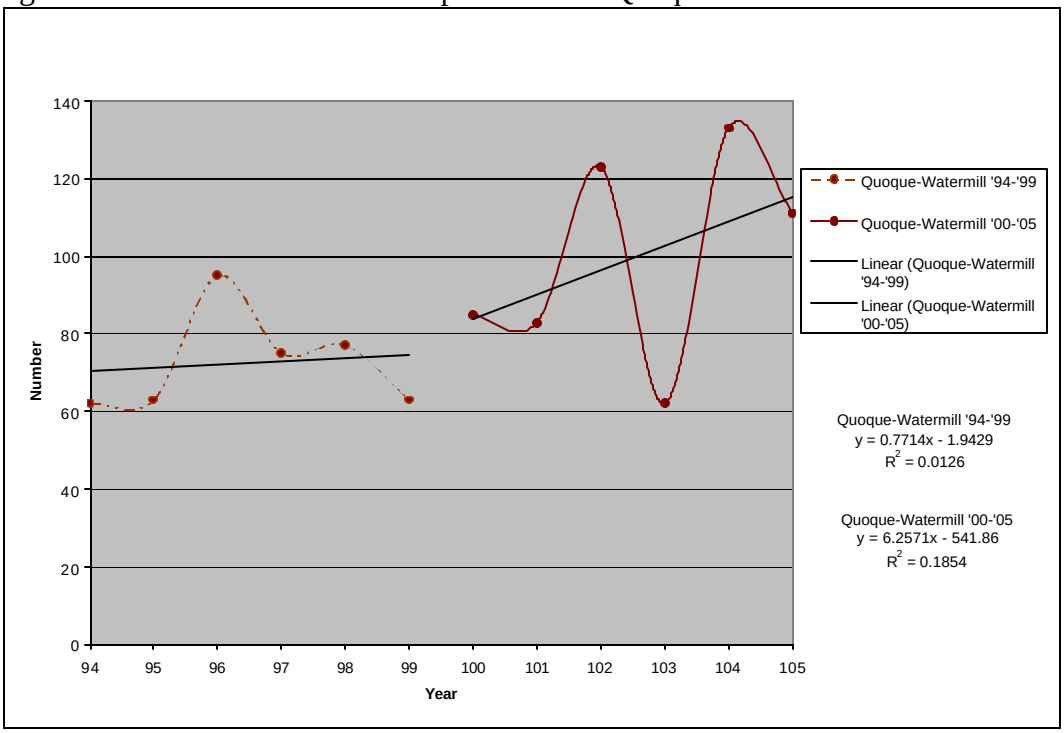
Comparisons of northern cardinal population means and/or trends for 1994 to 1999 with 2000 to 2005 do not show general decreases in population. Cardinal populations decreased after 1999 in only four of the nine Christmas bird count circles. The other five either increased in number or remained the same. Therefore, it seems likely that northern cardinals were not affected by WNV after its introduction in 1999 and are still not affected today.

Table 3-17. Northern cardinal CBC counts

Area	Year	Mean	Trend (%)
Captree	1994-1999	113.2	0.7
	*2000-2005	79.6	-3.5
Central Suffolk	1994-1999	149.8	-7.2
	2000-2005	119	3.8
Montauk	1994-1999	256.7	-2.9
	2000-2005	288.7	-4.0
Northern Nassau	1994-1999	240.2	1.7
	2000-2005	221	-3.3
Orient	1994-1999	303	8.7
	2000-2005	243.7	1.9
Quoque-Watermill	1994-1999	72.5	1.1
	2000-2005	99.5	6.3
Sagaponack	1996-1999	137.3	33.2
	2000-2005	258.2	10.3
Smithtown	1994-1999	120.7	3.4
	2000-2005	126.3	3.9
Southern Nassau	1994-1999	156.3	-7.3
	2000-2005	169	-1.8

*2005 data point intentionally disregarded in analysis.

Figure 3-14. Northern Cardinal Populations for Quoque-Watermill from 1994-2005



House Sparrow

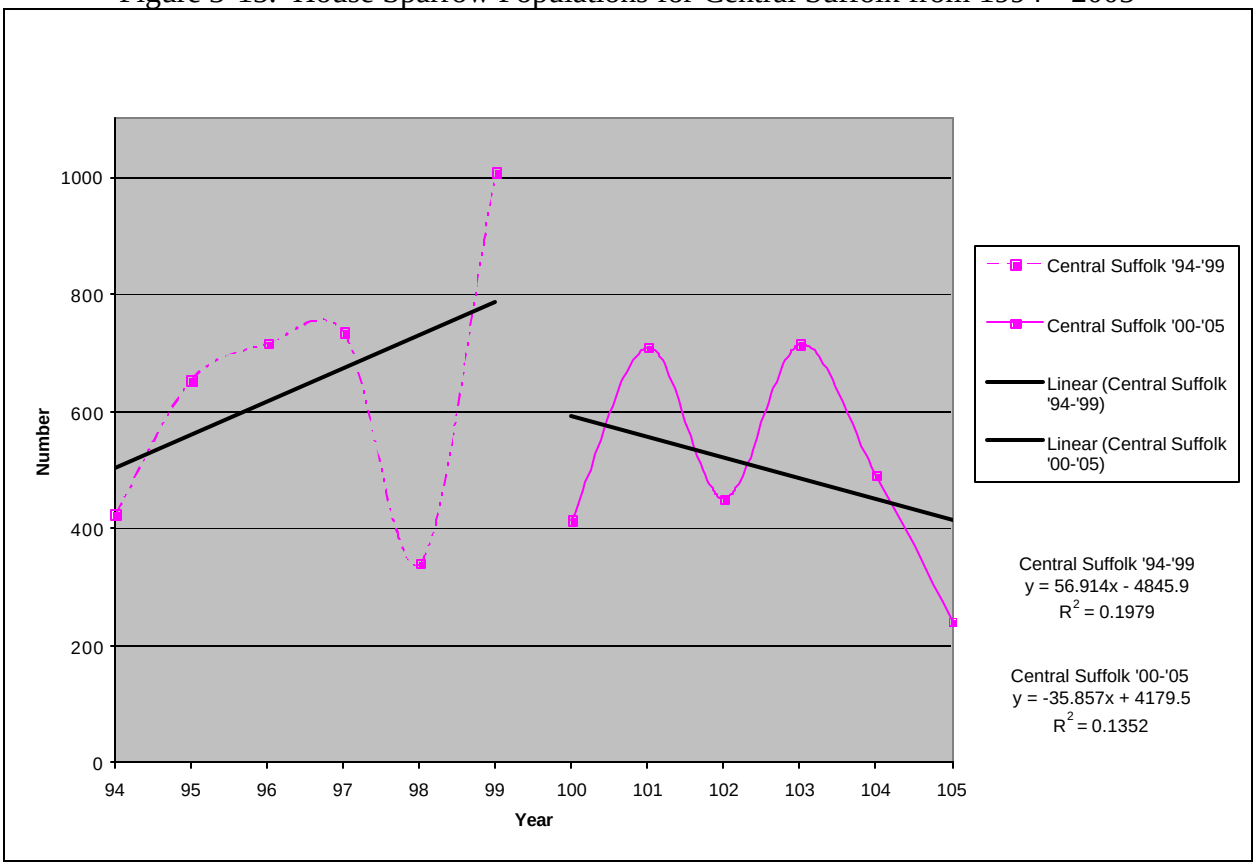
Comparisons of six-year trends and/or means of house sparrow populations before and after 1999 yield inconclusive results. In five out of nine bird count circles, house sparrow populations decreased with the introduction of WNV in 1999. The four areas that did not show reduction in population numbers were Montauk, Sagaponack, Smithtown, and Southern Nassau.

However, house sparrow populations have generally been declining over the last 100 years. This further complicates the issue, and so clear declines in populations should be determined to impute a role for WNV.

Table 3-18. House sparrow CBC counts

Area	Year	Mean	Trend (%)
Captree	1994-1999	519.2	-13.9
	2000-2005	381.2	-3.0
Central Suffolk	1994-1999	646.3	8.8
	2000-2005	504.2	-7.1
Montauk	1994-1999	401.7	-1.3
	2000-2005	723.5	-6.5
Northern Nassau	1994-1999	613.3	5.2
	2000-2005	650.5	-9.6
Orient	1994-1999	479.8	10.8
	2000-2005	474.3	1.3
Quoque-Watermill	1994-1999	149	-1.4
	2000-2005	185.3	-6.2
Sagaponack	1996-1999	311.8	15.9
	2000-2005	405.7	-1.3
Smithtown	1994-1999	212.3	-0.2
	2000-2005	212.8	1.1
Southern Nassau	1994-1999	667.7	-4.2
	2000-2005	900.8	-5.6

Figure 3-15. House Sparrow Populations for Central Suffolk from 1994 - 2005



Based on analysis of Audubon CBC data, certain species of birds in Suffolk County have been impacted by WNV. American crows on Long Island have been affected by WNV, but impacts appear to be decreasing. Fish crows and blue jays also have been affected, and it is not clear whether the effects are continuing or not. This generally supports national information that corvids have been impacted by the virus. There are some suggestions in the data that the impact may not be evenly distributed across the County, but rather that greater impacts were received by western populations. The two songbirds studied, although known to be infected by WNV, do not seem to have had population impacts.

3.3.2.5. Horses and WNV

In 1999, when the first human cases of WNV occurred in New York City, WNV was also diagnosed in 20 horses in Suffolk County (Trock et al., 2001). In 2000, another 63 equine cases were diagnosed in the northeast US (Peterson and Roehrig, 2001), and in 2001, 631 confirmed horse cases, mostly in Florida (492 cases) were diagnosed. In 2002, with the explosion of WNV

across much of the Midwest and southern US, 14,717 cases were diagnosed in horses (American Horse Council, undated). Another 4,000 cases were diagnosed in 2003 (Castillo-Olivares and Wood, 2004).

Epidemiologically, 10 percent of equine cases result in neurological disorders (Petersen and Roehrig, 2001), with mortality rates between 30 and 40 percent (Cantile et al., 2001). Up to 25 percent of horses stabled with symptomatic horses test positive for the virus (Castillo-Olivares and Wood, 2004). Symptoms are consistent with spinal cord injury, and include paralysis of legs (usually the hind limbs), muscle tremors and rigidity. A proportion of horses also displayed symptoms associated with brain damage, ranging somnolence to hyperexcitability and aggression, or facial and tongue paralysis (Ostlund et al., 2001). Early signs of potential infection include loss of coordination (stumbling) and anorexia (Williams et al., undated). Treatment is intended to reduce central nervous system inflammation symptoms, preventing the horse from injuring itself, and palliative care (Castillo-Olivares and Wood, 2004).

Experimental studies on equine WNV have shown that horses are not likely to be an amplifying host of the disease. Horses infected with WNV most often develop viremias of low magnitude and short duration that are not conducive to re-infecting mosquitoes or other animal species (Bunning et al., 2002).

A whole virus killed vaccine has been developed for horses (Ng et al., 2003). It has been shown to be 93 to 95 percent effective, and is licensed by the US Department of Agriculture. It is recommended that two treatments be given initially at three to six week intervals in March or April, followed by annual boosters (Williams et al., undated). Passive immunity can be developed in foals by vaccinating pregnant mares (Poland et al., 2005). If this course is not followed, foals should be vaccinated at six to eight weeks of age with three injections three to six weeks apart. In either case, a booster should be given in early August. A recombinant DNA vaccine has also been developed (Williams et al., undated). Each vaccination costs approximately $25 (APHIS, 2003).

3.3.3. Eastern Equine Encephalitis

EEE is a mosquito transmitted pathogen that occurs naturally in a wide variety of birds along the eastern flyway of the United States (Morris, 1988; Scott and Weaver, 1989). The virus produces

the clinical disease eastern equine encephalomyelitis in humans with a mortality outcome between 30 and 75 percent and is virtually 100 percent fatal in horses (Morris, 1988). Chamberlain (1958) felt that EEE reached highest levels in coastal areas where freshwater swamps joined salt marsh habitat. *Cs. melanura*, a bird feeding mosquito that uses acid water swamps as habitat, has been identified as the primary enzootic vector in Georgia (Chamberlain et al., 1958), Maryland (Dalrymple et al., 1972), New Jersey (Burbutis and Jobbin, 1957), New York (Morris, et al. 1980), Connecticut (Wallis, 1959), and Massachusetts (Grady et al., 1978). *Culiseta melanura* appears to be a fixed avian feeder (Edman et al., 1972) and is probably not responsible for the transmission of EEE to either humans or equine hosts. Although the epidemic vectors probably vary over the geographic range of this virus, salt marsh mosquitoes play an important role in areas where tidal marshland impinges on the breeding habitat utilized by *Cs. melanura*. Epidemiological data collected during the 1959 outbreak of EEE in New Jersey led Hayes et al. (1962) to hypothesize that the salt marsh mosquito, *Oc. sollicitans,* served as the primary epidemic vector in the coastal zone where all of the human cases occurred. Crans (1977) used Koch's postulates to show that *Oc. sollicitans* met basic criteria to indirectly prove vector status and suggested that the species should be controlled for the prevention of human disease whenever EEE is found to be active.

Descriptions in the veterinary literature suggest that EEE was probably cycling between mosquitoes and birds to the north and south of Suffolk County prior to the turn of the 20th century (Cohen and Sussman, 1957). Goldfield and Sussman (1968) reported that several hundred horse deaths with symptoms consistent with EEE were reported by New Jersey veterinarians as early as 1905. They list additional equine episodes, probably, caused by EEE, in 1912, 1920, 1928, and 1933. Beadle (1952) provides similar historical evidence for EEE on Long Island. The first definitive evidence associating equine deaths with EEE was obtained when Ten Broeck and Merrill (1933) isolated the type specimen for EEE from a horse that died from the disease in Burlington County, New Jersey. The location where the isolation was obtained is less than 150 miles from Suffolk County. EEE was recognized as a fatal pathogen of wild ring-necked pheasants in Connecticut five years after the initial virus isolation (Tyzzer et al., 1938) and was linked to numerous large-scale commercial pheasant die-offs in New Jersey in subsequent years (Beaudette and Hudson, 1945; Beaudette and Black, 1948). Unlike native

species, exotic birds such as the ring-necked pheasant lack natural immunity to EEE and succumb to the virus fairly early in the amplification cycle. The first evidence for human involvement with this mosquito-borne pathogen came when 34 human cases accompanied the 1938 epizootic in Massachusetts (Fothergill et al., 1938). The severity of the pathogen was underscored when 67 percent of those infected died from the disease (Calisher, 1994). Notable outbreaks of EEE involving 10 or more cases in the northeast have taken place in Massachusetts (1938, 1947, 1955, and 1956) and New Jersey (1959 and 1965). Lesser numbers have occurred sporadically in the intervening years. Calisher reported an incidence of zero to 36 human cases, with an average of seven, per year in the eastern United States since 1955. During the period 1964 to 1992 the average dropped to five cases per year, which is thought to be a result of vector control intervention.

EEE is perpetuated in eastern North America in a benign maintenance cycle that involves the bird feeding mosquito, *Cs. melanura*, as the primary enzootic vector and passerine birds as the amplification hosts. The term epiornitic is often used to describe the amplification phase of this virus because birds are the only vertebrate hosts involved in this portion of the cycle (Howard et al., 1994). When EEE reaches epiornitic proportions in the northeastern region, clinical disease is frequently reported from horses. Epiornitics leading to human infections are sporadic and considerably less frequent than equine involvement. Since *Cs. melanura* does not accept mammalian hosts, mosquitoes that accept both mammals and birds function as bridge vectors to pass the virus from avians to mammals during recognized outbreak periods.

Two rather distinct ecological cycles have been described for EEE in the northeastern United States. An inland cycle that emanates from red maple swamps (*Acer rubrum* L.) and surrounding wet woodlands has been reported from Massachusetts (Grady et al., 1978), Connecticut (Wallis et al., 1974), upper New York State (Morris et al., 1980), and New Jersey (Crans et al., 1986a). The inland cycle involves *Cs. melanura* as the amplification vector and *Ae. vexans*, *Oc. canadensis*, or *Cq. perturbans* as vectors to mammalian hosts (Calisher, 1994). A similar inland cycle has been described from the central flyway where *Cq. perturbans* functioned as a bridge vector in Michigan (McLean et al., 1985). For as yet unknown reasons, the inland cycle claims large numbers of equine cases, but only rarely involves humans.

A coastal cycle of EEE amplification occurs almost annually from Maryland to New Jersey in areas where Atlantic white cedar swamps (*Chamaecyparis thyoides* [L.]) drain into the salt marsh ecotone originally described by Chamberlain (1958). The coastal cycle involves *Cs. melanura* as the amplification vector and *Oc. sollicitans* as the primary vector to mammalian hosts. Convincing evidence exists that the salt marsh mosquito, *Oc. sollicitans*, functions as the primary vector to humans (Kandle, 1960; Hayes et al., 1962; Goldfield and Sussman, 1968; Crans, 1977; Crans et al., 1986b). Unlike the inland cycle where horses are the primary mammalian victims, EEE episodes in coastal areas often involve human cases.

Cs. melanura exhibits a bivoltine life cycle in the zone where human outbreaks have occurred that creates a seasonal cycle of virus amplification that contributes to salt marsh mosquito involvement (Mahmood and Crans, 1998). Unlike most mosquito species, *Cs. melanura* overwinters as a larva in subterranean habitats where cold water temperatures slow down larval growth considerably. The first generation of adults emerges during the month of May from larvae that have spent the winter in their fourth instar. These mosquitoes seek hosts in the canopy and blood feed on nesting adult birds. No juveniles are present in the bird population when the first generation of *Cs. melanura* is building. A high proportion of the adult birds that are being fed upon on the ecotonal edge of salt marsh-cedar swamp habitat have antibodies to EEE from a prior year's infection, preventing widespread amplification during late May and early June. Crans et al. (1994) found evidence for EEE recrudescence in a small proportion of adult birds at this time of year. If that is the case, EEE is present, but is maintained at very low levels due to the low number of susceptible hosts. The eggs from the first generation of adult mosquitoes are laid in cedar swamp habitat during the months of May and June. Cold habitat water retards development of the second generation of *Cs. melanura*, slowing down emergence of new adults until sometime in July and August. When this generation of *Cs. melanura* emerges, young of the year birds lacking immunity to EEE make up a high proportion of the avian population. This sets the stage for virus amplification directly in the saltmarsh–cedar swamp ecotone, historically described as the primary EEE focus. Salt marsh mosquitoes appear in broods during the summer months that are generated by lunar tides and typically move inland to search for blood (Headlee, 1945). Whenever amplification of EEE coincides with an inland migration of *Oc. sollicitans*, newly emerged mosquitoes pass directly through the area of greatest

virus activity as they penetrate the upland in quest of blood, setting the stage for contact with viremic birds and potential vector involvement.

Chamberlain (1956) was the first to point out that a mosquito's ability to transmit by bite is considerably more important than its ability to become infected, because many species possess innate barriers that prevent them from passing the virus on. His rationale was drawn from studies (Chamberlain et al., 1954) where *Oc. sollicitans* was rated as "excellent" after 75 percent of the specimens in a test sample successfully transmitted EEE to susceptible chicks. He then compared 19 potential vectors of EEE. Most of the mosquitoes tested in these studies yielded transmission rates of less than 50 percent. *Ae. vexans*, for example, a recognized vector of EEE in the northeast, showed only a 63 percent infection rate and 13 percent transmission rate. Table 3-19 lists mosquitoes found on Long Island in terms of the two categories used by Chamberlain in his study.

Table 3-19. Comparative EEE Infection and Transmission Rates for Long Island Mosquitoes

Mosquito Species	Percent Infected	Percent Transmitting
Oc. triseriatus	100	86
Oc. sollicitans	100	75
Cx. restuans	45	33
Ps. confinnis	100	22
Cq. perturbans	94	18
Ps. ciliata	83	18
Ps. ferox	100	15
Ae. vexans	63	13
An. quadrimaculatus	79	0
Cx. salinarius	3	0

3.3.4. Recent History of Mosquito-borne Disease in Suffolk County

Public health emergencies due to mosquito-borne diseases have been declared in seven of the ten years prior to 2004 in Suffolk County. These health threats occurred in 1994, 1996, 1999, 2000, 2001, 2002 and 2003 (Graham and Harper, 2004). Health emergencies were also declared in 2004 and 2005.

The health threats prior to 1999 were due to EEE. Despite the fact that conditions appeared to constitute a risk of transmission of EEE from bird reservoirs to people, there were no resulting cases. In 1999, human health threats were declared due to malaria and the initial outbreak of WNV. The human health emergencies declared from 2000 to 2002 were all due to WNV. The

health emergencies in 2003 through 2005 were also due to WNV, although EEE discovered in the Montauk area also resulted in a declaration of a health emergency in 2003 (S. Campbell, SCDHS, personal communications, 2004, 2006)

From 1999 through 2004, 19 human cases of WNV meningitis or encephalitis occurred, and two human cases of malaria occurred. The cases occurred as follows:

- Two children contracted malaria in 1999;
- One person contracted WNV meningitis in 2001;
- Eight people contracted WNV meningitis or encephalitis in 2002; two cases resulted in death;
- 10 people contracted WNV meningitis or encephalitis in 2003; two cases resulted in death.

In 2005, an additional seven people contracted WNV meningitis (S. Campbell, SCDHS, personal communications, 2004, 2006)

Of the 19 human cases of WNV meningitis or encephalitis in Suffolk County prior to 2005, 11 resulted in neurological complications lasting months (P. Dillon, SCDHS, personal communication, 2004). There were no cases in 2004. There seven cases in 2005. The diseases of concern in Suffolk County are thus WNV and EEE. Although the two cases of malaria in 1999 appear to have been locally transmitted, as the infected children had not traveled to any malarial regions, malaria is not considered to be a disease of special concern in the County. Please note that when malaria was endemic in the County (up until the 1920s) it was the bacterial parasite *P. vivax* that was responsible (a temperate zone malaria) rather than the *P. falciparum*, which continues to kill so many worldwide. *P. vivax* was isolated from the campers in 1999 (D. Ninivaggi, SCVC, personal communication, 2004).

Certain species of mosquitoes are recognized, or suspected, vectors for EEE, WNV and other mosquito-borne diseases. *Ochlerotatus sollicitans, Oc. taeniorhynchus,* and *Oc. cantator* are salt marsh mosquitoes, and are considered to be aggressive feeders. *Aedes vexans* is another flood water mosquito, albeit from fresh water habitats, and is also an aggressive biter (CA-CE, 2004).

These flood water mosquitoes present a particular worry as a vector threat because of their aggressive biting, and their large populations in areas of Suffolk County.

Ae. vexans and *Oc. sollicitans* are known vectors for EEE, whereas *Oc. taeniorhynchus* and *Oc. cantator* are unlikely, or are not capable, vectors for EEE. *Ae. vexans* and *Oc. cantator* are considered poor transmitters of WNV, while *Oc. sollicitans* and *Oc. taeniorhynchus* are considered moderate transmitters of WNV. All of these salt marsh mosquitoes have tested field positive for WNV on Long Island (CA-CE, 2004).

Oc. trivittatus and *Coquillettidia perturbans* are both fresh water mosquitoes and are aggressive feeders. Both have tested field positive for WNV in Suffolk County. While *Oc. trivittatus* is an unlikely transmitter of EEE, it is a likely transmitter of WNV. *Cq. perturbans* is a known vector for EEE, and is a poor transmitter of WNV. *Ochlerotatus canadensis* is another fresh water mosquito that is moderately aggressive. Although, it is a spring mosquito, it is extremely long-lived for a mosquito, and so is a potential vector for both EEE and WNV (CA-CE, 2004).

Suffolk County's container breeding mosquitoes are *Oc. japonicus, Oc. triseriatus, Culex pipiens,* and *Cx. restuans*. *Oc. japonicus* and *Oc. triseriatus* are moderately aggressive to aggressive feeders which breed in containers and tree holes. They are not known, or are unlikely, to transmit EEE. However, they have tested field positive for WNV and are considered moderate to good transmitters of the disease (CA-CE, 2004).

Cx. pipiens and *Cx. restuans* breed in fresh, permanent water and in containers. They have both tested field positive for EEE and WNV in Suffolk County. *Cx. restuans* is considered to be an amplification vector for WNV, as it appears to solely feed on birds. *Cx. pipiens* is not very aggressive, but is considered to be the prime vector for West Nile virus in the northeast US (CA-CE, 2004).

Anopheles punctipennis is a potential bridge vector for WNV. *Anopheles quadrimaculatus* is the malaria vector in Suffolk County (CA-CE, 2004).

Cx. salinarius and *Culiseta melanura* are the final key mosquito species in Suffolk County. *Cx. salinarius* breeds in fresh and brackish water habitats, and is an unlikely transmitter of EEE. It has tested field positive for WNV. It is a moderate transmitter of WNV, and has been identified as the major WNV vector in Connecticut (Andreadis et al., 2004). This mosquito is an

aggressive biter, but has not been considered to be major pest species in Suffolk County because it does not have much available habitat to breed in. Its incidence has always been low in trap analyses in the past, but 2005 trap analyses showed much higher counts than before (S. Campbell, SCDHS, personal communication, 2006). It is not clear whether this is an aberrational result, or comes from more careful species identifications (*Culex* species are especially difficult to distinguish, and mistakes in speciation are so common that, for example, *Cx. pipiens* and *Cx. restuans* are rarely distinguished). *Cs. melanura* breeds in maple and cedar swamps, and is a bird biter. It is an epiornitic vector for EEE, transmitting the disease between bird species, which amplifies the incidence of the disease. *Cs. melanura* has tested field positive for WNV, but is not considered a transmitter of the disease (CA-CE, 2004). Table 3-20 summarizes the above discussion.

Table 3-20. Mosquito Species of Concern in Suffolk County

Species	**Vector Status**	**Other Issues**
Aedes vexans	Known WNV bridge vector Probable EEE bridge vector	Aggressive, SC's major fresh flood water mosquito
Anopheles punctipennis	Possible WNV bridge vector	Pesky, enters houses
Anopheles quadrimaculatus	Malaria vector	Moderately aggressive
Coquillettidia perturbans	EEE bridge vector	Aggressive human biter, breeds in emergent fresh marshes
Culex pipiens	WNV amplification vector Probable WNV bridge vector	Breeds near (containers, catch basins, other standing water) and enters houses
Culex restuans	WNV amplification vector	Often breeds with *Cx. pipiens*
Culex salinarius	WNV bridge vector	Irritating biter, breeds in brackish flood water (rare here)
Culiseta melanura	EEE amplification vector Probable WNV amplification vector	Breeds in environmentally-sensitive habitats, making control problematic
Ochlerotatus canadensis	Probable EEE bridge vector Possible WNV bridge vector	Spring fresh water mosquito, extremely long lived, avid human biter
Ochlerotatus cantator		Spring salt water mosquito, moderately aggressive
Ochlerotatus japonicus japonicus	WNV bridge vector	Tree-hole (tire) mosquito, causes local biting complaints, moderately aggressive
Ochlerotatus sollicitans	EEE bridge vector Probable WNV bridge vector	SC primary pest species, extremely aggressive, salt water flood mosquito
Ochlerotatus taeniorhynchus		Aggressive salt water flood mosquito

Ochlerotatus triseriatus	Possible WNV vector LaCrosse encephalitis vector	Irritating pest, containers-tree holes-tires mosquito
Ochlerotatus trivittatus	Possible WNV vector	Aggressive fresh flood water (recharge basins) mosquito

Amplification vector: increases the incidence of disease in host organisms
Bridge vector: infected by host, and transmits disease to people (or other target organism)

3.3.5. Additional Impacts from Mosquito Biting

The public welfare is directly impacted by the diseases that mosquitoes transmit, but there are a number of sub-clinical effects that result from mosquitoes that have taken previous blood meals from non-human sources. Mosquitoes are potent vectors of three types of organisms that are pathogenic to mammalian hosts (Harwood and James, 1979). These include the plasmodia (causal agents of the animal malarias), filarial worms (causal agents of animal filariasis), and viruses (causal agents of a wide variety of vertebrate arboviral infections). Parasitic agents that are vectored by mosquitoes rely on an alternate life cycle strategy to perpetuate themselves (Eldridge and Edman, 2000). One portion of the life cycle is completed in the mosquito and the second portion takes place in the vertebrate host. In all cases, the parasite has a life cycle stage that enters the blood. This allows the parasite to be ingested when the insect finds the host to take a blood meal. The parasitic protozoans, filariae, or viruses that are picked up during blood feeding undergo a period of incubation in the insect where they either replicate or change in form. The incubation period varies from several days to several weeks depending on the parasite involved. Transmission back to a suitable vertebrate host, therefore, requires an insect, like the mosquito, that accepts multiple blood meals and blood feeds repeatedly over intervals of days, weeks or months.

Many of the mosquito-borne parasitic agents are species specific and can only develop to maturity in one vertebrate species. As a result there are numerous animal malarias, animal filarial worms, and animal viruses present in wild-caught mosquitoes that can be accidentally transferred to humans even though people are an unnatural host for the parasite. However, parasites rarely develop fully if they are transferred to a non-specific vertebrate host. In most cases, the parasites circulate for a brief period in the blood stream and eventually die and disintegrate. In some cases, the parasites invade host tissue and persist as a benign agent which does little or no harm. Viruses and protozoans are microscopic parasites that often promote

antigenic reactions in atypical hosts. Many persistent welts that follow some mosquito bites are believed to represent parasite introductions that resulted in aborted or benign infections (Feingold et al., 1968). Filarial worms are macroscopic parasites that are too large to enter the salivary glands of the mosquito and be transmitted by injection with salivary fluid. The infective stage of mosquito-borne filarial worms travels down the proboscis and break out onto the skin when the mosquito is probing for the blood meal. (Hawking and Worms, 1961). The worms then enter the wound that the mosquito leaves behind and circulate in the blood until their preferred tissue is found. Because of their size, zoonotic filariae pose a relatively larger sub-clinical risk than viruses and protozans. If the parasites grow to full maturity in the human host, the adult worms of some species can grow to a foot or more in length. (Eldridge and Edman, 2000).

The causal agent of dog heartworm is a filarial worm that can develop to maturity in humans but cannot reproduce. The parasite is usually discovered in humans at autopsy in the heart and adjacent vessels or folded in the pulmonary artery when lung sections are removed during cancer surgery (Harwood and James, 1979). Neafie and Piggott (1971) feel that dog heartworm infections in the heart and pulmonary artery of humans are far more prevalent than realized. Beaver and Orihel (1965) report occult cases of filarial infection (presence of adult worms without microfilariae in the bloodstream) where the parasites lodge and grow in atypical tissue. They feel that filarial worms recovered from human subcutaneous nodules and abscesses include a wide variety of animal filariae that were accidentally transmitted to humans by mosquitoes. Representative filarial parasites accidentally acquired from mosquito bites include:

- *Dirofilaria tenuis* (raccoons)
- *Dirofilaria scapiceps* (cottontail rabbits)
- *Dirofilaria subdermata* (porcupines)

Bartlett and Anderson (1981) described occult filariasis in crows where the adult birds served as atypical hosts for *Slendidofilaria caperata*, an insect-borne filarial worm that infects a relatively wide range of other passerine hosts. Turell et al. (1984) provide interesting evidence to suggest that the presence of zoonotic microfilariae in mosquitoes actually enhances their ability to transmit arboviruses to mammalian hosts, which is of more than passing interest given the recent connection between human biting mosquitoes, crows, and WNV virus in the northeastern US.

Allowing large mosquito populations to age naturally in the absence of known human pathogens can pose a masked risk to human health. Flood water mosquitoes, in particular, pose the greatest risk in this regard because of their unique life cycle type. This group of mammalian feeding mosquitoes appears in waves during the summer season and feeds repeatedly until natural mortality eliminates the adults. Blood meal tests clearly show that the majority of the biting adults feed on the same kinds of wildlife that harbor known filarial parasites in nature (Apperson et al., 2004). Little information is available on the actual rate of wildlife parasites in human biting mosquito species but data are available for *Dirofilaria immitis*, the causal agent of dog heartworm, from many parts of the US. Crans and Feldlaufer (1973) examined large numbers of wild-caught mosquitoes from coastal areas of New Jersey to implicate salt marsh mosquitoes as vectors of this canine disease. They found filarial infection rates approaching 10 percent in some of the longer lived human biting species, when the residual mosquito populations were at their oldest. Many of the infective stage larvae present in the mosquitoes were filariae from unknown wildlife, as measurements and morphological features ruled out *D. immitis* as the species.

D. immitis infestations have been reported from a wide variety of human biting flood water species found in Suffolk Co. including: *Ae. vexans* (Bemrick and Sandholm, 1966), *Ae. trivittatus* (Christensen and Andrews, 1976), *Ae. canadensis* (Crans and Feldlaufer, 1973), and *Ae. triseriatus* (Intermill, 1973). Investigators have also reported filarial worms in wild-caught mosquitoes from areas immediately adjacent to Long Island including Massachusetts (Arnott and Edman, 1978), Connecticut (Magnarelli, 1978), and upstate New York (Todaro et al., 1977). Although no immediate health threats can be directly attributed to sub-clinical parasite introductions, the incidence of any such events can be reduced by targeting human-biting mosquito populations merely because they are indeed biting people. Adult control of pestiferous populations prevents large numbers of mosquitoes from becoming physiologically "old." This minimizes the probability of being bitten by a mosquito that has fed on blood before.

3.3.6. Novel Disease Threats for Suffolk County

Novel mosquito-borne diseases for Suffolk County can be classified as those that are endemic but do not cause illness, and those exotic pathogens that may be introduced. Endemic diseases of concern include:

- Jamestown Canyon virus
- La Crosse virus

Exotic pathogens of concern include:

- Sindbis virus
- Rift Valley fever virus
- Japanese encephalitis virus
- Usutu virus

This list is not comprehensive, but is intended to identify reasonable prospects for introduction to Suffolk County, and to describe the impacts that could result if they began to transmit human disease.

3.3.6.1. Jamestown Canyon virus

Human disease associated with Jamestown Canyon virus infection represents a rapidly emerging arboviral zoonosis, a public health threat that may be underestimated. Following the original isolation of this arbovirus in 1961, few episodes of human disease were diagnosed, and little attention was paid to the frequent demonstration of seroconversion. Clinical disease began to be recognized during the 1980s (Grimstad et al., 1986), particularly in the north central US and in adjacent parts of Canada. Encephalitis is a prominent feature of this disease, and adults are affected as well as children. Transmission in the northeastern states, however, appears to be more intense than the frequency of reported cases would suggest (Grimstad et al., 1987). Various *Ochlerotatus* mosquitoes (Grimstad et al., 1987) as well as anophelines (DeFoliart et al., 1986) have been identified as vectors. The spring-hatch mosquitoes, *Oc. abserratus*, *Oc. canadensis*, and *Oc. cinereus*, appear to be the most important vectors of this infection (Andreadis et al., 1994). White-tailed deer serve as reservoirs (Watts et al., 1982), with about half of the Michigan herd acquiring infection each year (Boromisa and Grimstad, 1987). The sera of virtually all such deer react with antigen from Jamestown Canyon as well as Cache Valley virus in Michigan (Neitzel and Grimstad, 1991), while about a quarter of the general deer population in Connecticut are reactive (Zamparo et al., 1997). Risk of human infection

corresponds to the spatial and temporal distribution of these animals. The transmission season spans May through July (Grimstad et al., 1987).

3.3.6.2. La Crosse virus

La Crosse virus is endemic throughout much of the northeastern United States (Gerhardt et al., 2001). Some 70 cases of encephalitis due to this infection are reported to CDC each year, mostly in children younger than 16. Symptoms include fever, headache, stiff neck, lethargy, nausea, disorientation, mental confusion, and sometimes seizures. Infection in adults tends to be asymptomatic. Although transmission has, in the past, focused mainly in the Upper Midwest (hence the name), the range of this infection has recently extended toward the south, including the Carolinas and Tennessee. West Virginia reported more than half the total number of cases during the late 1990s. This change in distribution has been attributed to the recent invasion of the Asian tiger mosquito, *Aedes albopictus*. This mosquito was imported from Japan in used automobile tires in the vicinity of Houston, Texas, in 1984. It has even invaded Italy, where it is plaguing the citizens of Rome. This mosquito feeds avidly on people and small rodents such as chipmunks and squirrels. It is exceedingly competent as a vector for La Crosse virus (Kitron et al., 1998). Previously, the virus had perpetuated vertically (inherited infection) in the tree hole mosquito, *Ochlerotatus triseriatus*. Infected mosquitoes passed the virus to approximately half of their offspring. During the summer months, the cycle was amplified horizontally through chipmunks. *Ae. albopictus* can also inherit infection by this virus from their mothers. Their range has been extending and seems poised to include Suffolk County, as they have been found in Nassau County. Although *Oc. triseriatus* is native to Long Island, the closest La Crosse virus infections seem to occur in the Albany region. Another somewhat similar Asian import, *Oc. japonicus*, has recently invaded Suffolk County (Sardelis et al., 2002; Erwin et al., 2002). Its role in La Crosse transmission, however, has not been established.

The disease caused by La Crosse virus can be exceedingly burdensome. Although infection generally is silent (meaning no overt signs of illness), in some children lasting neurologic sequellae occur (Utz et al., 2003). Each case costs society at least $48,000 and some more than $3,000,000. Half of affected children experience seizures and more than one in 10 suffer from

gravely increased intracranial pressure (McJunkin et al., 2001; Balkhy and Schreiber, 2000). Loss of cognitive and behavioral functions may continue for more than a year.

Severe cases tend to cluster locally, perhaps in the vicinity of a breeding site in which a particularly virulent variant of the virus is being passed vertically (Kitron et al., 1997). *Oc. triseriatus* and *Ae. albopictus* deposit their eggs in the basal holes of hardwood trees, as well as in such artificial containers as discarded automobile tires. If such a breeding area is near a residence, generations of these mosquitoes (and any diseases they harbor) may consistently plague one family or neighborhood.

3.3.6.3. Sindbis virus

An outbreak of a novel disease became evident in Finland in 1981 when a single physician saw some 73 patients experiencing the novel syndrome caused by this pathogen (Turunen et al., 1998). This febrile disease is characterized by a diffuse rash and arthritis, with some patients becoming severely immobilized. The name Pogosta disease was applied to this syndrome, and Sindbis virus proved to be the etiologic agent (Kurkela et al., 2004). Discrete outbreaks were recorded in 1981 to 1982, 1988, and 1995. A startling 11 percent of Finns test positive for this pathogen (Laine et al., 2003). More than 2,000 identified cases have already been diagnosed in the country, with 18 cases per thousand residents occurring in Karelia Province (Brummer-Korvenkontio et al., 2002). Sindbis virus is hypothesized to be an important cause of rheumatoid arthritis in Scandinavia and Central Europe.

Sindbis virus may have been introduced into Finland from the Middle East. The pathogen first was recognized in Egypt during the 1950s and continues to circulate there (Turell et al., 2002). Various *Culex* mosquitoes serve as vectors, including the house mosquito, *Cx. pipiens*, and house sparrows appear to serve as the reservoir. Migratory birds are the likely vehicles of importation of this pathogen (Malkinson et al., 2001; Sammels, 1999). Sindbis virus seems a likely candidate for introduction into the northeastern US because it shares essential biological features with WNV and because it seems already to have been carried into Europe and Australia.

3.3.6.4. Rift Valley fever virus

Rift valley fever virus is a desert-adapted African pathogen that has been responsible for devastating epidemics following the occasional heavy rains or dam construction that result in

local flooding. The vector in these desert situations is *Aedes macintoshi*, a mosquito whose eggs can withstand decades of drying and whose reproductive tissues remain infected by this virus from generation to generation. Transmission in these desert situations is entirely vertical. Amplification by transmission through some avian or mammal host may be uncommon and would seem to be unnecessary. A severe illness, however, follows the bite of one of these infected mosquitoes, affecting people as well as cattle, goats, and sheep (Gerdes, 2002). The human disease includes an influenza-like illness, frequently accompanied by encephalitis or retinitis. In hoofed animals, abortion is frequent, as is hepatitis, particularly in the young.

Several outbreaks of Rift Valley fever have struck the Nile Valley of Egypt, first in 1979 and 1980, then in 1993, and again in 2001 and 2002 (Madani et al., 2004). No *Ae. macintoshi* are present in Egypt; instead, the virus cycles through house mosquitoes, *Cx. pipiens*, and a human reservoir. Nearly 1,000 cases were recorded in the more recent of the two outbreaks, and these people experienced fever, nausea, vomiting, abdominal pain, diarrhea, jaundice, encephalitis, severe bleeding, vision loss, and severe anemia. The mortality rate was 14 percent. These Egyptian outbreaks may have been imported when infected camels were brought in from Sudan (Abd el-Rahim et al, 1999). The potential for Rift Valley virus to invade new territories is illustrated further by a series of outbreaks that have occurred on the Arabian Peninsular in recent years. This pathogen, however, has not crossed the Mediterranean or spread across Gaza into Israel.

3.3.6.5. Japanese encephalitis virus

Japanese encephalitis is another candidate for introduction into the United States, and the Suffolk County environment appears generally suitable. It constitutes an important public health burden throughout much of Asia, affecting China, Southeast Asia, and the Indian subcontinent. This pathogen appeared for the first time in Australia, on western Cape York Peninsula in 1998. Subsequent study, however, failed to derive evidence that it has persisted there (Johansen et al., 2003). It can be controlled in populations through vaccination. However, where public health services are not extensive, or where the population is naïve, major impacts can result. Hundreds of people are reported to have died from an outbreak in India in the late summer of 2005 (WHO, 2005).

3.3.6.6. Usutu virus

This novel arbovirus, designated as Usutu virus, was recognized recently in Central Europe (Chvala et al., 2004). Before 2001, Usutu was known solely in southern Africa, where it is maintained in a cycle involving certain birds and *Culex* mosquitoes. This virus subsequently became endemic in Central Europe where it poses a severe environmental threat. Rodents appear to be accidental hosts, and the virus was isolated once from a person who was experiencing fever and a rash. No human neurological involvement, however, has yet been attributed to this virus. Massive mortality in birds followed its introduction into Austria. The European blackbird, (*Turdus merula*), an analog of the American robin (*T. migratorius*), is particularly vulnerable to this pathogen, and about a third of the population is said to have been lost each year. Many other European songbirds have suffered, as well. The vehicle of introduction of Usutu virus into Central Europe has not been identified.

As in Austria, Usutu virus seems a likely candidate for introduction into North America, and the residents of Suffolk County should be alert to the possibility that this threat to bird life may appear there. The circumstances that would permit transmission exist because *Cx. pipiens* mosquitoes appear to serve as vector and a variety of birds as reservoir.

3.4 Background Pesticide Uses and/or Exposures in Suffolk County

3.4.1 Introduction

There are many databases available that address pesticide use and exposure; however, each of these databases has their limitations. In addition, it is difficult to link these databases so that risks can be estimated. Use databases, such as the USEPA pesticides Industry Sales and Usage Market Estimates and the New York State Pesticide Sales and Use, quantify the amount of pesticides used but are of limited value in estimating actual risk, because these databases do not provide direct estimates of exposure. In addition, the amount of pesticide product used as opposed to active ingredient is reported. The quantities reported include inert ingredients or fertilizers that may constitute a much higher percentage than the active ingredient. CDC has conducted a biomonitoring study in which blood and urine samples were collected and analyzed for pesticides. This study provides evidence that exposure is occurring and may allow trends to be tracked over time. However, it is not known whether the levels detected result in health

effects. The New York State Pesticide Poisoning Registry attempts to document adverse effects from pesticide use, but is limited because exposure is not often quantified. There are many databases that attempt to address potential exposure by monitoring occurrence of pesticides in food or water. By making assumptions about consumption patterns, these databases are useful in estimating potential exposures, but do not document actual health impacts.

3.4.2 Background Exposures to Pesticides

According to a recent USEPA document, there are over 865 pesticidal active ingredients (i.e., ingredients that are responsible for pesticidal activity). Each of these active ingredients is formulated into many pesticide products and approximately 350 pesticides are used on food, in the home, and to protect pets (USEPA, 2003a).

In assessing the risk from exposure to pesticides, not only is the toxicity of the pesticide important, but also the manner in which exposure occurs and the population that is being exposed (e.g., young children or healthy workers). Exposure to pesticides can occur through contact with food, drinking water, indoor surfaces/furnishings, air in homes, lawns and soil outdoors, flea and tick products used on pets, and medical treatments such as lice shampoos. Children are a particular concern because they may be more susceptible to toxicity. In addition, children are likely to be more highly exposed due to their activity patterns (e.g., hand-to-mouth activity, activities closer to the ground, lawns, and carpets) and higher food consumption rates relative to their body weight (compared to adults). The use of pesticides in schools has been a local concern in New York State. In 2000, the Attorney General's Office issued a report on the use of pesticides in New York State schools. Of the schools that responded to their questionnaire, 87 percent used pesticides. 76 percent of the schools acknowledged using pesticides indoors and 63 percent used pesticides for outdoor purposes. Insecticides were used by 80 percent of the responding schools, and rodenticides and herbicides were used in 41 and 50 percent of the schools, respectively (Spitzer, 2000).

USEPA regulates the registration of pesticides and determines which products can be used, as well as the manner in which they can be used. USEPA requires testing on the efficacy of the product in controlling the pest of interest, as well as testing of its toxicity in order to address potential risks. This is to ensure that the pesticides "…do not pose unreasonable risks to human