In a two year feeding study of malathion to rats, female rats developed liver tumors at all tested doses ranging from 2.4 to 817 mg/k/g/day. In an 18 month feeding study of mice, an increased incidence of liver tumors was observed in the female mice fed the highest doses of malathion, 1707 and 3448 mg.kg/day (USEPA, 2000c). The conclusion drawn by USEPA to both studies is that liver tumors only occurred at excessive doses in these animals (National Pesticide Telecommunications Network, 2001). USEPA has classified malathion as having suggestive evidence of carcinogenicity, but not sufficient to assess human carcinogenic potential (USEPA, 2000c). IARC has categorized malathion as unclassifiable as to carcinogenicity in humans (http://monographs.iarc.fr/monoeval/crthall.html). A more recent study found that subcutaneous or intraperitoneal injections of malathion resulted in the formation of mammary tumors in rats. The results of this study prompted the authors to propose that alterations at the nervous system level, as caused by exposure to malathion, may alter the molecular pathways that initiate cellular proliferation leading to mammary carcinogenesis (Cabello et al., 2001). Follow-up work by these researchers have shown that malathion, when tested in breast cancer cell studies, is capable of altering cell proliferation and transformation (Cabello et al., 2003)

Malaoxon is a cholinesterase inhibitor that is metabolite of malathion. In a feeding study of this chemical in rats, malaoxon was not found to be carcinogenic. USEPA has determined that there is no evidence of carcinogenicity in male or female rats (USEPA, 2000c).

Hormonal Activity

Many breast cancer risk factors are hormone related. Epidemiologic and laboratory studies have implicated both exogenous (originating outside the body) and endogenous (produced within the body) sources of estrogen in the etiology of breast cancer. Thus, evidence of hormonal activity associated with any of the pesticides under consideration is included in this review.

The estrogenic potential (the ability to act like the hormone, estrogen) of certain pyrethroids has been evaluated in several ways. Using MCF-7 human breast carcinoma cell lines, sumithrin, fenvalerate, d-trans-allethrin, and permethrin were tested for hormone disruption. All demonstrated an ability to influence several cellular pathways, although not necessarily the same pathway (Go et al., 1999). Testing these same four pyrethroids in two other cell lines resulted in significant estrogenic activity identified for fenvalerate and sumithrin; however, none blocked the action of estrogen (that is, demonstrated estrogen antagonism) or acted as the steroid hormone, progesterone (Garey and Wolff, 1998). Similarly, several pyrethroids, including permethrin and deltamethrin, were found to induce MCF-7 cell proliferation and inhibit binding of estradiol to the estrogen receptor. Inhibition of estrogen receptor binding of estradiol by a compound indicates that the compound may interfere with normal hormonal activity (Chen et al, 2002). Deltamethrin was found to cause weak, but significant, MCF-7 cell proliferation, however, no estrogen receptor activation was observed (Andersen et al., 2002). A lack of estrogen receptor activation indicates that the compound may not work in the same way as naturally occurring estrogen. In contrast, the estrogenic or anti-estrogenic activity of several pyrethroids (d-transallethrin, cypermithrin, empenthrin, fenvalerate, imiprothrin, permethrin, d-phenothrin, and prallethrin) could not be demonstrated in a suite of in vitro assays based on human estrogen receptor alpha-mediated mechanisms (Saito et al., 2000). Similarly, in an in vivo test system used for the evaluation of endocrine disruptor activity, esfenvalerate, fenvalerate, and permethrin were not found to exert estrogenic, anti-androgenic, or adrogenic influences (Kunimatsu et al., 2002). Animal studies provide some evidence that exposure to pyrethroids may influence endocrine function, in that plasma testosterone levels were reduced in a rat feeding study of 39.66 mg/day of cypermethrin (Elbetieha et al., 2001) and decreased ovarian weight was observed in a study of female rats fed a daily dose of 23.98 mg fenpropathrin per kg (ATSDR, 2001). Thus, evidence both for and against the hormonal potential for pyrethroids can be found in the literature.

Among the remaining mosquito control pesticides under consideration, malathion was the only pesticide with any studies conducted for hormonal activity. An estrogenic potential evaluation of malathion in an E-screen, MCF-7 cell proliferation, and estrogen receptor binding tests found no estrogenic activity for this insecticide (Chen et al., 2002).

Epidemiological Links Between Breast Cancer and Pesticides Exposure

Epidemiologic studies of breast cancer with measures of exposure to the specific insecticides under consideration, whether through biomarkers, records of pesticide use/application, or self-report, have not been reported in the literature. The vast majority of epidemiologic studies conducted to date on specific insecticide exposure and breast cancer risk have focused on the organochlorine pesticides such as DDT, dieldrin and chlordane, or metabolites of these pesticides, such as the DDT metabolite DDE (dichlorodiphenyl dichloroethylene). However, the association between exposure to this family of pesticides and breast cancer risk is not relevant to this review, as the pyrethroids and malathion are not chemically or structurally similar in the least to these traditional organochlorine pesticides.

A large array of literature exists that assesses cancer incidence and pesticide exposure through occupation in industries such as agriculture or pesticide application. The majority of these studies have focused on men, due to the small proportion of women employed in these industries. When possible, many have included women in their study populations, and have assessed breast cancer as an outcome. However, the relevance of these studies is limited, as the specific identification of exposure to the particular mosquito control insecticides of interest is difficult, if not impossible, to infer. Although a job title of farmer or agricultural worker may mean that the study participant was exposed to pesticides on the job and may even provide some indication of the class of pesticides, there is a high likelihood that the person was exposed to many different pesticides, not a single product. Thus, use of this type of exposure determination prevents the identification of an association between specific pesticides and breast cancer. They can indicate whether the potential for such an association exists.

Nonetheless, this occupation-based literature is the only epidemiologic literature currently available for use in evaluating whether insecticides used for mosquito control may increase breast cancer risk. In sum, none of the 19 investigations reviewed (Table 7-32) found any significant increased risk of breast cancer incidence or increased breast cancer mortality associated with:

- working on a farm or in an agricultural industry
- being the spouse of a farmer
- being a resident of a farm
- holding a job as a pesticide applicator.

Many of these investigations suffer from a lack of control for potential confounding factors as well as potential exposure misclassification. Additionally, given the time periods covered by many of these studies, there is reduced potential for the pesticide exposures experienced by the study participants to be among the mosquito control pesticides that are being evaluated here.

Even with these limitations, it is unlikely that any large or even moderate increases in risk are being obscured.

Table 7-32. Summary of epidemiologic studies of pesticide exposure and breast cancer risk

Study design and population	**Exposure**	**Measure of association**	**Variables adjusted for confounding**
Wiklund 1983 Retrospective cohort - Sweden, Cancer-Environment registry - 354,228 involved in agriculture, 36,711 females - follow-up 1961-1973 - 444 cases	Female agricultural workers	O/E[b] 99% CI[c] 0.81 (0.71-0.91)	Age and sex
Olsen 1987 Population-based linkage study - Denmark - 18,403 cases in all occupational categories diagnosed 1970-1979 - 152 cases in agriculture; 112 cases in electrical manufacturing	Agriculture, hunting, forestry and fishing	SPIR[e] 95% CI 101 (86-118)	Age and calendar year
Ewertz 1988 Population-based case-control - Denmark - 1694 cases diagnosed 3/1/83-2/29/84, <70 years - 1705 controls, age stratified sample	Women's occupation Home, farming Husband's occupation Farming	OR[f] Farmers used as reference category - all other occupations OR>1.00 Farmers used as reference category - all other husband's occupations OR$\geq$1.00 except shop sales (OR=0.98)	Age, place of residence, parity and both factors in table
Kato 1990 Standardized Proportional Ratio - Aichi, Japan, Cancer Registry, - General population of Aichi prefecture estimated from 1980 and 1985 census data - 103 cases, diagnosed 1979-1987, >30 years	Female agricultural workers	O/E p 0.73 <0.01	Age according to age distribution of cancer patients
Franceschi 1993 Case-control - Northeastern Italy - 132 cases diagnosed 1985-1991, <80 years - 968 controls	Female farmer	RR[g] 95%CI 0.8 (0.5-1.3)	Age, smoking and alcohol

Study design and population	**Exposure**	**Measure of association**	**Variables adjusted for confounding**
Rubin 1993 Nested case-control and Proportionate mortality study - USA - Mortality database maintained at NIOSH (covers 23 states), represents 2.9 million death certificates - 59,196 breast cancer deaths in all occupational groups - 197 cases in farming occupational group, 124 white and 73 black diagnosed 1979-1987 - 59,196 controls randomly selected from all white women who did not die from breast cancer or malignancies of the female reproductive system freq-age matched by 5-yrs	Farming, forestry and fishing occupational group White Black	OR 95% CI 0.84 (0.66-1.07) PMR[h] p 75 <0.01 61 <0.01	None reported
Costantini 1994 Cross-sectional - Italy - 579 cases, 21 in agriculture industry, died 1981-1982, 18 to 64 years - 2,038 deceased and economically active in all occupational groups	Employed in agriculture industry	MOR[i] p 57 0.02	
Wiklund 1994 Prospective cohort - Sweden, 1970 Swedish Population and housing census - 50,682 women worked ≥20 hrs/week in agriculture - Expected number of cases calculated on basis of annual cancer incidence among women in 5-yr age groups - Follow-up 1/1/71 to 12/31/87 or death - 1,159 cases	Female agricultural worker	SIR[j] 95% CI 0.83 (0.78-0.88)	

Study design and population	**Exposure**	**Measure of association**	**Variables adjusted for confounding**
Cantor 1995 Nested case-control - USA - Mortality database maintained at NIOSH (covers 24 states) - 33,509 cases, 29,397 white and 4,112 black, died between 1984-1989 - 117,794 controls (non-cancer deaths), 102,955 white and 14,839 black, frequency matched for age (5-yr), gender and race	Exposure to insecticides scale (0 to 3) 0 1 2 3	Whites: OR(1) OR(2); Blacks: OR(1) OR(2) 0: 1.0 1.0 1.0 1.0 f 1: 1.19 1.42 0.58[k] 0.57[k] 2: 1.07 1.07 ------- ------- 3: ------- ------- ------- -------	Age and SES[l] (1) all probability levels of exposure (2) excluding women with low probability of exposure
Kristensen 1996 Retrospective Cohort - Norway - Farm holders or their spouses (253,624 total; 113,949 women) born after 1924 - Follow-up 1969 to 1991 or death or emigration - 598 cases engaged in agriculture; 148 cases working ≥500 hrs/yr on a farm	Women engaged in agriculture Women working ≥500hrs/yr on a farm	SIR 95% CI 105 (96-113) 84 (72-99)	None reported
Folsom 1996 Population-based prospective cohort - Iowa, USA - 36,295 randomly selected women in 1986 - follow-up through 1992 - 934 cases	Lived on a farm	RR 95% CI Age-adj 0.96 (0.82-1.13) Multi-adj 1.03 (0.87-1.23)	Age, smoking, body mass index, waist-to-hip ratio, education, physical activity, marital status, alcohol use, family history of breast cancer, reproductive characteristics
Pukkala 1997 Retrospective cohort - Finland - 85,151 women farmers on 12/31/78 - follow-up 1/1/79 to 12/31/93 or death or emigration - 1,474 cases	Finnish female farmer	SIR 95% CI 0.77 (0.73-0.80)	None reported
Avnon 1998 Case-control study - Israel - 734 women members of 3 kibbutzim 1989 - 7 cases of breast cancer	Female member of agricultural settlement	Kibbutz O/E p A 3/3.25 >0.05 B 2/1.69 >0.05 S 2/0.93 >0.05	None

Study design and population	**Exposure**	**Measure of association**	**Variables adjusted for confounding**
Fleming 1999 Retrospective cohort study - Florida - 3,503 female licensed pesticide applicators (1975-1993) - 26 breast cancer cases	Female pesticide applicator	SIR 95% CI 0.61 (0.40, 0.90)	Age and calendar year
Simpson 1999 Proportional incidence study - England and Wales - 381,915 women diagnosed with cancer through UK cancer registry (1971-1990) - 93,951 breast cancer cases	Agricultural occupation	Only reported occupations with significantly increased cancer rates - No pesticide-related occupations and no agricultural occupations were found to have elevated breast cancer rates	Age and social class
Settimi 1999 Hospital-based case-control study - 5 areas in Italy - incident cancer cases among residents of catchment area admitted to 8 collaborating hospitals (1990-1992) - reference group drawn from other cancer sites excluding lung cancer, cancers of the female reproductive systems and women with previous oophorectomy - 67 premenopausal and 192 postmenopausal breast cancer cases - 72 premenopausal and 424 postmenopausal controls	Job title of farmer or farm laborer	OR 95% CI Premenopausal 1.4 (0.5, 3.4) Ever employed 1-9 yrs 1.2 (0.3, 4.1) 10-19 yrs 0.4 (0.0, 4.6) 20+ yrs 2.9 (0.5,16.2) Postmenopausal 0.4 (0.3, 0.7) Ever employed 1-9 yrs 0.9 (0.4, 2.3) 10-19 yrs 0.8 (0.3, 2.1) 20+ yrs 0.4 (0.2, 0.8)	Premenopause Use of oral contraceptives alcohol consumption, family history of breast cancer, parity Postmenopause Age at menarche, age at last menstruation, hormone replacement therapy, alcohol consumption, age at first birth, BMI

Study design and population	**Exposure**	**Measure of association**	**Variables adjusted for confounding**
Duell 2000 Sub-study from a population-based case control study (eligibility was reported farming) - Eastern and Central North Carolina - Invasive breast cancer cases diagnosed 1993-1996 - Controls selected from motor vehicle roster (<65) and Health Care & Finance roster (65+) - Randomized recruitment to achieve 50% African-American and 50% under age 50 -327 cases and 381 controls who reported farming participated -451 cases and 409 controls reported that they never lived or worked on a farm	Reported having lived or worked on a farm	OR 95%CI Ever farmed 1.0 0.8-1.2 Duration (yrs) 1-10 1.2 0.8-1.7 11-17 0.8 0.5-1.2 18-23 0.7 0.8-1.1 >23 0.6 0.4-0.9	Age, race, age at menarche, parity/age at first birth, lactation, current body size, education, duration of smoking, alcohol consumption, family history of breast cancer, oral contraceptive use, duration of laundry for pesticide user
Wang 2002 Retrospective cohort study - New York State - 6,310 female farm residents (1980-1993) - 141 breast cancer cases -622,268 comparison female residents of non-urbanized areas	Female farm resident (farmer or adult relative sharing same last name of farmer who had been a Farm Bureau member)	SIR 95% CI All 0.89 (0.75, 1.05) 30-49 yr 1.17 (0.85, 1.58) 50-69 yr 0.80 (0.64, 0.99) 30-49 yr 0.82 (0.43, 1.47)	None
Fleming 2003 Retrospective cohort study of mortality - United States National Health Interview Survey - 208,855 women (1986-1994) - 7 female pesticide applicators and 1,718 female farmers	Female pesticide exposed worker	RR 95% CI 0.4 (0.1, 1.5)	Age

[b] observed/expected
[c] confidence interval
[d] standardized mortality ration
[e] standardized proportionate incidence ration
[f] odds ratio
[g] relative risk
[h] proportionate mortality ratio
[i] mortality odds ratio
[j] standardized incidence ratio
[k] 95 percent CI excludes 1.0
[l] soicioeconomic status

7.9.2.4. Special Considerations Regarding Potential Toxicity to Children

There is some limited information available concerning direct linkages between the identified mosquito control chemicals and childhood illnesses.

Pyrethrins and pyrethroids can interfere with normal neurological function. In adults, short-term, high level exposure to these insecticides may cause:

- dizziness
- headache
- nausea
- muscle twitching
- reduced energy
- changes in awareness
- convulsions
- loss of consciousness

It is likely that the same effects would be experienced by children exposed to high levels of pyrethrum (ATSDR, 2001). Overall, pyrethrins and pyrethroids reportedly pose low chronic toxicity to humans, the most common problems resulting from the allergenic properties of pyrethrum (EXTOXNET 1994). In rats, developmental exposure to pyrethroids has been shown to have long-lasting effects causing neurobehavioral and neurochemical deficits in adulthood. Inhalation of pyrethroid-containing mosquito repellants early in a rat's life has been shown to cause damage to the blood brain barrier, which may indicate that early life exposure to these chemicals could lead to adverse neurological effects (Sinha and Shukla, 2003).

Piperonyl butoxide is reported to have low to very low toxicity when ingested, inhaled or absorbed through the skin by mammals (National Pesticide Telecommunications Network, 2000).

Malathion is an organophosphate pesticide that inhibits cholinesterase activity. Cholinesterase is an enzyme that removes the chemical neurotransmitter acetylcholine from the junctions between nerve cells. Cholinesterase serves as the nervous system's "off switch" and is essential to the normal function of the nervous system. USEPA requires neurotoxicity testing, both acute and sub-chronic, in animal studies for all pesticides submitted for registration by USEPA. All studies reviewed by USEPA were found to be acceptable and to have met the agency's guidelines (USEPA, 2000c). ATSDR reported that children who have accidentally swallowed or had skin contact with high amounts of malathion experienced symptoms such as:

- difficulty breathing
- chest tightness
- vomiting
- cramps
- diarrhea
- watery eyes
- salivation
- sweating
- headaches
- dizziness
- loss of consciousness
- death

(ATSDR, 2003)

Note that "high amounts of malathion" was not defined in the ATSDR toxicological profile summary of health effects of malathion exposure in children. Very young animals have been identified as more susceptible to the effects of malathion than older animals. Rapid medical treatment of high level exposure to malathion prevents long-term effects, and low level exposure appears to pose few or no health problems (ATSDR, 2003).

Malaoxon (metabolite of malathion) is a cholinesterase inhibitor that is metabolite of malathion. USEPA did not identify any acute toxicity testing of the malaoxon but calculated that this malathion metabolite appears to have approximately 10 to 30 times greater toxicity (USEPA, 2000c).

Based on epidemiological studies discussed in Section 3, there is evidence to support a positive relationship between pesticide exposure and childhood leukemia, lymphomas, and brain cancer. However, due to the lack of focus in research on individual pesticides, and the imperfect measures of exposure assessment, it is difficult to identify which substances are safe for future use. No conclusions can be drawn with respect to the specific mosquito control pesticides that are under consideration

There is biologic plausibility for an association between organophosphate pesticides, such as malathion, and asthma or other respiratory symptoms. Organophosphate pesticides may contribute to respiratory problems through cholinesterase inhibition. Cholinesterase is an enzyme that removes the chemical neurotransmitter acetylcholine from the junctions between never cells. Cholinesterase serves as the nervous system's "off switch" and is essential to the normal function of the nervous system. Decreased cholinesterase can cause impairment of a physiological regulatory mechanism of the autonomic (or involuntary) control of airways (Eskenazi et al., 1999), which may promote constriction of the bronchial air passages (Hoppin et al., 2002). Small increases in wheezing were found for malathion use in Ethiopian homes (Yemaneberhan et al., 1997).

Pyrethroids act on the sodium ion channels, which are involved in the control of the sensory nervous system. Stimulation of these channels causes their prolonged opening resulting in sensory neurons to be stimulated. This action is suspected to be related to the paresthesia symptoms associated with pyrethroid intoxication (Narahashi, 1992). Paresthesia symptoms are those that cause a sensation of pricking, tingling, or creeping on the skin having no objective cause, and usually are associated with injury or irritation of a sensory nerve or nerve root. A biological explanation for the development of respiratory symptoms due to pyrethroid exposure has not been proposed.

Nonetheless, one case report of long-term occupation exposure to tetramethrin (a pyrethroid insecticide) may have led to asthmatic symptoms (Vandenplas et al., 2000) and an accidental exposure to aerosol pyrethroid insecticide resulted in respiratory problems and an asthma attack (Muller-Mohnssen, 1999).

A case-series of Japanese children and adults with long-term exposure to malathion through helicopter spraying linked reported neuro-opthalmological symptoms to the reported exposure (Ishikawa et al., 1993). However, no rigorous epidemiologic investigation was conducted so that no association between malathion exposure and this neurological problem can be drawn.

There are clear links between pesticide exposure and childhood cancers. Some studies trace other childhood illnesses to malathion or pyrethroids that are not included in the Long-Term Plan. However, there is no link between the selected pyrethroids or malathion to childhood cancers, especially at the exposures associated with mosquito control. The linkages between malathion and asthma or neurological impacts in children are not compelling, and neither is the tenuous link between pyrethroid exposure and asthma.

7.9.2.5. Long-Term Plan Field Work Results

The Caged Fish study looked at three aspects of resmethrin behavior in the environment. These results, and pertinent results of parallel of earlier efforts, will also be discussed. In addition, County efforts to detect this chemical in dry deposition studies will be briefly discussed.

In short, resmethrin proved to be difficult to find in the environment, even immediately after applications. It apparently degrades extremely swiftly. Its synergist, PBO, could be detected for some time after applications, and at higher concentrations. Resmethrin appeared to have no impacts on shrimp or fish, either in the natural environment or laboratory simulations. The following details some of the results.

Caged Fish Experimental Results (Adulticide)

The original plan for this study called for all field work to be conducted prior to the beginning of August to avoid anticipated low DO events that are more prevalent during the hottest period of the summer. Unfortunately, due to many delays in obtaining permission to conduct the study, this was not possible. Preliminary data on caged fish and shrimp survival at all sites showed good survival during July. However, by the time the fully replicated study was performed, this was not the case.

The static renewal studies conducted in the laboratory, which used water collected 30 minutes post application from each site, had excellent survival in exposed shrimp.

Taken all together, these data do not present consistent evidence of toxicity due to resmethrin to the exposed organisms. It is not clear, however, whether the pesticide applications in conjunction with other stressors, such as low DO conditions, might not result in greater mortality than would have occurred without pesticide applications. This experiment was not able to separate impacts from low DO and the pesticide applications. The notion that pesticides can have synergistic impacts in conjunction with other stressors is a current research topic that is attracting much interest, but without many published results (see Arnold and McLachlan, 1996; on the contrary, see Hayes et al., 2006).

The absence of acute mortality due to resmethrin exposure is not terribly surprising given its short presence in aquatic settings. The pesticide appears to degrade extremely rapidly, as it was not detectable in any sediment samples. Because it so quickly degrades, the exposure of organisms to this toxic compound is too short (and, data suggests, at too low a concentration) for any negative impacts to occur.

Resmethrin Fate and Transport

As discussed in Section 6, the sampling efforts associated with the Caged Fish experiment appear to show that resmethrin has a relatively short lifespan in the environment. Sampling showed that the resmethrin to PBO ratio rapidly increased following release of the pesticide. Since it is assumed there is no PBO source, it seems that the resmethrin is degrading. Efficacy studies (see below) show that it is effective at eliminating mosquitoes, but the sampling shows an apparent loss of the compound by the time it is deposited on, for instance, a water surface. There the ratios of resmethrin to PBO can be hundreds of times lower than was the case in the original formulation released from the helicopter. It rapidly becomes undetectable at the low part per trillion/high part per quadrillion range. This is well below concentrations shown to have impacts to organisms. For example, the McElroy laboratory calculated LC_{50}s for grass shrimp at approximately 600 parts per trillion for a 96-hour exposure to scourge (resmethrin and PBO combined), and 1.2 parts per billion for resmethrin without the synergist. The concentrations measured only two hours after application were either not detectable (less than five parts per trillion, or 500 parts per quadrillion), or, when detected, in the five to 20 part per trillion range. Resmethrin does not appear to be transported to sediments in any measurable quantity, either.

All of this suggests it is not possible to expose organisms to concentrations of this pesticide at the amounts that have been shown to cause impacts.

Caged Fish Benthic Sampling

Johns Neck received larvicides and adulticide applications in 2004, but Timber Point received no adulticide applications in 2004. Therefore, the combined statistical analysis of treatment sites against control sites does not determine the impact of adulticide treatments alone.

The pairwise comparisons often found significant differences between one control site, Havens Point, and Johns Neck for at least one analysis approach. Several others were very close to being determined to be significant. However, this may be explained by substrate differences, which apparently did exist between the sites. It is clear that differences in sediment types leads to differences in benthic invertebrate populations (Cerrato et al., 1989).

That significant differences were also found between the two control sites under some of the statistical tests suggests the pesticide applications were not the cause of differences in populations and abundances. Therefore, this experiment does not find any impacts to the tested benthic invertebrate populations associated with adulticide use, although the finding is certainly not as compelling as was the case with the larvicides.

County Dry Deposition Sampling

SCDHS has worked since 2001 to develop a reliable method of collecting deposition data associated with adulticide events. It has been difficult to find a good method. The best method developed using chilled glass trays (standard pyrex baking trays). Resmethrin was found to be extremely sensitive to light, and to be very volatile. Volatility varied with temperature, and so current County procedure involves setting out experimental apparati (where the pans are set in coolers over ice) immediately prior to an application, and collecting them again within two hours of the end of the application (to minimize loss of the chemical. The trays are kept cold until they are analyzed. Despite these precautions, field spike recoveries tend to be in the 50 percent range, suggesting there is still major loss of any deposited compounds. In addition, work by RTP in conjunction with the risk assessment deposition modeling suggested that the chilled trays may create a microlayer of cold, denser air within the tray area that may deflect the very fine pesticide droplets and inhibit deposition (the droplets tend to be small enough to be controlled by atmospheric forces and diffusion, rather than gravity).

The difficulties involved in this effort spotlight the ephemeral nature of the resmethrin applications. Because it degrades so quickly, and is so volatile, it is difficult for it to impact any part of the environment other than the immediate atmospheric area that the spray plume traverses. This necessarily limits any potential impacts to terrestrial or aquatic organisms.

7.9.2.6. Efficacy of the Current Program

General Studies

New insecticides are tested in wind tunnels, where they are applied at different rates to caged mosquitoes. The volume that is effective is compared to some known insecticide, such as malathion. This is how the manufacturer determines optimal applications rates. Resmethrin, for example was found to be seven times (for *Psorophora spp.*) to 50 times (for *Anopheles spp.*) more toxic than malathion in such tests. Field testing found that, based on 36 studies involving species from *Aedes*, *Psorophora*, *Culex*, and *Anopheles* species (this was prior to the creation of *Ochelerotatus*), that malathion itself killed 95 percent of caged mosquitoes when applied at rates ranging from 0.01 to 0.326 pounds of active ingredient per acre (lbs AI/ac). Resmethrin also achieved 95 percent kill rates against members of the same four genuses at rates ranging from 0.001 to 0.078 lbs AI/ac., based on 11 studies. Sumithrin had two tests in this data set where 95 percent kill rates were measured, for *Anopheles* and *Psorophora* mosquitoes, both at a 0.0012 lbs AI/ac application rate (Mount, 1998).

However, it should be understood that it is actually the droplet size that determines the effectiveness of the pesticide against mosquitoes. Particular sizes of droplets carry appropriate amounts of pesticide to kill a single mosquito. The amount of pesticide applied over an area is intended to provide a cloud of droplets that ensures that all flying mosquitoes encounter a droplet. If the size of the droplets is appropriate, this ensures that the mosquitoes will be killed. If the droplets are too small, then the amount of pesticide may not be enough to kill mosquitoes. If the droplets are too large, then not only is the dose inefficient, but the presumably, if the application amount has been optimized, there will be fewer droplets and therefore the possibility exists that some mosquitoes will not encounter any pesticide. Generally, optimal droplet sizes range from seven to 25 um volume median diameter (Mount, 1998). However, there are usually many more droplets made than there are mosquitoes in the target zone. One estimate is that as little as 0.0001 percent of the pesticide applied actually impinges on mosquitoes (Pimental, 1995), although this conclusion was reported without any accompanying documentation.

The effectiveness of an adulticide application therefore depends on the droplet sizes generated by the applicator, meteorological conditions (as winds and other atmospheric forces can disperse the pesticide cloud), and the physical setting where the mosquitoes are – as the presence of buildings, trees, and other obstacles can reduce the chances that the pesticide will hit a mosquito before it contacts something else (Mount, 1998; Mount, 1996).

A review of aerial applications of malathion reported reductions in caged mosquitoes ranging from 0 percent to 100 percent, depending on the location of the cage in relation to the targeted area, in the 18 hours following applications. Most caged mosquitoes in the area directly targeted by the spray experienced 95 percent or more mortalities. The paper concluded that ULV applications are "highly efficacious," given vagaries of environmental factors (Mount, 1996). However, applications can fail to achieve mosquito control because of dose, meteorology, or environmental problems associated with the application. Tests with less than 95 percent kill rates are rather common, but most are explicable in terms of these factors. It should also be noted that adulticide use can fail to meet its goal of mosquito control not only because of application problems, but also due to rapid reinfestation of the area (Mount, 1998).

Recent general evaluations of these products have been made in various jurisdictions. In Ontario, Canada, malathion was found to have acceptable efficacy for use as an adulticide (pyrethroids are not licensed for use in Canada) (Shapiro and Miccucci, 2003). Delaware determined that resmethrin was more effective than sumithrin for ground-based applications (Lesser, undated[2]). New York City has found sumithrin to be effective for its purposes (NYCDHMH, 2004). New Jersey, in its annual advisory of allowable mosquito control pesticides, found resmethrin, sumithrin, and malathion acceptable for ground applications, and malathion and resmethrin acceptable for aerial applications, based upon evaluations of "safety, economy, and efficiency" (Brattsten and Sutherland, 2005).

It should be understood that, although it is likely to have a high correlation, reduction of mosquito numbers alone is not sufficient to ensure reductions in disease risks (Hopkins et al., 1975). However, use of adulticides is recommended as part of a comprehensive program to reduce disease risks, albeit as the last resort due to the potential for adverse human or ecological impacts (Rose, 2001; CDC, 2003).

Suffolk County Efficacy Tests

Suffolk County's New Jersey light trap network is sited and primarily used to detect failures of larval controls, such as water management and larvicide applications. The traps are not placed or serviced so as to produce good data for adulticide effects. Therefore, special efforts must be made to determine impacts from adulticide applications.

Malathion was no longer used for aerial applications in Suffolk County after 1999. Sumithrin was used in 2000, and resmethrin after 2001 (no aerial applications were made in 2001). A special sampling effort was made using CDC light traps to test the efficacy of a sumithrin application in 2000 in Babylon. Three traps were placed in the control area; each was sampled twice pre-application, including the night before the insecticide was applied, and then the night following the application. The post-application counts data were compared to the mean pre-application counts. They also were compared to a reference site. At the reference site, the trap counts increased after the application date. Therefore, the post-application counts at the treatment sites were compared to the "expected" counts that might have been encountered if no treatment had occurred, and the populations at all four sites had varied consistently (Table 7-33).

Table 7-33. Babylon Sumithrin Efficacy Test, July 31-August 5, 2000 (Treatment Date, August 4)

Trap	Mean Count, Pre-application	Post-Application Count	"Expected" Post-Application Count	Raw Percent Control	Adjusted Percent Control
Horse Ranch					
Culex pre.	18	8	31	56	74
Total females	55.5	24	187	57	87
Lower Belmont					
Culex pre.	17	4	29	77	86
Total females	26.5	5	89	81	94
Upper Belmont					
Culex pre.	9.5	3	16	68	82
Total females	41.5	8	140	81	94
Bergen Point (ref.)					
Culex pre.	453.5	781			
Total females	511	1718			

Culex pre = *Cx. pipiens* + *Cx. restuans*

CDC light trap data for aerial applications in 2003 were accessed. These data, for resmethrin applications, are presented in Table 7-34.

Table 7-34. 2003 Selected CDC Light Trap Data

Trap	Trap Location	Date of Application	Mosquito Count Before Application	Mosquito Count After Application	Percent Reduction
BHL Goldengate	Mastic-Shirley	8/28	447	201	55
BHL WF181PK	Mastic-Shirley	8/28	611	78	87
SDL Bayview	Southold	8/27	195	27	86
SML Blydenburg	Hauppauge	8/26	129	116	10

The 2000 Babylon data show control efficacies (using the adjusted percentages) of 75 to 94 percent. These are similar to other data sets reported for sumithrin nationwide, and show that adulticide use in the field can achieve large reductions in mosquito populations. The 2003 aerial applications of resmethrin had more variable data, with reductions ranging from 10 percent (ineffective control) and 55 percent (moderate control) to nearly an order of magnitude reduction in two instances. Together, these two local data sets show that large reductions of mosquitoes can be achieved in practice – but not always, even when demonstrably effective insecticides are applied.

7.9.2.7. Relative Effects on Mosquito-borne Disease

Controlling mosquito-borne disease is not simply the product of adulticides usage. Disease risk management is the result of all the actions associated with the Long-Term Plan, and will be discussed in detail in Section 7.11.

However, the proposed adulticide program should be similar in impacts under Health Emergency conditions as the current approach is. There are some elements that may lead to improved management of disease. These include:

- For WNV, *Cx. pipiens* may be better controlled through better targeted applications of adulticides for viral control, in terms of timing and application means, in fresh water environments where trapping indicates that *Cx. pipiens* is the dominant mosquito present.

- For EEE, when the risk profile warrants (if there are large numbers of bridge vectors in the vicinity of an amplification center), adulticides will be applied to reduce the risk of human or equine cases, and, although the reduction of EEE risks may be an unintentional by-product, if warranted salt marsh mosquitoes will be controlled using adulticides; this appears to reduce the chances that this very efficient potential vector of EEE is not allowed to infect people.

The two steps outlined above should also assist in reducing risks associated with novel diseases.

7.9.2.8. Resistance Management

Resmethrin appears to be the best choice as an adulticide for the Long-Term Plan. It is effective, and does not appear to have as many potential environmental impacts, even if they are of short duration and limited scope, as permethrin and malathion appear to have. Sumithrin and natural pyrethrum have not been demonstrated to degrade as quickly as resmethrin (at least, in local settings). This means that the Long-Term Plan has preferentially identified resmethrin as the adulticide of choice; sumithrin will be used on Fire Island, since the resmethrin label does not offer the opportunity for hand-held applications.

Reliance on one insecticide always presents the specter of pest resistance development. Resistance develops when individuals with a certain heritage find that these traits make them less susceptible to the insecticide. If these individuals can create a breeding population, they can begin to reinforce the traits and ensure that they are inherited by future generations. Further applications of the pesticide to this population merely serve to remove individuals without the genetic advantage against the pesticide, and so results in further cross fertilization among the resistant individuals. Soon, a substantial portion of the pest population may become resistant, even if the traits that provide resistance are less advantageous in other ways.

However, there appear to be reasons that the chances of resistance developing to resmethrin are minimal in Suffolk County. With the exceptions of certain areas on Fire Island, most areas of the County that receive adulticide applications are only sprayed once or twice in a season; some areas, such as certain parts of Mastic-Shirley, may receive four or more applications in a year; and some parts of Fire Island have received up to a dozen applications in a year. The small area of the County that receives adulticide applications in any year (on the order of five percent of the County), and the even smaller portion of the County that receives multiple applications, means that only a small portion of the County's adult mosquito population is ever exposed to adulticides. In addition, the applications of adulticides are not made in the marshes where the larvae are, but rather in the human habitations where the biting mosquitoes are. This, along with the large areas where no control occurs, means there are large pools of untreated mosquitoes to dilute any resistant populations that develop. Therefore, just as it is impossible that the Long-Term Plan would eliminate mosquitoes from the County, it is also extremely unlikely that resistance to resmethrin will develop.

Resistance to sumithrin in the higher application areas of Fire Island might seem more plausible. However, some research and a great deal of personal observations on Fire Island indicate that the National Seashore areas (where control is not undertaken) are the sources of the mosquito infestations in the communities. This means, similarly, that source areas are not being sprayed, and will probably continue to be a source of untreated mosquitoes that overwhelms any resistant populations that may develop.

Nonetheless, prudence dictates that these observations be verified by actual science. Resistance testing is called for in the Long-Term Plan, using resources either at Rutgers or one of the larger mosquito control companies, to ensure that using a single pesticide for adult control does not lead to ineffective pesticide use.

7.10. Impacts of the Long-Term Plan: Part 8, Management Structure

The Long-Term Plan calls for a management structure composed of two elements. One would be administered by SCDPW, and other would be part of SCDHS operations.

SCVC would remain as part of SCDPW. SCVC would be responsible:

- Education and outreach: website maintenance, operational spray efficacy reporting, Wetlands Strategy Plan, convening annual seminar between educators and field crews
- Surveillance: population surveillance, including associated laboratory work
- Source reduction: response to citizens' complaints, storm water structure treatments, fish management
- Water management: planning and execution of work in accordance with the Wetlands Management Plan, generate annual Wetlands Strategy Plan

- Larval control: surveillance data interpretation, notifications, operational control; efficacy testing
- Adult control: surveillance data (population) interpretation, decisions regarding non-Health Emergency actions, operational control; efficacy testing
- SCVC would prepare the Annual Plan of Work.

SCDHS would be responsible for the following:

- Education and outreach: public education efforts, some website maintenance, no-spray list maintenance and outreach (Public Health Division); triennial report preparation (Office of Ecology)
- Surveillance: disease surveillance, including associated laboratory work (ABDL)
- Source reduction: no major responsibilities
- Water management: review of any ditch maintenance proposal, review and monitoring responsibilities for major projects (Office of Ecology)
- Larval control: no major responsibilities
- Adult control: surveillance data (disease) interpretation, review of decisions regarding non-Health Emergency (ABDL); decisions regarding Health Emergency actions (Public Health Division)

The Office of Ecology would review and assist on the Annual Plan of Work.

This management structure allows for decisions to be made by appropriate personnel:

- operational decisions remain the responsibility of operational managers
- mosquito population determination and decisions relating strictly to population issues are the province of SCVC
- health-related issues require input (and in many cases) decisions by managers in the Department of Health Services.
- the Office of Ecology provides a degree of environmental input into SCVC operations.

This structure maximizes departmental strengths (SCDPW is a department that implements physical projects for the County, and maintains those kinds of responsibilities here, and SCDHS has a strong public outreach capability and background), and provides a clear delineation of responsibilities across the various aspects of the Management Plan.

The analysis of regional programs, discussed In Section 2 above, found that the best management structures for vector control agencies were those where separate funding was possible, by being a separate authority. In terms of maximizing funding potential, this is not doubt true. However, by being part of the general County government, greater cost controls and oversight are possible. It is believed that the degree of public visibility of the program, especially as augmented through better public outreach efforts under the Long-term Plan, will ensure that the fiscal controls are not so efficient as to impede the functioning of the program.

The selected management structure should serve to enhance all aspects of the Long-Term Plan as it strives to reduce public health impacts from mosquito-borne disease, reduce pesticide impacts, and ensure that water management actions improve mosquito control efforts and result in wetlands restorations.

7.11. Impacts of the Long-Term Plan: Part 9, Risks from Mosquito-borne Disease

7.11.1. Relative Effects on Mosquito-borne Disease

Researchers from the Harvard School of Public Health have created an analytical model of mosquito transmission. This model can forecast impacts from mosquito-borne disease under specified conditions. It is derived from principles of mosquito ecology and disease transmission. However, in order for the model to be properly run, a great many variables need to be specified. Under almost all conditions, many (or even most) of the values for the variables are not known.

This was the case for Suffolk County, despite its robust surveillance program and the wealth of data generated by the program over the past several decades. The kind of data generated by SCVC and the ABDL are intended to inform mosquito control efforts, and did not meet the Harvard model data needs. The researchers believed that the process of matching model output to known disease conditions in the County would invariably require making judgments on issues such as whether the local mosquito ecology or mosquito control efforts were responsible for the apparent reductions in disease risk experienced by the County. They had hoped that the data sets would resolve these issues adequately, but that was not possible. Because many of the assumptions that would be necessary for the model to run invariably determined the questions of interest under the modeling approach, it was determined to be pointless to make the effort to create a Suffolk County specific model. Therefore, independent assessment of disease risk under various control approaches proved impossible to determine quantitatively.

It does seem possible to discuss the impacts of control programs in a qualitative sense. In order to accomplish this, the apparent effects of control on mosquito-borne disease will be discussed for the current program. A comparison of the expected impacts under the Long-Term Plan to the current program will then be undertaken.

7.11.2. Mosquito-borne Disease Impacts under the Current Program

For Suffolk County, it seems likely that the impacts of WNV are less than might have occurred if there was no control program. Potential disease impacts with no control will be discussed in Section 9, and a complete comparison of the potential impacts under the Long-Term Plan as compared to alternatives will be discussed in Section 13. For now, it is just to be understood that the disease impacts from WNV were much greater in other areas of the country than they have been in Suffolk County.

This strongly suggests that there is something different between the conditions in the County and areas that experienced greater impacts from WNV. It seems likely that the differences could be attributable to differences in mosquito ecology, or differences in mosquito control, or a combination of the two.

It is possible that differences in mosquito ecology could be important. Although most of the areas that were used for the comparison do not have important West Nile virus vectors such as *Cx. quinquefasciatus* (present in western parts of the country) and *Cx. tarsalis*, as is the case in parts of the country to the south and west, it is quite probable that *Cx. salinarius* is more prevalent in these comparison areas than it is in Suffolk County. *Cx. salinarius* prefers to breed in brackish water, and these habitats are not common in Suffolk County[1], partly because the permeable soils here do not lead to as much runoff as is the cases in places such as Connecticut,

[1] 2005 season surveillance in Suffolk County found more *Cx. salinarius* than in other years. Distinguishing between various *Culex* species is always difficult, and it is not clear if the 2005 data represents a shift in local populations or changes in identification proficiency in the Suffolk laboratories.

where *Cx. salinarius* is much more prevalent. In fact, *Cx. salinarius* has been identified as the most probable bridge vector for most cases of WNV in Connecticut (Andreadis et al., 2004). On the other hand, Connecticut does not have the numbers of *Oc. sollicitans* that are found in Suffolk County, and that mosquito can, at the high numbers found in the County, have a relatively high risk factor for disease transmission.

Relative risks for mosquito species as vectors of WNV can be computed using a model developed by the New York State Department of Health (Kilpatrick et al., 2005). Using the model shows that indeed *Cx. salinarius* appears to present the greatest degree of relative risk for Connecticut. It carries approximately three times the relative risk of *Cx. pipiens* for transmitting WNV (see Table 7-35); although *Cx. pipiens* is usually considered to be the dominant vector of WNV in the northeast US (Anderson et al., 2004).

Table 7-35. Model of the relative risk for WNV transmission, based on Connecticut mosquito distributions

Species	Relative Abundance (percent)	MIR[1]	Vector Competence	Percent Mammal Meals	Relative Risk	Percent of Total Risk
Ae. vexans	15.9	0.05	0.17	86	0.12	4.4
Cq. perturbans	22.0	0.01	0.11	83	0.02	0.8
Cx. pipiens/ restuans	7.1	0.95	0.38	19	0.48	18.6
Cx. salinarius	7.3	0.85	0.36	67	1.50	57.4
Cs. melanura	7.4	0.17	0.28	11	0.04	1.5
Oc. canadensis	22.3	0	0.55	100	0	0
Oc. japonicus	0.5	0.33	0.93	95	0.16	6.1
Oc. sollicitans	2.4	0.07	0.16	100	0.03	1.0
Oc. trivittatus	15.2	0.05	0.55	64	0.27	10.2

1. MIR = minimum infection rate, which relates the minimum number of infected mosquitoes that would produce the positive mosquito pools results. Mosquitoes are tested in pools of individuals, so a positive pool of 10 mosquitoes would correspond to a MIR of 0.10; a positive pool of seven mosquitoes would have a MIR of 0.14; a negative pool of eight mosquitoes would have a MIR of 0; and a positive pool of 10 mosquitoes, a positive pool of seven mosquitoes, and a negative pool of eight mosquitoes would lead to a MIR of 0.08.

Using recent trap data for Suffolk County, where use of methoprene as a larvicide resulted in great reductions of *Oc. sollicitans*, the same model finds that *Cx. pipiens* is the mosquito species with the greatest degree of relative risk for Suffolk County (Table 7-36). The recently introduced species, *Ochlerotatus japonicus*, due to its competence as a vector despite its relatively low numbers, also is a major risk for disease transmission. *Oc. sollicitans* also has a low but not negligible portion of the risk distribution.

Table 7-36. Model of the relative risk for WNV transmission, based on Suffolk County 2004 mosquito distributions (effective larval control of salt marsh mosquitoes)

Species	Relative Abundance (percent)	MIR	Vector Competence	Percent Mammal Meals	Relative Risk	Percent of Total Risk
Ae. vexans	27.5	0.05	0.17	86	0.20	7.3
Cq. perturbans	13.0	0.01	0.11	83	0.01	0.4
Cx. pipiens/ restuans	17.2	0.95	0.38	19	1.18	42.8
Cx. salinarius	0	0.85	0.36	67	0	0
Cs. melanura	2.5	0.17	0.28	11	0.01	0.5
Oc. canadensis	1.3	0	0.55	100	0.00	0.0
Oc. japonicus	3.3	0.33	0.93	95	0.95	34.5
Oc. sollicitans	34.6	0.07	0.16	100	0.39	14.1
Oc. triviattus	0.6	0.05	0.55	64	0.01	0.4

However, at least some of the risk reduction might relate to effective mosquito control. It was related that the introduction of methoprene to Suffolk County had a large impact of *Oc. sollicitans* numbers (Campbell et al., 2005). A reconstruction of pre-1995 WNV relative risks is shown in Table 7-37.

Table 7-37. Model of the relative risk for WNV transmission, based on Suffolk County 1994 mosquito distributions (before very effective larval control of salt marsh mosquitoes)

Species	Relative Abundance (percent)	MIR	Vector Competence	Percent Mammal Meals	Relative Risk	Percent of Total Risk
Ae. vexans	7.9	0.05	0.17	86	0.06	2.2
Cq. perturbans	7.1	0.01	0.11	83	0.01	0.2
Cx. pipiens/ restuans	29.1	0.95	0.38	19	2.00	75.2
Cx. salinarius	0	0.85	0.36	67	0	0
Cs. melanura	1.2	0.17	0.28	11	0.01	0.2
Oc. canadensis	2.1	0	0.55	100	0	0
Oc. japonicus	0	0.33	0.93	95	0	0
Oc. sollicitans	52.5	0.07	0.16	100	0.59	22.1
Oc. triviattus	0.1	0.05	0.55	64	0	0.0

The salt marsh mosquito does have more potential as a vector in that population distribution. In addition, some criticisms of this model point out that the percent mammal meals listed for *Cx. pipiens* is much larger than others have suggested (Apperson et al., 2002); however, it has also been suggested that *Cx. pipiens* changes its feeding habits later in the season (Spielman, 2001), or that hybridization between subsets of the mosquito that specialize in either human- or bird-feeding has led to the severity of West Nile virus in the US (Fonseca et al., 2004). If the percentage were lower, the relative risk associated with *Cx. pipiens* would decrease proportionately, and its share of overall risk would decrease (with other species' shares increasing). In addition, trap counts may not accurately reflect the distribution of human-biting mosquitoes (Anderson et al., 2004), although they are designed to mimic attributes of warm-blooded animals that attract mosquitoes for meals. This means that the data the model depends on may not be entirely reliable.

It is not responsible to directly compare the different data sets for Connecticut and Suffolk County, however. They merely represent degrees of relative risks. Actual risks associated with these data would depend on whether or not virus is actually present to create the opportunity for illness, and the numbers of mosquitoes present. For example, if overall mosquito numbers in Connecticut are actually much less than on Long Island, the actual degree of risk presented by *Cx. pipiens* here might be equivalent to the risk posed by *Cx. salinarius* there. In addition, if mosquito control efforts in the 1990s actually decreased the numbers of all mosquito species, not just salt marsh mosquitoes, then the risk of disease represented by "natural" distributions of mosquitoes, especially *Oc. sollicitans*, might have been much greater than current distributions and numbers show.

The data are suggestive, however, that there is a difference in the mosquito ecology between Connecticut and Suffolk County. If the difference in speciation is not accompanied by vastly different numbers of mosquitoes, the different ecology would alter risks for disease transmission significantly. In Connecticut, the comparative risks computed for *Cx. salinarius* were three times those of *Cx. pipiens*, for example. Since the mosquito that has the greatest degree of risk in Suffolk County (*Cx. pipiens*) only has one-third of the relative risk of *Cx. salinarius* (albeit in Connecticut conditions), overall disease risk in Suffolk County might be considered to be much lower due to differences in mosquito ecology.

However, unpublished sampling results from 2005, focusing on distinguishing among the various *Culex* species, found a much higher incidence of *Cx. salinarius* than had been anticipated (S. Campbell, SCDHS, personal communication, 2006). This suggests its abundance might in fact be much greater than has been hitherto calculated, and the differences in mosquito ecology between Suffolk County and Connecticut might be much less than initially described.

Another way of examining the issue is to compare actual patterns of disease across the two different areas. West Nile virus has followed a similar course in Connecticut as in Suffolk County. There have been relatively few cases, and most occurred in 2002 and 2003. Connecticut in composed of eight counties; four are on the coast and four are inland. It might be argued that coastal Connecticut is more similar to Long Island than the state as a whole is, although neither comparison is especially apt. USGS maps of WNV incidence by County led to the following description of WNV in Connecticut. WNV incidence was defined as a bird testing positive for WNV, a mosquito pool testing positive for WNV, or a human or equine case of WNV. Table 7-38 shows the results of the analysis (see Figure 7-11 for a map of Connecticut). For Suffolk County, the zip codes where these conditions were recorded were mapped for 2000 to 2004 (Figures 7-12 to 7-16). To simplify the work, it was assumed that no one in Suffolk County was exposed to the disease in 1999, and only Fairfield County was exposed in Connecticut (Mostashari et al., 2001b). All members of a zip code were assumed to have been exposed if it met the criteria, and the degree of exposure for all "positive" zip codes was assumed to be the same. Population for Suffolk County as a whole for 2000 to 2004 was assumed to be constant, using the 2004 population generated by ESRI with the GIS zip code coverage. Connecticut populations were set at 2000 Census levels. Table 7-39 shows the results of this analysis.

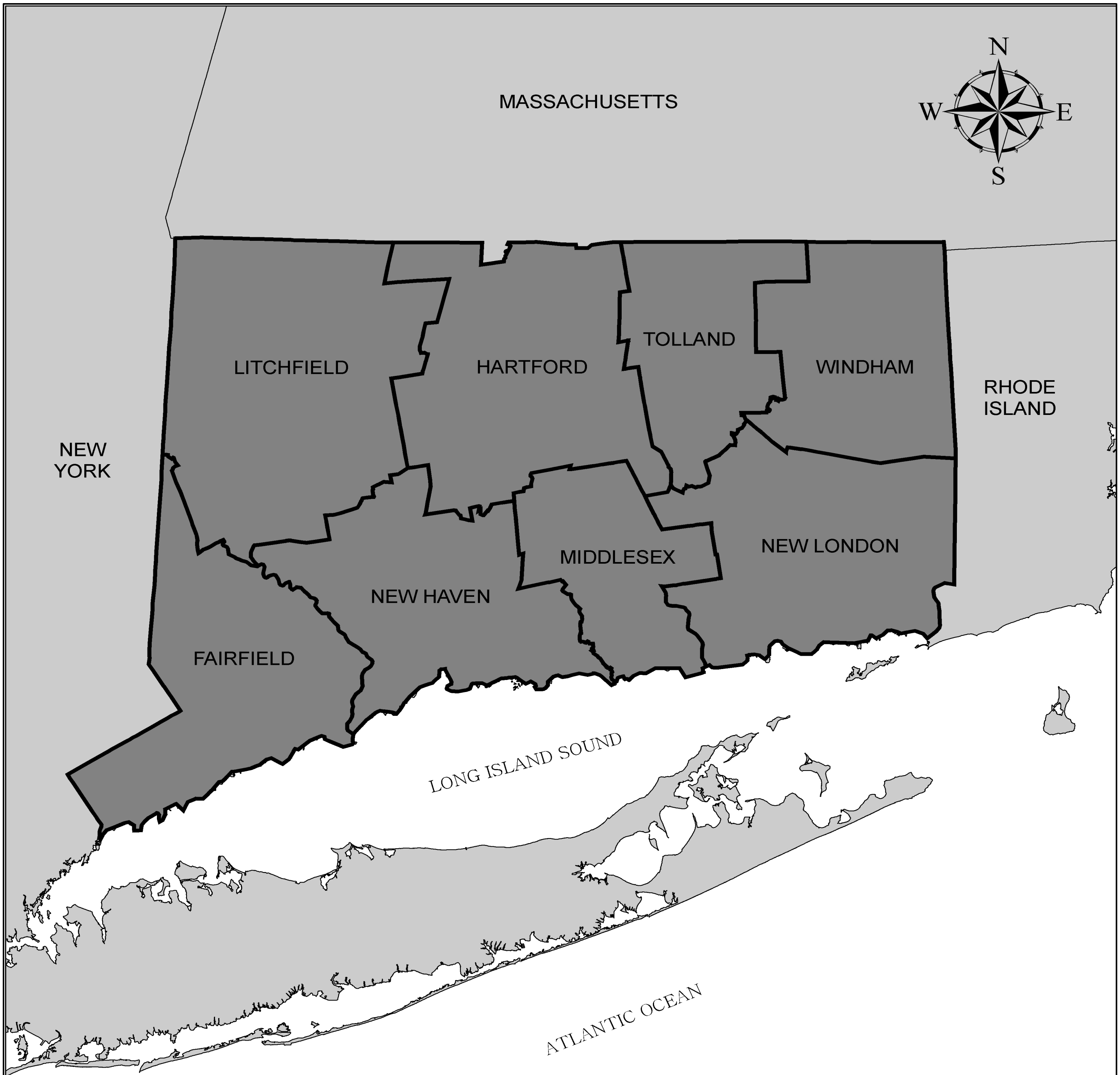

FIGURE 7-11
DRAFT GENERIC ENVIRONMENTAL IMPACT STATEMENT
CONNECTICUT COUNTIES

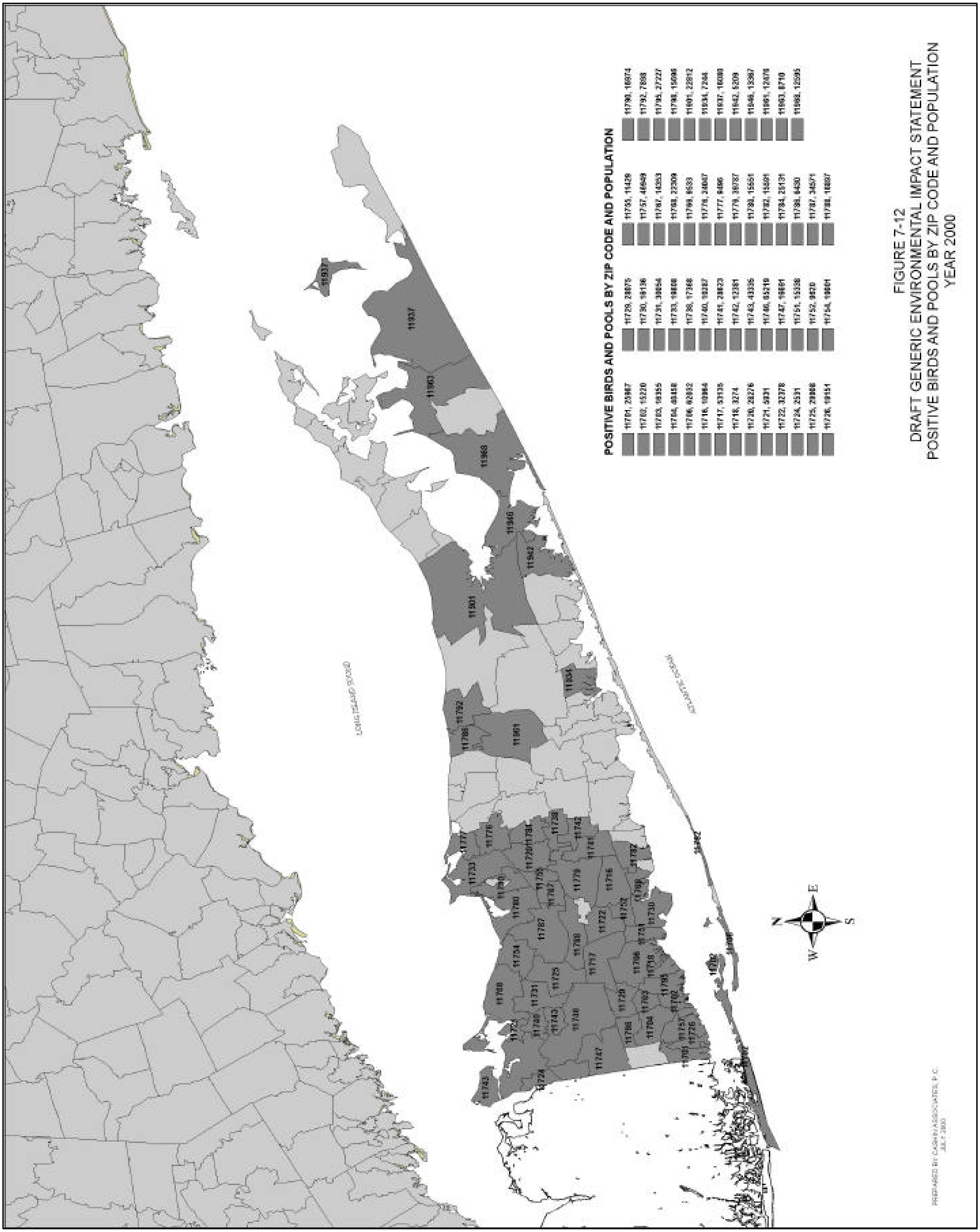

FIGURE 7-12
DRAFT GENERIC ENVIRONMENTAL IMPACT STATEMENT
POSITIVE BIRDS AND POOLS BY ZIP CODE AND POPULATION
YEAR 2000

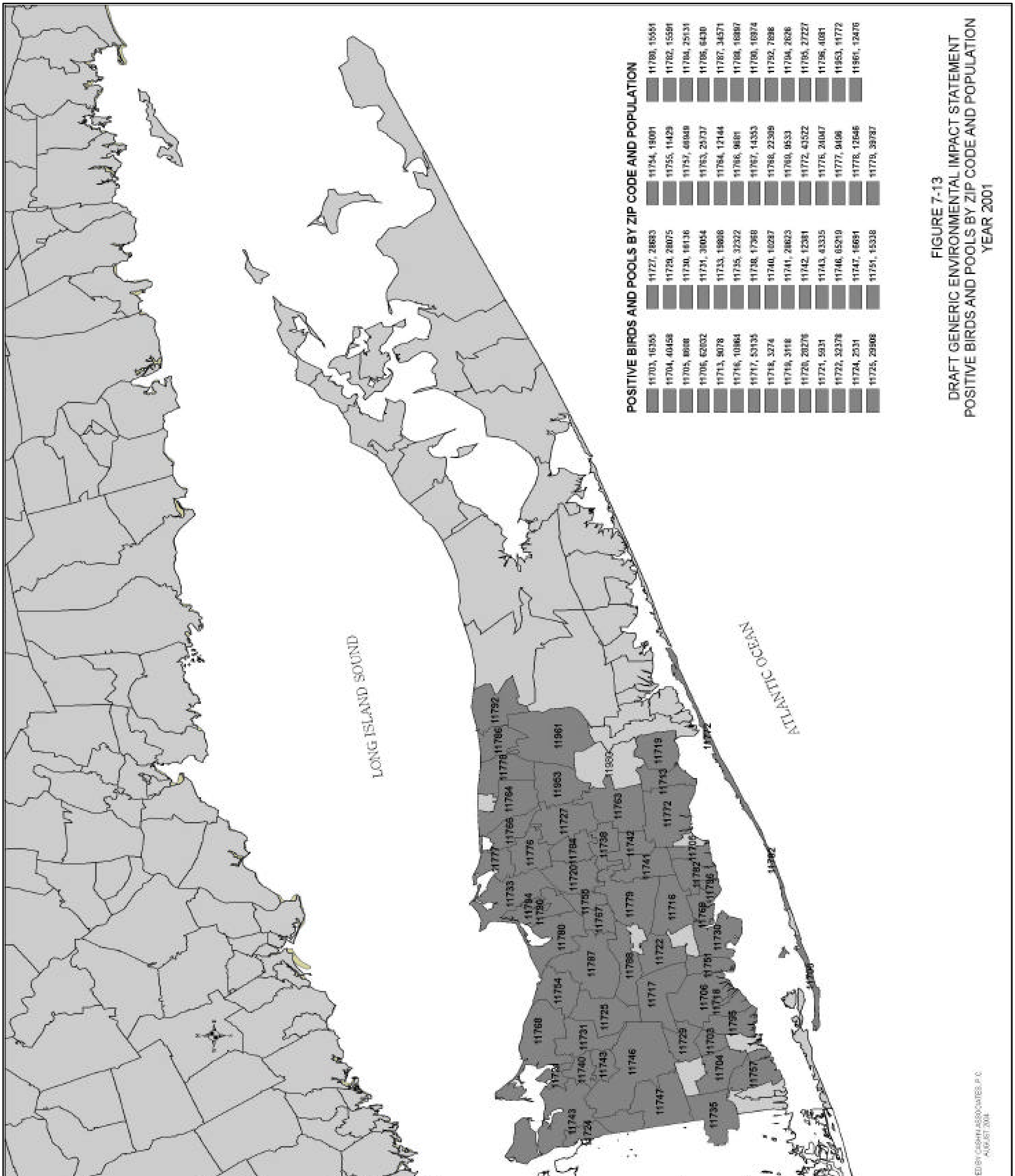

FIGURE 7-13
DRAFT GENERIC ENVIRONMENTAL IMPACT STATEMENT
POSITIVE BIRDS AND POOLS BY ZIP CODE AND POPULATION
YEAR 2001

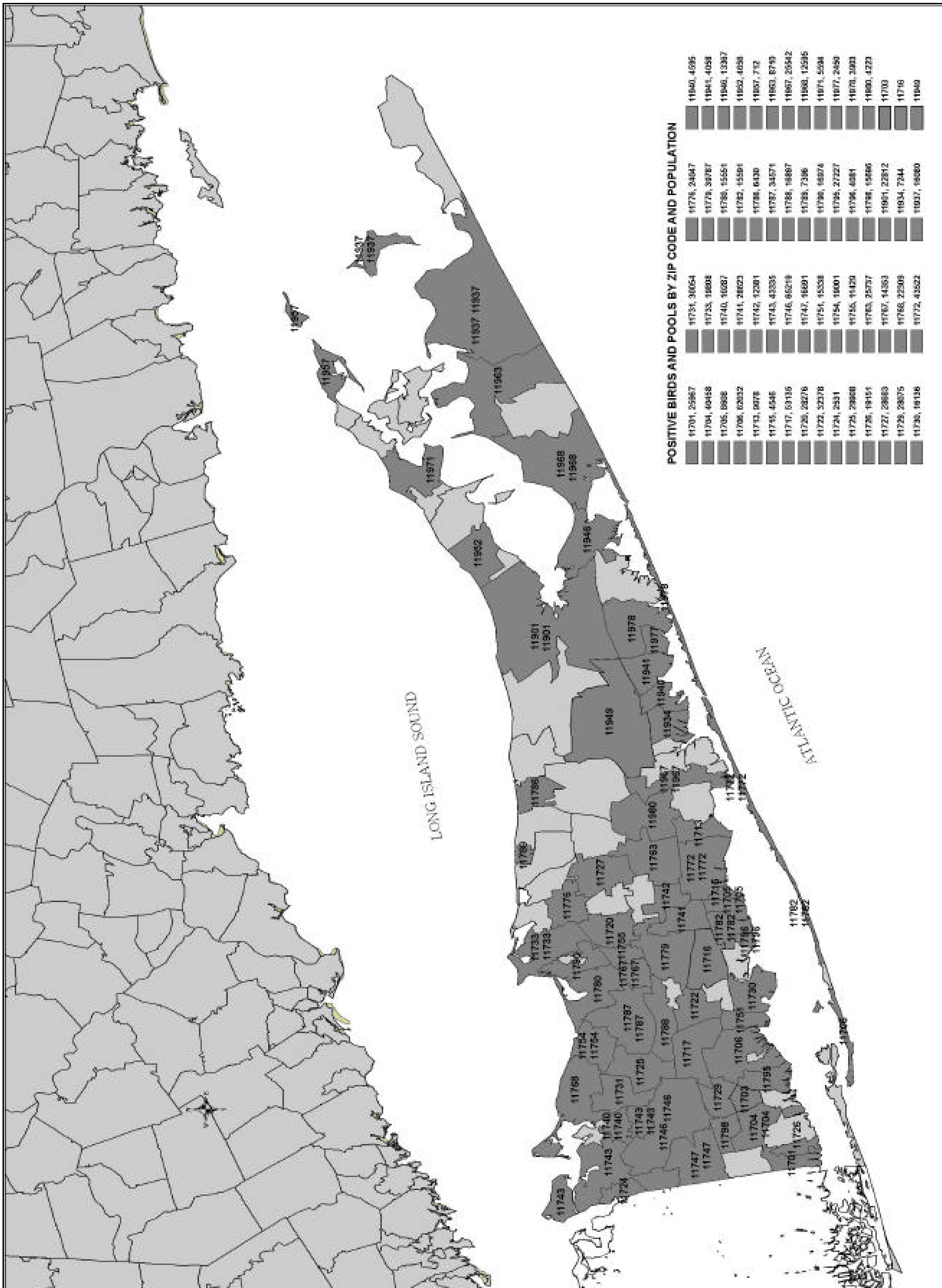

FIGURE 7-14
DRAFT GENERIC ENVIRONMENTAL IMPACT STATEMENT
POSITIVE BIRDS AND POOLS BY ZIP CODE AND POPULATION
YEAR 2002

PREPARED BY CASHIN ASSOCIATES, P.C.
JULY 2005

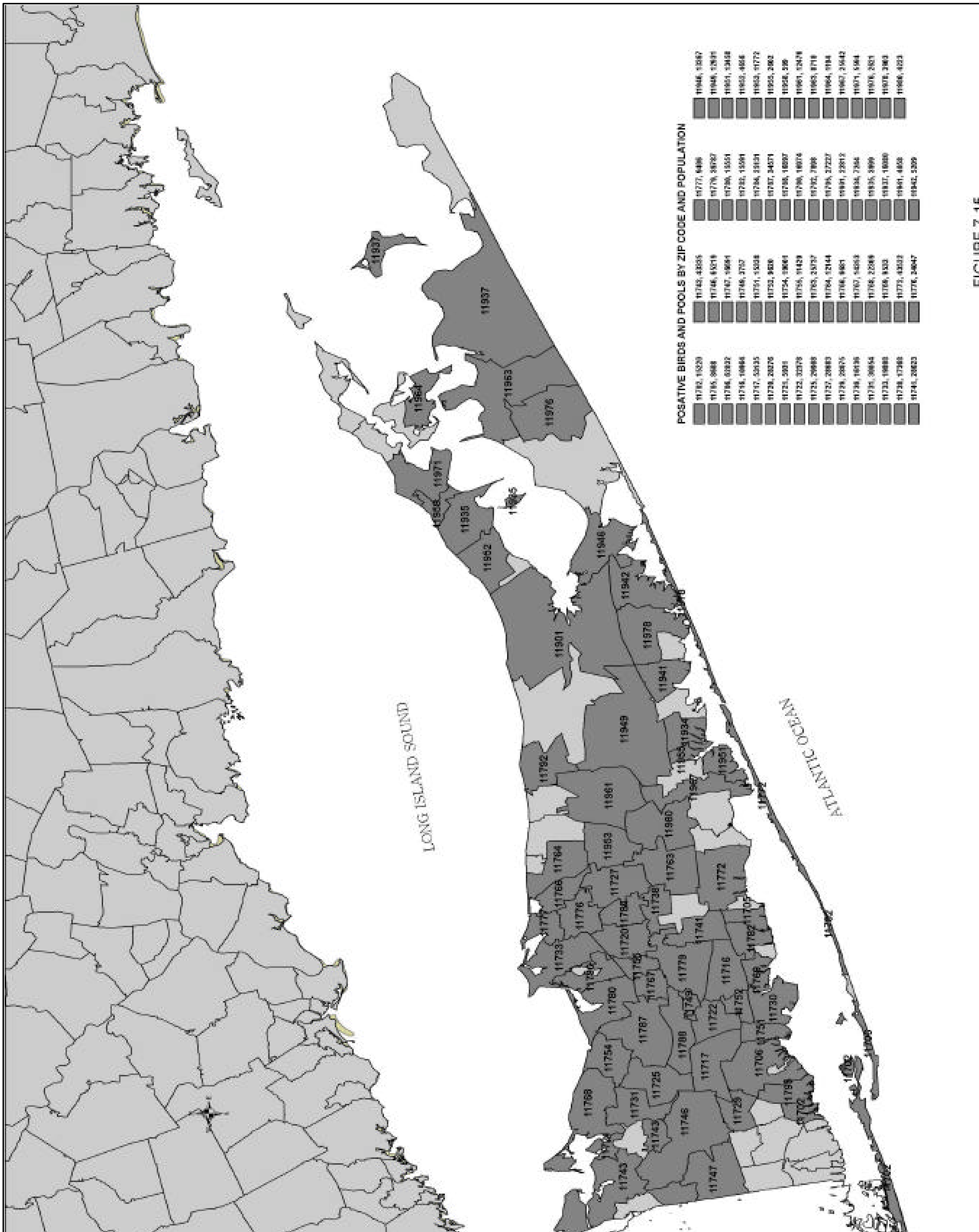

FIGURE 7-15
DRAFT GENERIC ENVIRONMENTAL IMPACT STATEMENT
POSITIVE BIRDS AND POOLS BY ZIP CODE AND POPULATION
YEAR 2003

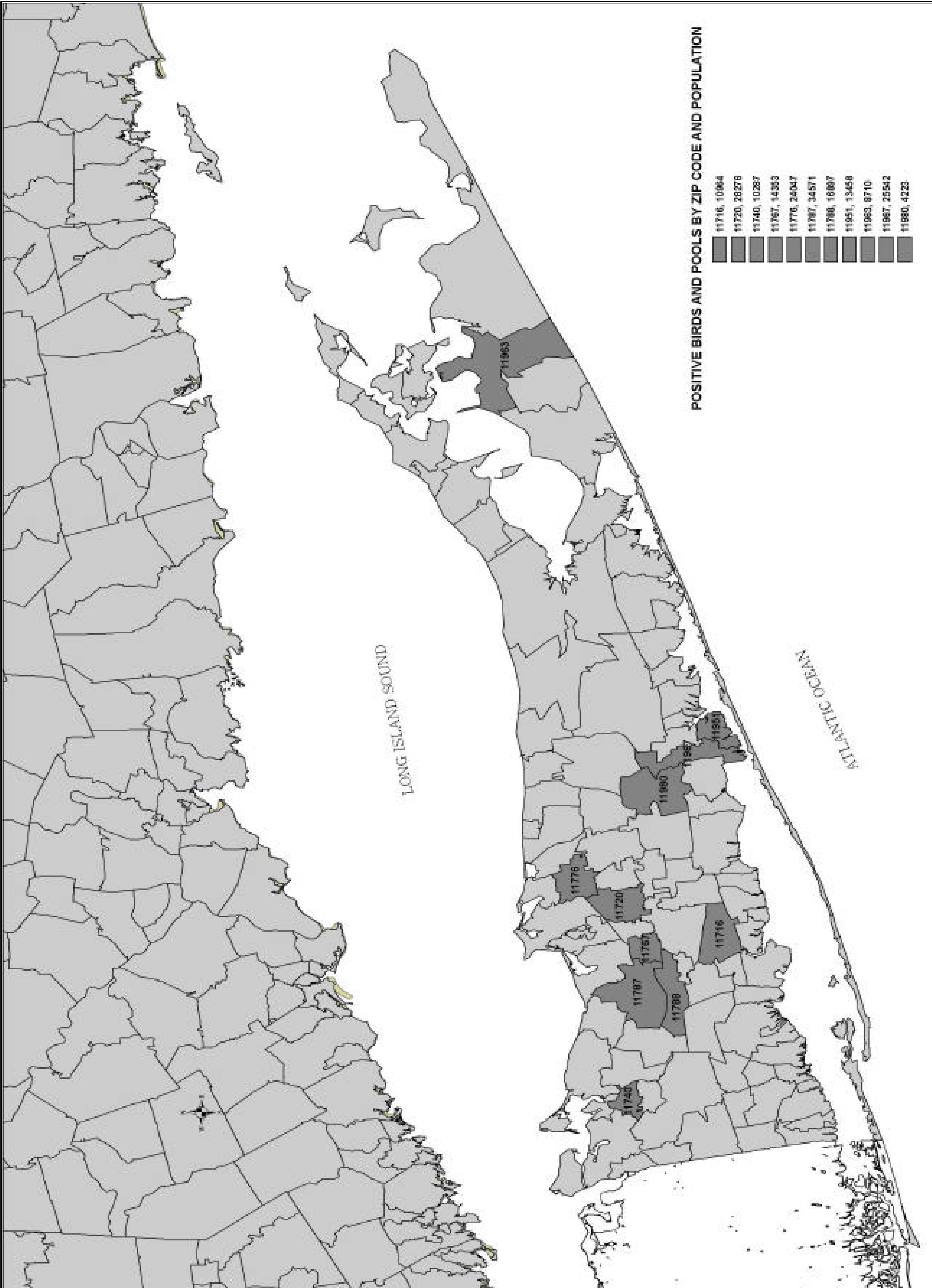

FIGURE 7-16
DRAFT GENERIC ENVIRONMENTAL IMPACT STATEMENT
POSITIVE BIRDS AND POOLS BY ZIP CODE AND POPULATION
YEAR 2004

Table 7-38. Connecticut WNV Exposure

County	Population	1999	2000	2001	2002	2003	2004
Fairfield	882,567	X	X	X	X	X	X
Hartford	857,183		X	X	X	X	X
Litchfield	182,193		X		X	X	X
Middlesex	155,071		X	X	X		
New Haven	824,008		X	X	X	X	X
New London	259,088		X	X	X	X	
Tolland	136,364		X	X	X	X	X
Windham	109,091		X	X	X	X	X

Table 7-39. WNV cases and exposures in Connecticut and Suffolk County, 1999-2004

	Population	1999	2000	2001	2002	2003	2004
Connecticut	3,405,565	0	1	6	17	17	1
Four Coastal Counties of Connecticut	2,120,734	0	1	3	11	12	1
Four County Exposed Populations		882,567	2,120,734	2,120,734	2,120,734	2,120,734	1,706,575
Suffolk County	1,482,824	0	0	1	8	10	0
Suffolk County Exposed Population		na	1,135,878	1,195,260	1,168,088	1,227,931	191,328

Comparisons can then be made between the rates of cases occurring in the two areas (Table 7-40).

Table 7-40. Rates of WNV cases (per million population) in Connecticut and Suffolk County, 1999-2004

	1999	2000	2001	2002	2003	2004	Mean Rate	Maximum Rate
Connecticut	0	0.29	1.76	4.99	4.99	0.29	2.06	4.99
Four Coastal Counties of Connecticut	0	0.47	1.41	5.19	5.66	0.47	2.20	5.66
Four County Exposed Populations	0	0.47	1.41	5.19	5.66	0.59	2.22	5.66
Suffolk County	0	0	0.56	4.62	5.49	0	1.78	5.49
Suffolk County Exposed Population	na	0	0.84	6.85	8.14	0	3.17	8.14

The similarities of disease variation across time in both areas are striking. It should be noted that the differences in the mean rates are not statistically significant. In addition, it should be understood that the zip code definition is too fine a scale to compare the Suffolk County data to the Connecticut data, as it may be that certain zip codes in the Connecticut counties also did not report positive birds of mosquito pools. If Connecticut data had been tested on a zip code scale, infection rates would probably have been higher, because county-wide data sets minimize infection rates by including people who do not fit the definition used for exposure in the Suffolk County zip code analysis.

Comparing county-wide Suffolk County data to the Connecticut data finds that infection rates in Suffolk County were less than or equal to Connecticut rates, however computed, each year. In fact, the mean infection rates for Connecticut were 10 to 20 percent higher than the county-wide Suffolk County mean rate. The difference could be attributable to differences in mosquito ecology, discussed above. Or the differences could be due to different mosquito control programs.

Connecticut's water management program emphasizes more progressive means of water management than does the current Suffolk County program. Officials there claim that larvicide use is eliminated in marshes that have undergone progressive marsh restoration. Connecticut has an aggressive larvicide program in both fresh water and salt water marshes. The State does not adulticide, although specific towns may choose to use adulticides.

Suffolk County has focused much of its control program on salt marsh mosquitoes, through ditch maintenance, larviciding, and some adulticiding. With WNV has come increased efforts to control *Cx. pipiens*, but the focus of Suffolk County's arbovirus program has mostly been reductions of potential bridge vectors.

It is difficult to clearly partition the responsibility for Suffolk County's decreased rate of WNV illnesses between mosquito control and mosquito ecology, especially since mosquito control is responsible for parts of the mosquito ecology. This was another sticking point in the Harvard effort to model the County's conditions. Nonetheless, the similarity between the Suffolk County data and those for to Connecticut are striking. It is also notable that these rates are very different from those where no or ineffective mosquito control occurred during West Nile virus outbreaks (Douglaston, Cuyahoga County, and Ontario, see Section 9, where the per million risk of hospitalization from disease was found to be more on the order of 100, compared to the results between two and five for Connecticut and Suffolk County). These comparisons suggest that adoption of some form of IPM can have significant impacts on the effects of WNV to area residents. The lesser rates of infection for Suffolk County compared to Connecticut also may suggest that the use of adulticides does have the effect of reducing risk of disease even more; this argument is not as well supported by the available data.

It is extremely difficult to determine, analytically, risks associated with EEE. It has not occurred as often as WNV, or as predictably. Nonetheless, EEE is inextricably linked to particular habitat types. Of particular concern are white cedar swamps found near salt marshes, as this represents the key situation described for EEE transmission in New Jersey.

Edinger et al. (2002) report that Atlantic white cedar swamps are restricted to the coastal lowland zone in New York State, which includes Long Island. The reference site for Atlantic white cedar swamps is given as the Cranberry Bog County Park. This abandoned cranberry bog is adjacent to Sweezy and Cheney Ponds, in the 460-acre park, and is the largest of Long Island's surviving Atlantic white cedar swamps. This a somewhat anomalous white cedar swamp, in that it is not associated with salt marshes. Atlantic white cedars can be found in coastal plain poor fens, but they are not the dominant vegetation.

Cashin Associates (2004) reported that this community type can be found within Cranberry Bog County Park and Cedar Swamp (along the perimeter of Cheney Pond and Cedar Pond, on the north side of County Road 51 southwest of downtown Riverhead behind the County Complex). The coastal plain Atlantic white cedar swamp is a predominantly evergreen or mixed evergreen/deciduous swamp that occurs on organic soils along streams and in poorly drained depressions and kettle holes of the Long Island coastal plain. The peat deposits that are prevalent typically overlie a substrate of sand. Atlantic white cedar (*Charmaecyparis thyoides*) comprises over 50 percent of the canopy cover in these communities. Red maple (*Acer rubrum*) trees are often co-dominant in impure Atlantic white cedar stands, although lesser black gum and pitch pine may be present as well. Atlantic white cedars require full sunlight and moist soils to thrive. However, long-term soil saturation associated with damming, or, conversely, desiccation associated with the draining of surface water bodies can result in tree loss. Changes in long-term precipitation patterns will have a similar impact. Moreover, degraded water quality (from stormwater discharges and/or polluted groundwater) can adversely affect the health of Atlantic white cedar trees. Ironically, a major threat to Atlantic white cedar regeneration and sustainability is the inhibition of light penetration caused by the screening effect of the trees' own foliage on potential seedlings. As individual trees are lost or become stressed, opportunities arise for competing species to gain dominance. Forest fires, chronic flooding, and windthrow, however, occasionally open these stands to larger expanses of sunlight, thereby allowing the stand to regenerate.

Atlantic white cedar forests were once prevalent on Long Island (Spring-Rushia and Stewart, 1996), but are now considered to be rare with only a few significant occurrences on Long Island. This community type has a global ranking of G3G4 and a State ranking of S1 making it "especially vulnerable" in the State of New York (Edinger et al., 2002).

Contacts were made with local natural resources experts to determine if other Atlantic white cedar swamps are identifiable in Suffolk County (Table 7-41).

Table 7-41. Atlantic White Cedar Swamps in Suffolk County

Town	**Location**
Babylon	Belmont State Park[1]
Brookhaven	None[2]
East Hampton	None[3]
Huntington	None[4]; perhaps on the east side of Crab Meadow[5]
Islip	None identified
Riverhead	None[6]
Shelter Island	None known[7]
Smithtown	Blydenburgh Park[8]
Southampton	Cranberry Bog County Park[9] Cedar Swamp[10] Along Route 24, at and around Hubbard Creek[11] Owl Pond, Birds Creek County Park[7] Sears Bellows County Park[12] Hubbard County Park[12]
Southold	None identified

[1] J. Guarino, Senior Environmental Analyst, Town of Babylon, Department of Environmental Control, personal communication, 2005
[2, 8] J. Turner, Director of Environmental Protection, Town of Brookhaven, personal communication, 2005
[3] B. Frank, Chief Environmental Analyst, Town of East Hampton, personal communication, 2005
[4] J. Dieterich, Department of Maritime Services, Town of Huntington, personal communication, 2005
[5] M. Myles, Department of Planning, Town of Huntington, personal communication, 2005
[6] J. Hall, Town of Riverhead Planning Department, personal communication, 2005
[7] L. Bavaro, Peconic Estuary Program, SCDHS Office of Ecology, personal communication, 2005
[9] Edinger et al., 2002
[10] Cashin Associates, 2004
[11] M. Brusseau, Cashin Associates, personal observations
[12] W. Sickles, Suffolk County Department of Parks, personal communication, 2006

Although there are not many Atlantic white cedar swamps in the County, there are also some red maple swamps in close proximity to salt marshes. A prime example of this is the Mastic-Shirley area, where Cashin Associates found stands of red maple swamps in the fresh water wetlands immediately north and east of the salt marshes along the shoreline (see Section 5). There are also red maple swamps in Amityville near the shoreline, as well, according to Town officials. It is clear that similar guilds of birds inhabit each swamp, and that *Cs. melanura* can thrive in both. Therefore, coastal red maple swamps may very well serve the same function in areas of Suffolk County that coastal stands of Atlantic white cedar swamps appear to serve in New Jersey – that is, to create opportunities for salt marsh mosquitoes to come into contact with EEE-infected birds and so become vectors. Suffolk County therefore appears to have been fortunate not to have experienced human cases of EEE spread by this mechanism, given the competence of *Oc. sollicitans* as a vector of EEE.

Red maple swamps are otherwise fairly ubiquitous inland in the County. Notable examples are found in Manorville on and near the Peconic River and in the vicinity of Southaven County Park on the Carmans River. The text book example of this ecological community occurs primarily in the lower stretch of the Peconic River although it is fairly common throughout the County. This community can be described as a maritime, coastal, or inland hardwood swamp which commonly forms either a narrow transitional zone between a river (such as the Peconic River) and an adjacent pine barrens uplands or may occupy a poorly-drained topographic depression. Long Island's red maple-black gum coastal plain swamps usually have a thin surface layer of acidic, substantially decomposed peat over saturated sandy loam or loamy sand grading into sand at higher elevations within the pine barrens. Red maple-black gum swamps often occur in saturated and or damp soils that are subject to periodic riverine flooding and/or seasonally-high groundwater levels which rise to or near the ground surface. Inland examples usually occur on an acidic silty loam soil; however, this vegetative community can develop on soils of various textures. The ground surface often exhibits hummocky micro-topography and the roots of the red maple trees are at least partially exposed (Edinger et al., 2002).

Red maple swamps provide valuable habitat and breeding areas for a variety of resident birds. Swamps dominated by red maple are known to attract a plethora of insects, particularly in the early spring. The insects attract a variety of insectivorous birds such as vireos, warblers, and thrushes. Most migrate south at the start of cold weather. As winter arrives, kinglets (*Sylviidae*), nuthatches (*Sittidae*), woodpeckers (*Picidae*), titmice (*Paridae*), and brown creepers (*Certhia familiaris*) begin to infiltrate to feed on insect larvae from the dying, dead, and decaying trees (CPBJPPC, 1995).

Red maple black-gum swamps have a global rarity ranking of G3 and a State ranking of S2 and are therefore considered to be "very vulnerable" within the State (NYSDEC New York Natural Heritage Program, 2002).

The primary bridge vectors for settings such as red maple swamps are usually cited as *Cq. perturbans* and *Ae. vexans*. The relatively low percent transmission rate reported by Chamberlain (1956) (discussed in Section 3) may explain why the inland cycling of EEE appears to be less efficient at infecting people.

That these two fresh water mosquitoes are not as good at infecting people and that Suffolk County has not experienced human cases of EEE historically should not be reason to assume risks are small. EEE amplification in the bird population peaks in mid-summer. This is just when young-of-the-year birds migrate from natal areas to find territories of their own. As discussed above, these young birds that are more likely to have high levels of virus in their systems. Common sense dictates that the focus of concern when EEE amplifies in *Cs. melanura* populations setting should be the immediate vicinity of that swamp, bounded by some flight radius of the mosquito species of concern. Additional concerns should be raised anywhere that the fledglings fly from that particular swamp. Commonly, these birds will seek similar environments, meaning that when EEE is amplifying, attention should be paid to outbreaks in similar red maple or Atlantic white cedar swamps. However, there is no certainty that the migrating birds will in fact seek habitat similar to where they fledged. They may seek entirely different areas altogether. For that reason, in southern New Jersey, where EEE and Atlantic white cedar-red maple environments are more common, control efforts focus on all large populations of human biting mosquitoes whenever EEE has amplified to minimize risks of human disease. In New Jersey, it has been determined that it is not possible to safely predict where a human-biting mosquito may host off an infected bird and become an EEE vector. The theory is that reducing the numbers of any human biting mosquito will reduce disease risk.

Control measures used in Suffolk County and elsewhere are inherently somewhat inefficient. This is because it is difficult to control the amplification vector, *Cs. melanura*, reliably due to its life cycle and preferred habitats. In addition, *Cs melanura* habitats often intersect habitats of importance for rare-threatened-endangered species (which tend to be found in ephemeral fresh water wetlands, for one). This makes the potential for impacts to non-target organisms of more concern, and so both program managers and regulators tend to be less comfortable instituting controls, be they water management or pesticide applications. When monitoring reveals cycling of EEE in *Cs. melanura*, which means amplification of the virus in birds, control measures have targeted the obvious, local bridge vectors. In Suffolk County, that has generally meant special efforts to look for *Ae. vexans* and *Cq. perturbans* populations, and to adulticide those mosquitoes to reduce risk (while also conducting larviciding efforts in breeding habitats earlier in the season as prophylaxis).

Given the stunning efficiency of *Oc. sollicitans* as an EEE vector, it may be that the focus of Suffolk County on controlling salt marsh mosquito problems may have had an additional effect of reducing risk from EEE. Because vector control efforts here have always tried to keep salt marsh mosquito populations low to reduce human discomfort, the effect may have been to create many fewer chances for opportunistic feeding by *Oc. sollicitans* on migrating infected birds. Similarly, the geographic absence of large Atlantic white cedar swamps near salt marshes, and relatively low number of red maple swamps similarly located, also has reduced relative risks in the County.

This suggests that the current control program has reduced risks for EEE over a no mosquito control option, by aggressively reducing bridge vectors. This is likely to be a benefit for any of the novel disease risks:

- Jamestown Canyon virus
- La Crosse virus
- Sindbis virus
- Rift Valley fever virus
- Japanese encephalitis virus
- Usutu virus

It is clear that controlling major infestations of human-biting mosquitoes necessarily reduces the instances of non-clinical impacts from mosquitoes. These aggravating, and almost never fatal side effects of mosquito feeding on human blood occur less often when there is less mosquito feeding on people.

Ecological impacts of mosquito-borne disease under IPM are very difficult to ascertain. It is far from clear that risks to crows from WNV, for example, were reduced by mosquito control efforts across the County. Control efforts are certainly not targeted at alleviating ecological impacts. There is some interest in reducing WNV impacts to horses, or EEE impacts to horses, pheasants, or, if they were raised here, emus. Incidence of disease in birds tends to focus control interest in particular areas, and past incidents lead to greater surveillance efforts, which may lead to benefits for the birds over time. However, the interest does not extend as far as having control measures enacted to reduce the impacts.

All-in-all, it seems likely that the current program has had a demonstrable impact on disease risk in the County, although it is difficult to partition potential benefits from control efforts, the natural differences in mosquito ecology between Suffolk County and other locations, and the induced mosquito ecology resulting from decades of mosquito control efforts.

7.11.3. Mosquito-borne Disease Risks Under the Long-Term Plan

It seems clear that the Long-Term Plan will not increase risks of WNV illness compared to the current program. Implementation of progressive water management should, at worst, maintain, and actually is intended to decrease, salt marsh mosquito numbers. Other potential bridge vectors, such as *Ae. vexans*, will continue to be aggressively controlled. *Cx. pipiens* may be better controlled through wider use of larvicides in storm water systems, and more targeted applications of adulticides for viral control, in terms of timing and application means, in fresh water environments where trapping indicates that *Cx. pipiens* is the dominant mosquito present. Continuing public education should also pay dividends in reductions of mosquito environments in the near vicinity of residences and businesses. Therefore, it seems likely that WNV risks for people will be reduced through adoption of the proposed Plan.

Implementation of the Long-Term Plan may reduce risks somewhat from the current mosquito control situation for EEE, as well. This follows from the following elements:

- A strong EEE surveillance program in red maple swamps will be continued, allowing for early detection of the amplification cycle.
- If amplification is detected, surveillance will be extended to similar habitats following logical, local migration paths (mostly south and west), to determine if young-of-the-year birds are spreading the virus.

- Prophylactic control of obvious bridge vectors in the vicinity of historical amplification areas will be continued, using larvicides.
- When the risk profile warrants (if there are large numbers of bridge vectors in the vicinity of an amplification center), adulticides will be applied to reduce the risk of human or equine cases.
- Although the reduction of EEE risks may be an unintentional by-product, salt marsh mosquitoes will be controlled using larvicides, and, if warranted, adulticides; this appears to reduce the chances that this very efficient potential vector of EEE is not allowed to infect people.

Similarly, risks from endemic and novel diseases may be less under the proposed plan, for similar reasons. Good surveillance will determine the presence of the pathogens, and control of most bridge vectors means that opportunities for human infection are less than they might otherwise be.

Progressive water management, as it is implemented, holds the promise (according to experiences in neighboring jurisdictions), of reducing salt marsh mosquito populations as well as larvicides and current water management techniques do, except with greater consistency. This should lead to fewer "out-of-control" broods, and so fewer people experiencing reductions in the quality of life because of these mosquitoes.

Thus, the overall result of implementation of the Long-Term Plan should be a reduction in overall disease risk from mosquito-borne pathogens, when compared to those risks under the current program. The current program, it should be emphasized, appears to reduce disease risks quite significantly. This issue will be discussed more fully following the analysis of "no mosquito control," in Section 9.

Section 7 References

ATSDR. 2003. *Toxicological Profile for Malathion*. US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, Atlanta, GA. 287 pp. + appendices.

ATSDR. 2001. *Toxicological Profile for Pyrethrins and Pyrethroids*. US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, Atlanta, GA. 267 pp. + appendices.

Adamowicz, SC, and CT Roman. 2002. *Initial Ecosystem Response of Salt Marshes to Ditch Plugging and Pool Creation: Experiments at Rachel Carson National Wildlife Refuge (Maine)*. Final Report. USGS Patuxent Wildlife Research Center, Narragansett, RI. 102 pp.

Ahl, JSB, and JS Brown. 1990. Salt-dependent effects of juvenile hormone and related compounds in larvae of the brine shrimp, Artemia. *Comparative Biochemistry and Physiology, A* 95A(4):491-496.

Amweg, EL, DP Weston, and NM Ureda. 2005. Use and toxicity of pyrethroid pesticides in the central Valley, California, USA. *Environmental Toxicology and Chemistry* 24:966-972.

Amweg, EL, DP Weston, J. You, and MJ Lydy. 2006. Pyrethroid insecticides and sediment toxicity in urban creeks from California and Tennessee. *Environmental Science and Technology* 40(5):1700-1706.

Andersen, HR, AM Vinggaard, TH Rasmussen, IM Gjermandsen, and EC Bonefeld-Jorgensen. 2002. Effects of currently used pesticides in assays for estrogenicity, androgenicity, and aromatase activity in vitro. *Toxicology and Applied Pharmacology* 179:1-12.

Anderson, JF, TG Andreadis, AJ Main, and DL Kline. 2004. Prevalence of West Nile virus in tree canopy-inhabiting *Culex pipiens* and associated mosquitoes. *American Journal of Tropical Medicine and Hygiene* 71(1):112-119.

Andreadis, TG, JF Anderson, CR Vossbrinck, and AJ Main. 2004. Epidemiology of West Nile virus in Connecticut: a five-year analysis of mosquito data 1999-2003. *Vector-borne and Zoonotic Diseases* 4(4):360-378.

Apperson, CS, Harrison, BA, TR Unnasch, HK Hassan, WS Irby, HM Savage, SE Aspen, DW Watson, LM Rueda, BR Engber, and RS Nasci. 2002. Host-feeding habits of *Culex* and other mosquitoes (Diptera: Culicidae) in the borough of Queens in New York City, with characters and techniques for identification of *Culex* mosquitoes. *Journal of Medical Entomology* 39(5):777-785.

Arnold, SF, and JA McLachlan. 1996. Synergistic signals in the environment. *Environmental Health Perspectives* 104(10):1020-1023.

Azziz-Baumgartner, E. 2004. Mosquito control and exposure to pesticides: Virginia and North Carolina, 2003. Presentation at the *5th Annual Conference on West Nile Virus in the United States*, February 3-5, Denver, CO. Available at http://www.cdc.gov/ncidod/dvbid/westnile/conf/pdf/Azziz_Baumgartner_6_04.pdf. Accessed on 2/2/05.

Balcer, MD, KL Schmude, J. Snitgen, and AR Lima. 1999. *Long-term Effects of the Mosquito Control Agents Bti (Bacillus thuringiensis israelensis) and Methoprene on Non-target Macroinvertebrates in Wetlands in Wright County, Minnesota (1997-1998).* Metropolitan Mosquito Control District, Minneapolis, MN.

Barnes, RK. 2005. *Pesticides Used to Control West Nile Virus: Toxicity to the Estuarine Grass Shrimp, Palaemonetes pugio.* Masters Thesis. Stony Brook University.

Bart, DJ, and JM Hartman. 2002. Constraints on the establishment of *Phragmites australis* in a New Jersey salt marsh and possible links to human disturbance. *Wetlands* 22:201-213.

Batzer, DP, and RD Sjogren. 1986. Potential effects of Altosid (methoprene) briquette treatment on *Eubranchipus bondyi* (Anostraca: Chiroccphalidac). *Journal of the American Mosquito Control Association* 2(2):226-227.

Bertness, MD. 1999. *The Ecology of Atlantic Shorelines*. Sindauer Associates, Inc., Sunderland, MA. 417 pp.

Bertness, MD, PJ Ewanchuk, and BR Silliman. 2002. Anthropogenic modification of New England salt marsh landscapes. *Proceedings of the National Academy of Sciences* 99(3):1395-1398.

Bircher, L., and E. Ruber. 1988. Toxicity of methoprene to all stages of the salt marsh copepod, *Apocyclops spartinus* (Cyclopoida). *Journal of the American Mosquito Control Association* 4(4):520-523.

Birchfield, N., R. Mahler, and B. Montague. Undated. *EFED's Response to "Comments from Cheminova A/S on EPA's Preliminary Risk Assessment for Malathion."* Memorandum to B. Shackleford and P. Moe, US Environmental Protection Agency, Washington, DC. www.epa.gov/pesticides/op/malathion/efed_response.pdf

Bourn, WS, and C. Cottam. 1950. *Some Biological Effects of Ditching Tide Water Marshes*. US Fish and Wildlife Research Report 19. 30 pp. + oversize figures.

Brattsten, LB, and DJ Sutherland. 2005. *Insecticides Recommended for Mosquito Control in New Jersey in 2005*. New Jersey Agricultural Experiment Station, Publication No. P-08001-01-05. www-rci.rutgers.edu~insects/bmpmcnj.htm.

Brimley, CS. 1938. *The Insects of North Carolina*. North Carolina Department of Agriculture, Raleigh, NC. 560 pp.

Brimley, CS. 1942. *Supplement to the Insects of North Carolina*. North Carolina Department of Agriculture, Raleigh, NC. 39 pp.

Britton, WE. 1920. *Checklist of the Insects of Connecticut*. State Geological and Natural History Bulletin 31. 397 pp.

Britton, WE, with others. 1916. *Guide to the Insects of Connecticut Part III. The Hymenoptera or Wasp-like Insects.* State Geological and Natural History Bulletin 22. 824 pp.

Britton, WE, with others. 1923. *Guide to the Insects of Connecticut, Part IV. The Hemiptera or Sucking Insects of Connecticut*. State Geological and Natural History Bulletin 34. 807 pp.

Britton, WE, and BJ Kasten. 1938. *Additions to the Checklist of the Insects of Connecticut, Supplement to Bulletin 31, Checklist of the Spiders of Connecticut*. State Geological and Natural History Bulletin 60. 201 pp.

Britton, WE, and BH Walden. 1911. *Guide to the Insects of Connecticut. Part I. The Euplexoptera and Orthoptera of Connecticut*. State Geological and Natural History Bulletin 16. 169 pp.

Brody, JG, A. Aschengrau, W. McKelvey, RA Rudel, CH Swartz, and T. Kennedy. 2004. Breast cancer risk and historical exposure to pesticides from wide-area applications assessed with GIS. *Environmental Health Perspectives* 112(8):889-897.

Bromley, SW. 1946. *Guide to the Insects of Connecticut Part VI. The Diptera or True Flies of Connecticut. Third Fascicle: Asilidae, or Robber Flies.* State Geological and Natural History Bulletin 69. 15 pp.

Brown, W. 1998. *Mosquito Larvicides - Non-Target Organism Effects.* US Fish and Wildlife Service.

Brown, TM, and AWA Brown. 1980. Accumulation and distribution of methoprene in resistant *Culex pipiens pipiens* larvae. *Entomological Experimental Applications* 27(1):11-22.

Brown, M., J. Carter, D. Thomas, D. Purdie, and B. Kay 2002. Pulse-exposure effects of selected insecticides to juvenile Australian crimson-spotted rainbowfish (*Melanotaenia duboulayi*). *Journal of Economic Entomology* 95(2):294-298.

Brush, T., RA Lent, T. Hruby, BA Harrington, RM Marshall, and WG Montgomery. 1986. Habitat use by salt marsh birds and response to open marsh water management. *Colonial Waterbirds* 9:189-195.

Brzozowski, C. 2004. Maintenance goes underground: owners and vendors talk about how to keep systems working. *Stormwater* 5(5):76-87.

Buchsbaum, R. 2001. Coastal marsh management. Chapter 11. In: Kent, DM (ed.). *Applied Wetlands Science and Technology*. CRC Press, Boca Raton, FL.

Buchsbaum, R, A. Cooper, and J. LeBlanc. 1998. Habitat issues faced by the Plum Island Sound estuary. pp. 148-158. In: Buchsbaum, R., T. Purinton, and B. Magnuson (eds.). *A Study of the Marine Resources of the Parker River-Plum +Island Sound Estuary: An Update After 30 Years*. Massachusetts Office of Coastal Zone Management.

Buei, K, S. Ho, T. Yamada, S. Gamo, and M. Kato. 1975. The effect of a juvenile hormone mimic, methoprene against mosquito larvae. *Japanese Journal of Sanitary Zoology* 26(2-3):105-111.

Burdick, DM, M. Dionne, RM Boumans, and FT Short. 1997. Ecological responses to tidal restrictions of two northern New England salt marshes. *Wetlands Ecology and Management* 4(2):129-144.

Burmaster, DE, and PD Anderson. 1994. Principles of good practice for the use of Monte Carlo techniques in human health and ecological risk assessments. *Risk Analysis* 14(4):477-481.

Burmaster, DE, and AM Wilson. 1996. An introduction to second-order random variables in human health risk assessments. *Human and Ecological Risk Assessment* 2(4):892-919.

Butler, WH, KL Gabriel, TG Osimitz, and FJ Preiss. 1998. Oncogenicity studies of piperonyl butoxide in rats and mice. *Human Exposure Toxicology* 17:323-330.

CA-CE. 2004. *Long Island Mosquitoes*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 3 (Literature Search), Book 1. Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. 58 pp. + appendix.

CA-IC. 2004. *Ecotoxicity Review of Primary List Mosquito Control Agents.* Task 3, Literature Review, Book 7, Suffolk County Vector Control and Wetlands Management Long-Term Plan and Generic Environmental Impact Statement. Prepared by Cashin Associates and Integral Consulting, for Suffolk County Department of Health Services, Yaphank, NY. Paged in sections.

CA-SCDHS. 2005. *Human Health and Domestic Animal Toxicity.* Task 3, Literature Review, Book 6 Part 2, Suffolk County Vector Control and Wetlands Management Long-Term Plan and Generic Environmental Impact Statement. Prepared by Cashin Associates and Suffolk County Department of Health Services, for Suffolk County Department of Health Services, Yaphank, NY. Paged in sections.

CA-USGS. 2005. *Water Sampling Report*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 12 (Early Action Projects Caged Fish Experiment). Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. 21 pp.

CDC. 2005. Human exposure to mosquito-control pesticides: Mississippi, North Carolina, and Virginia, 2002 and 2003. *Morbidity and Mortality Weekly Report* 54(21):529-532.

CDC. 2003a. *Epidemic/Epizootic West Nile Virus in the United States: Guidelines for Surveillance, Prevention, and Control*. 3rd Revision. National Center for Infectious Diseases, Division of Vector-Borne Infectious Diseases. Fort Collins, CO. 80 pp.

CDC. 2003b. Surveillance for acute insecticide-related illness associated with mosquito-control efforts – nine states, 1999 – 2002. *Morbidity and Mortality Weekly Report* 52(27):629-634.

CDC. 2001. *Severity Index for Use in State-based Surveillance of Acute Pesticide-related Illness and Injury*. Centers for Disease Control and Prevention. Available at http://www.cdc.gov/niosh/pestsurv/pdfs/pest-sevindexv6.pdf. Accessed on 9/30/04.

CDC. 2000. *Case Definition for Acute Pesticide-related Illness and Injury Cases Reportable to the National Public Health Surveillance System*. Centers for Disease Control and Prevention. Available at http://www.cdc.gov/niosh/pestsurv/pdfs/pest-casdef2.2000.pdf. Accessed on 9/30/04.

CPBJPPC. 1995. *Central Pine Barrens Comprehensive Land Use Plan*. Vol. 2: Existing conditions. Central Pine Barrens Joint Planning and Policy Commission, Great River, NY.

Cabello, G., A. Juarranz, LM Botella, and GM Calaf. 2003. Organophosphorous pesticides in breast cancer progression. *Journal of Submicroscopic Cytology and Pathology* 35:1-9.

Cabello, G. M. Valenzuela, A, Vilaxa, V. Duran, I. Rudolph, N. Hrepic, and G. Calaf. 2001. A rat mammary tumor model induced by organophosphorous pesticides parathion and malathion, possibly through acetylcholinesterase inhibition. *Environmental Health Perspectives* 109:471-479.

Campbell, SR, D. Ninivaggi, and JF Sanzone. 2005. The effectiveness of methoprene for the treatment of salt marsh mosquitoes in Suffolk County, New York. Poster No. 162. *71st Annual Meeting, The American Mosquito Control Association*. Vancouver, BC. April 3-7.

Carlson, DB, PD O'Bryan, and JR Rey. 1991. A review of current salt marsh management issues in Florida. *Journal of the American Mosquito Control Association* 7:83-89.

Caron, DM. 1979. Effects of some ULV mosquito abatement insecticides on honey bees. *Journal of Economic Entomology* 72:148-151.

Cashin Associates. 2006. *Salt Marsh Losses*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 3 (Literature Search), Book 9, Part 4. Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. 78 pp.

Cashin Associates. 2005a. *Salt Marsh Health*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 3 (Literature Search), Book 9, Part 1. Suffolk County Department of Health Services, Riverhead, NY. www.suffolkmosquitocontrolplan.org. 50 pp.

Cashin Associates. 2005b. *Task Report: Part I: Summary*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 8 (Impact Assessment). Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. 31 pp.

Cashin Associates. 2005c. *Task Report: Part III: Human Health and Ecological Risk Assessment of Vector Control Pesticides Considered for Use by Suffolk County*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 8 (Impact Assessment). Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. Paged in sections + appendices.

Cashin Associates. 2005d. *Water and Sediment Concentration Measurements*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 12 (Early Action Projects Caged Fish Experiment). Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. 31 pp. + appendices.

Cashin Associates. 2005e. *Effects on Organisms*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 12 (Early Action Projects Caged Fish Experiment). Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. 17 pp. + appendix.

Cashin Associates. 2004. *Health and Environmental Assessment for the Peconic River*. Suffolk County Department of Health Services, Hauppauge, NY. 3 vols.

Cehllayan, S. and GK Karnavar. 1989. Juvenile hormone induced ovipostion in virgin *Trogoderma granarium* (Dermestidac: Coleoptora). *Entomon* 14(3-4):187-190.

Celestial, DM, and CL McKenney, Jr. 1994. The influence of an insect growth regulator on the larval development of the mud crab, *Rhithropanopeus harrisii*. *Environmental Pollution* 85(2):169-173.

Cerrato, RM; HJ Bokuniewicz, and MH Higgins. 1989. *A Spatial and Seasonal Study of the Benthic Fauna of the Lower Bay of New York Harbor*. Marine Sciences Research Center Special Report 84 (89-1), SUNY at Stony Brook, Stony Brook, NY. 325 pp.

Chamberlain, RW. 1956. Virus-host relationships of the American arthropod-borne encephalitides. *Proceedings of the 10th International Congress of Entomology* 3:567-572.

Chapman, VJ. 1974 *Salt Marshes and Salt Deserts of the World*. 2nd. Ed. Strauss & Cramer GMBH, Leutershausen, Germany. 392 pp. + 101 pp. added for 2nd. Ed.

Charbonneau, CS, RD Drobney, and CF Rabeni. 1994. Effects of *Bacillus thuringiensis var. israelensis* on nontarget benthic organisms and factors affecting the efficacy of the larvicide. *Environmental Toxicology and Chemistry* 13:267–279.

Chen, H., J. Xiao, G. Hu, J. Zhou, H. Xiao, and X. Wang. 2002. Estrogenicity of organophosphorous and pyrethroid pesticides. *Journal of Toxicology and Environmental Health A* 65:1419-1435.

Christiansen, ME, JD Costlow, Jr., and RS Monroe. 1977. Effects of the juvenile hormone mimic ZR-515 (Altosid) on larval development of the mud crab, *Rhithropanopeus harrisii* in various salinities and cyclic temperatures. *Marine Biology* 39(3):269-279.

Christie, GD. 1990. Salt marsh mosquito control in Portsmouth, Rhode Island. *Journal of the American Mosquito Control Association* 6(1):144-147.

Clarke, JA, BA Harrington, T. Hruby, and FE Wasserman. 1984. The effect of ditching for mosquito control on salt marsh use by birds in Rowley, Massachusetts. *Journal of Field Ornithology* 55(2):160-180.

Clements, AN. 1992. *The Biology of Mosquitoes.* Vol.1*: Development, Nutrition and Reproduction*. Chapman & Hall, New York, NY. 509 pp.

Clifford, MA, KJ Eder, I Werner, HP Hedrick. 2005. Synergistic effects of esfenvalerate and infectious hematopoietic necrosis virus on juvenile Chinook salmon mortality. *Environmental Toxicology and Chemistry* 24:1766-1772.

Citra, M. 2004. Incorporating Monte Carlo analysis into multimedia environmental fate models. *Environmental Toxicology and Chemistry* 23(7):1629-1633.

Collins, JN, LM Collins, LB Leopold, and VH Resh. 1986. The influence of mosquito control ditches on the geomorphology of tidal marshes in the San Francisco Bay area: the evolution of salt marsh mosquito habitats. *Proceedings of the California Mosquito and Vector Control Association* 54:91-95.

Cook, EF, CP Alexander, and WR Nowell. 1963. *Guide to the Insects of Connecticut Part VI. The Diptera or True Flies of Connecticut. Eighth Fascicle: Scaropsidae and Hyperoscelidae. Blepharoceridae and Deuterophlebiidae. Dixidae*. State Geological and Natural History Bulletin 93. 115 pp.

Cory, EN, and SL Crosthwait. 1939. Some conservation and ecological aspects of mosquito control. *Journal of Economic Entomology* 32:213-215.

Costlow, JD, Jr. 1977. The effects of juvenile hormone mimics on development of the mud crab, *Rhithropanopeus harrisii* (Gould). pp. 439-457. In: Vernberg, FJ, A. Calabrese, FP Thurberg, and WB Vernberg (eds.). *Physiological Responses of Marine Biota to* Pollutants. Academic Press, New York, NY. 462 pp.

Courtney, WR, Jr., and GK Meffe. 1989. Small fishes in strange places: a review of introduced poeciliids. Pp. 319-331. In: Meffe, GK, and FF Snelson, Jr. (eds.). *Ecology and Evolution of Live-bearing Fishes (Poeciliidae).* Prentice-Hall, Englewood Cliffs, NJ.

Cowan, DP, T., Hruby, LS Litwin, and RA Lent. 1986. *Open Marsh Management on Great South Bay, Islip, New York: Baseline Study: 1984-1985*. Cornell University Laboratory of Ornithology, Islip, New York. 101 pp.

Crampton, GC. 1942. *Guide to the Insects of Connecticut Part VI. The Diptera or True Flies of Connecticut. First Fascicle: External Morphology, Key to Families: Tanyderidae, Ptychopteridae, Trichocoridae, Anisopodidae, Tipulidae*. State Geological and Natural History Bulletin 64. 509 pp.

Daiber, FC. 1986. *Conservation of Tidal Marshes*. Van Nostrand Reinhold, New York, NY. 341 pp.

Dale, PER, and K. Hulsman. 1990. Critical review of salt marsh management methods for mosquito control. *Reviews in Aquatic Science* 3(2,3):281-311.

Davidson C., HB Shaffer, and MR Jennings. 2001. Declines of the California red-legged frog: Climate, UV-B, habitat, and pesticides hypotheses. *Ecological Applications* 11:464-479.

Debboun, M. and JA Klun. 2005. Personal protective measures and repellants used against mosquitoes in the US military. Presentation 121. 71st Annual Meeting, *The American Mosquito Control Association*. Vancouver, BC. April 5.

DeChant, P. 2005. Structure and function of urban drainage systems and their impact on vector mosquito production. Presentation 23. 71st Annual Meeting, *The American Mosquito Control Association*. Vancouver, BC. April 4.

Deegan, LA, JE Hughes, and RA Rountree. 2000. Salt marsh ecosystem support of marine transient species. pp. 333-365. In: Weinstein, MP, and DA Kreeger (eds.). *Concepts and Controversies in Tidal Marsh Ecology*. Kluwer Acadmic Publishers, Boston, MA. 875 pp.

Diamond, JM, and TJ Case. 1986. Overview: introductions, extinctions, exterminations, and invasions. pp. 65-79. In: Diamond, JM, and TJ Case (eds.). *Community Ecology*. Harper and Row, New York, NY.

Dreyer, GD, and WA Niering. 1995. *Tidal Marshes of Long Island Sound: Ecology, History, and Restoration*. The Connecticut College Arboretum, No. 34.

Dunnet, GM. 1977. Observations on the effects of low-flying aircraft at seabird colonies on the coast or Aberdeenshire, Scotland. *Biological Conservation* 12:55-63.

Durda, JL, and DV Preziosi. 2000. Data quality evaluation of toxicological studies used to derive ecotoxicological benchmarks. *Human and Ecological Risk Assessment* 6(5):747-765.

ECOFRAM. 1999. *Ecological Committee on FIFRA Risk Assessment Methods: Report of the Aquatic Workgroup*. US Environmental Protection Agency, Office of Pesticide Programs, Washington, DC

Edinger, GJ, DJ Evans, S. Gebauer, TG Howard, DM Hunt, and AM Olivero (eds.). 2002. *Ecological Communities of New York State*. 2nd Ed. (A revised and expanded edition of Carol Reschke's Ecological Communities of New York State, draft for review). New York State Department of Environmental Conservation New York Natural Heritage Program, Albany, NY.

Edwards, RG, AG Broderson, RW Barbour, DF McCoy, and CW Johnson. 1979. *Assessment of the Environmental Compatibility of Differing Helicopter Noise Certification Standards*. US Department of Transportation, Washington, DC. 58 pp.

Effland, WR, NC Thurman, and I. Kennedy. 1999. *Proposed Methods for Determining Watershed-derived Percent Cropped Areas and Considerations for Applying Crop Area Adjustments to Surface Water Screening Models*. US Environmental Protection Agency Office of Pesticide Programs; Presentation To FIFRA Science Advisory Panel, May 27.

Eidson, M., N. Komar, F. Sorhage, R. Nelson, T. Talbot, F. Mostashari, R. McLean, and the West Nile Virus Avian Mortality Surveillance Group. 2001. Crow deaths as a sentinel surveillance system for West Nile Virus in the northeastern United States. *Emerging Infectious Diseases* 7(4): 615-620.

Elbetieha, A., SI Da'as, W. Khamas, and H. Darmani. 2001. Evaluation of the toxic potentials of cypermethrin pesticide on some reproductive and fertility parameters in the male rats. *Archives of Environmental Contamination and Toxicology* 41(4):522-528.

Emmel, TC, and JC Tucker. 1991. Mosquito control pesticides: ecological impacts and management alternatives. *Proceedings of a Conference held on January 18, University of Florida*, Gainesville, Florida. Scientific Publishers, Inc., Gainesville, Florida.

Eskenazi, B., A. Bradman, and R. Castorina. 1999. Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives* 107(Supplement)3:409-419..

Extoxnet. 1996a. *Pesticide Information Profiles: Methoprene*. http://extoxnet.orst.edu/pips/methopre.htm. Accessed on 7/12/04.

Extoxnet. 1996b. *Pesticide Information Profiles: Permethrin*. http://extoxnet.orst.edu/pips/permethr.htm. Accessed on 7/12/04.

Extoxnet. 1996c. *Pesticide Information Profiles: Resmethrin*. http://extoxnet.orst.edu/pips/resmethr.htm. Accessed on 7/12/04.

Extoxnet. 1996d. *Pesticide Information Profiles: Pyrethrins and Pyrethroids*. http://extoxnet.orst.edu/pip/pyrethri.htm. Accessed on 09/05.

Extoxnet. 1994. *Pesticide Information Profiles: Pyrethrin and Pyrethroids*.

FCCMC. 1998. *Florida Mosquito Control: The State of the Mission as Defined by Mosquito Collectors, Regulators, and Environmental Managers*. Florida Coordinating Council on Mosquito Control, University of Florida.

Fairchild, GB and CT Brues. 1950. *Guide to the Insects of Connecticut Part VI. The Diptera or True Flies of Connecticut. Fourth Fascicle:family Tabanidae, Family Phoridae*. State Geological and Natural History Bulletin 75. 92 pp.

Fell, PE, RS. Warren, WA. Niering. 2000. Restoration of salt and brackish tidelands in southern New England. pp. 845-858. In: Weinstein, MP, and DA Kreeger (eds.). *Concepts and Controversies in Tidal Marsh Ecology*. Kluwer Acadmic Publishers, Boston, MA. 875 pp.

Ferrigno, F., and DM Jobbins. 1968. Open Marsh Water Management. *Proceedings of the New Jersey Mosquito Extermination Association* 55:104-115.

Fischer, JM, JL Klug, T. Reed-Andersen, and AG Chalmers. 2000. Spatial pattern of localized disturbance along a southeastern salt marsh tidal creek. *Estuaries* 23(4):565-571.

Fletcher, JS, JE Nellessen, and TG Pfleeger. 1994. Literature review and evaluation of the EPA food-chain (Kenaga) nomogram, an instrument for estimation pesticide residues on plants. *Environmental Toxicology and Chemistry* 13(9):1383-1391.

Fonseca, DM, N. Keyghobadi, CA Macolm, C. Mehmet, F. Schaffner, M. Mogi, RC Fleisher, and RC Wilkerson. 2004. Emerging vectors in the *Culex pipiens* complex. *Science* 303:1535-1539.

Forbes, V.E., and A. Cold. 2005. Effects of the pyrethroid esfenvalerate on life-cycle traits and population dynamics of *Chironomid riparius* – importance of exposure scenario. *Environmental Toxicoogy and Chemistry* 24(1):78-86.

Fradin, MS and JF Day. 2002. Comparative efficacy of insect repellants against mosquito bites. *New England Journal of Medicine* 347(1):13-18

Frey, RW, and PB Basan. 1985. Coastal salt marshes. pp. 225-301. In: Davis, RA (ed.). *Coastal Sedimentary Environments.* 2nd Ed. Springer-Verlag, New York, NY. 716 pp.

Fultz, TO, Jr. 1978. Mosquito source reduction and present water management regulations in coastal Georgia. *Proceedings of the New Jersey Mosquito Extermination Association* 65:36-39.

Garey, J., and MS Wolff. 1998. Estrogenic and antiprogestagenic activities of pyrethroid insecticides. *Biochemical and Biophysical Research Communications* 251:855-859.

Garman, P. 1927. *Guide to the Insects of Connecticut: Part V. The Odonata or Dragonflies of Connecticut*. State Geological and Natural History Bulletin 39. 331 pp.

Gelernter, W.D. 2001. Environmental persistence of *Bacillus thurigiensis* and other bacterial insect pathogens. In: Baur, ME, and JR Fuxa (eds). *Factors Affecting the Survival of Entomopathogens*. Southern Cooperative Series Bulletin 400.

Gerig, L. 1985. Testing the toxicity of synthetic pyrethroids insecticides to bees. *Pesticide Science* 16:206-207.

Giddings, JM, KR Solomon, and SJ Maund. 2001. Probabilistic risk assessment of cotton pyrethroids: II. Aquatic mesocosm and field studies. *Environmental Toxicology and Chemistry* 20:660–668.

Gillette, JS, and JR Bloomquist. 2003. Differential up-regulation of striatal dopamine transporter and alpha-synuclein by the pyrethroid insecticide permethrin. *Toxicology and Applied Pharmacology* 192, 287-293, 2003.

Glare, J.R. and M. O'Callaghan. 1999. *Report for the Ministry of Health: Environmental and Health Impacts of the Insect Juvenile Hormone Analogue S-Methoprene*. As cited in Westchester, 2001.

Glasgow, RD. 1938. Mosquitoes and wild life as interrelated problems in human ecology. *New York State Museum Bulletin* 316:7-20.

Go, V., J. Garey, MS Wolff, and BG Pogo. 1999. Estrogenic potential of certain pyrethroid compounds in the MCF-7 human breast carcinoma cell line. *Environmental Health Perspectives* 107:173-177.

Goodsell, JA, and LB Kats. 1999. The effect of introduced mosquitofish on Pacific treefrogs and the role of alternate prey. *Conservation Biology* 13(4):921-924.

Greenlaw, JS. 1992. Seaside sparrow: *Ammodramus maritimus*. pp. 211-232. In: Schneider, KJ, and DM Pence (eds.). *Migratory Nongame Birds of Management Concern in the Northeast*. USFWS Region 5, Newton Corner, MA. 400 pp.

Gubler, DJ. 2001. Human arbovirus infections worldwide. pp. 13-24. In: White, DJ, and DL Morse (eds.). *West Nile Virus: Detection, Surveillance, and Control*. Annals of the New York Academy of Science, V. 951. New York, NY. 374 pp.

Gunn, WWH, and JA Livingstone (eds.). 1974. *Disturbance to Birds by Gas Compressor Noise Simulators, Aircraft, and Human Activity in the Mackenzie Valley and the North Slope*. Arctic Gas Biological Report Series #14. 280 pp.

Gupta, SK, MK Pandya, JP Jani, and SK Kashyap. 1980. Health risks in ultra-low-volume (ULV) aerial spray of malathion for mosquito control. *Journal of Environmental Science and Health* B15(3):287-294.

HSDB. 2005. *Toxicological Profile for Permethrin*. Hazardous Substances Data Bank. hhttp://toxnet.nlm.nih.gov/cgi-bin/sis/search.

HSDB. 2003. *Toxicological Profile for Piperonyl Butoxide*. Hazardous Substances Data Bank http://toxnet.nlm.nih.gov/cgi-bin/sis/search. Accessed on 7/13/04.

Hall, LW, Jr., MC Scott, WD Killen, and MA Unger. 2000. A probabilistic risk assessment of tributyltin in surface waters of the Chesapeake Bay watershed. *Human and Ecological Risk Assessment* 6:141–179.

Hardy, DE, and E. Pritchard. 1958. *Guide to the Insects of Connecticut Part VI. The Diptera or True Flies of Connecticut. Sixth Fascicle: March Flies and Gall Midges*. State Geological and Natural History Bulletin 87. 218 pp.

Havens, KJ, H. Berquist, and WI Priest, III. 2003. Common reed grass, *Phragmites australis,* expansion into constructed wetlands: are we mortgaging our wetlands future? *Estuaries* 26(2B):417-422.

Hayes, TB, P. Case, S. Chui, D. Chung, C. Haefele, K. Haston, M. Lee, VP Mai, Y. Marjuoa, J. Parker, and M. Tsui. 2006. Pesticides mixtures, endocrine disruption, and amphibian declines: are we underestimating the impact? *Environmental Health Perspectives* doi:10.1289/ehp.8051 (published online 1-24-06).

Hayes, T., K. Haston, M. Tsui, A. Hoang, C. Haeffele, and A. Vonk. 2003. Atrazine-induced hermaphroditism at 0.1 ppb in American leopad frogs (*Rana pipiens*): laboratory and field evidence. *Environmental Health Perspectives* 111(4):568-575.

Health Canada. 2004. *Proposed Phaseout of Citronella-Based Personal Insect Repellents.* Pest Management Regulatory Agency Information Note, 17 September.

Hershey, AE, AR Lima, GJ Niemi, and RR Regal. 1998. Effects of *Bacillus thuringiensis israelensis* (Bti) and methoprene on nontarget macroinvertebrates in Minnesota wetlands. *Ecological Applications* 8(1):41-60.

Hester, PG, KR Shaffer, NS Tietze, H. Zhong, and NL Griggs, Jr. 2001. Efficacy of ground-applied ultra-low volume malathion on honey bee survival and productivity in open and forested areas. *Journal of the American Mosquito Control Association* 17(1):2-7.

Hitchcock, SW. 1974. *Guide to the Insects of Connecticut Part VII. The Plecoptera or Stoneflies of Connecticut*. State Geological and Natural History Bulletin 107. 262 pp.

Hodges, RW. Undated. *Diversity and Abundance of Insects*. US Department of Agriculture Systematic Entomology Laboratory. Biology.usgs.gov/s+t/frame/f067.htm.

Hoerger, FD, and EE Kenaga. 1972. Pesticide residues on plants: Correlation of representative data as a basis for estimation of their magnitude in the environment. pp. 9-28. In: Coulston, F., and F. Korte (eds.). *Environmental Quality.* Academic Press, New York. Volume I.

Hoffman, FO, and JS Hammonds. 1994. Propagation of uncertainty in risk assessments: the need to distinguish between uncertainty due to lack of knowledge and uncertainty due to variability. *Risk Analysis* 14(5):707-712.

Hopkins, CC, FB Hollinger, RF Johnson, HJ Dewlett, VF Newhouse, and RW Chamberlain. 1975. The epidemiology of St. Louis encephalitis in Dallas, Texas, 1966. *American Journal of Epidemiology* 102(1):1-15.

Hoppin, JA, DM Umbach, SJ London, MC Alvanja, and DP Sandler. 2002. Chemical predictors of wheeze among farmer pesticide applicators in the Agricultural Health Study. *American Journal of Respiratory and Critical Care Medicine* 165:683-689.

Horst, MN, and AN Walker. 1999. Effects of the pesticide methoprene on morphogenesis and shell formation in the blue crab *Callinectes sapidus*. *Journal of Crustacean Biology* 19(4):699-707.

Howard, PH, RS Boethling, WF Jarvis, WM Meylan, and EM Michalenko. 1991. *Handbook of Environmental Degradation Rates.* Lewis Publishers, Chelsea, MI.

Howarth, FG. 1991. Environmental impacts of classical biological control. *Annual Reviews of Entomology* 36:485-509.

IARC. 1991. *IARC Monographs Permethrin.* International Agency for Research on Cancer, Lyon, France. Vol. 53:329.

Ishikawa, S., M. Miyata, A. Aoki, and Y. Hanai. 1993. Chronic intoxication of organophosphorous pesticide and its treatment. *Folia Medicine Cracov* 34:139-151.

James-Pirri, M-J, CT Roman, and RM Erwin. 2001. *Field Methods Manual: US Fish and Wildlife Service (Region 5) Salt Marsh Study*. US Geological Survey, Narragansett, RI.

Jensen, T., SP Lawler, and DA Dritz. 1999. Effects of ultra-low volume pyrethrin, malathion, and permethrin on non-target invertebrates, sentinel mosquitoes, and mosquitofish in seasonally impounded wetlands. *Journal of the American Mosquito Control Association* 15(3):330-338.

Johannsen, OA and HK Townes. 1952. *Guide to the Insects of Connecticut Part VI. The Diptera or True Flies of Connecticut. Fifth Fascicle: Family Tendipedidae, Family Heleidae, Family Fungivoridae*. State Geological and Natural History Bulletin 80. 225 pp.

Johnson, PTJ, and JM Chase. 2004. Parasites in the food web: linking amphibian malformations and aquatic eutrophication. *Ecology Letters* 7:521-526.

Johnson, A., and K. Kinney. 2006. *Methoprene Concentrations in Surface Water Samples from Grant County Mosquito Control District No. 1*. Washington State Department of Ecology Publication No. 06-03-001. 19 pp.

Johnson, PTJ, KB Lunde, DA Zelmer, and JK Werner. 2003. Limb deformities as an emerging parasitic disease in amphibians: evidence from museum specimens and resurvey data. *Conservation Biology* 17:1724-1737.

Kaiser, J. 2003. Fifty years of deformed frogs. *Science* 301:904.

Karpati, AM, MC Perrin, T. Matte, J. Leighton, J. Schwartz, and RG Barr. 2004. Pesticide spraying for West Nile virus control and emergency department asthma visits in New York City, 2000. *Environmental Health Perspectives* 112(11):1183-1187.

Key, P., M. DeLorenzo, K. Gross, K. Chung, and A. Clum. 2005. Toxicity of the mosquito control pesticide Scourge to adult and larval grass shrimp (*Paleomonetes pugio). Journal of Environmental Science and Health, Part B* 40:585-594.

Kiesecker, JM. 2002. Synergism between trematode infection and pesticide exposure: A link to amphibian limb deformities in nature? *Proceedings of the National Academy of Sciences* 99:9900-9904.

Kilpatrick, AM, LD Kramer, SR Campbell, EO Alleyne, AP Dobson, and P. Daszak. 2005. West Nile virus risk assessment and the bridge vector paradigm. *Emerging Infectious Diseases* 11(3):425-429.

Kitron, U., J. Michael, J. Swanson, and L. Haramis. 1997. Spatial analysis of the distribution of LaCrosse encephalitis in Illinois, using a geographic information system and local and global spatial statistics. *American Journal of Tropical Medicine and Hygiene* 57:469-475.

Klaasen, C., M. Amdur, and J. Doull. 1986. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 3rd Ed. Macmillan, New York, NY.

Kluh, S., P. DeChant, and JE Hazelrigg. 2005. Routine mosquito control in underground storm drains in urban Los Angeles. Presentation 25, *71st Annual Meeting of the American Mosquito Control Association*. Vancouver, BC. April 4.

Knapp, RA, and KR Matthews. 2000. Non-native fish introductions and the decline of the mountain yellow-legged frog from within protected areas. *Conservation Biology* 14(2):428-438.

Kramer,VL, JN Collins, K. Malamud-Roam, and C. Beesley. 1995. Reduction of *Aedes dorsalis* by enhancing tidal action in a Northern California marsh. *Journal of the American Mosquito Control Association* 11(4): 389-395

Kreeger, DA, and RIE Newell. 2000. Trophic complexity between producers and invertebrate consumers in salt marshes. pp. 187-220. In: Weinstein, MP, and DA Kreeger (eds.). *Concepts and Controversies in Tidal Marsh Ecology*. Kluwer Acadmic Publishers, Boston, MA. 875 pp.

Kuenzler, EJ, and HL Marshall. 1973. *Effects of Mosquito Control Ditching on Estuarine Ecosystems*. Report No. 81, Water Resource Research Institute, University of North Carolina. 83 pp.

Kunimatsu, T., T. Yamada, K. Ose, O. Sunami, Y. kamita, Y. Okuno, T. Seki, and I. Nakatsuka. 2002. Lack of (anti-) androgenic or estrogenic effects of three pyrethroids (esfenvalerate, fenvalerate, and permethrin) in the Hershberger and uterotrophic assays. *Regulatory Toxicology and Pharmacology* 35:227-237.

Kushlan, JA. 1979. Effects of helicopter censuses on wading bird colonies. *Journal of Wildlife Management* 43:756-760.

Lacey, LA, and RW Merritt. 2003. The safety of bacterial microbial agents used for black fly and mosquito control in aquatic environments. pp. 151-168. In: Hokkanen, HMT, and AE Hajek (eds.). *Environmental Impacts of Microbial Insecticides: Need and Methods for Risk Assessment*. Kluwer Academic Publishers, Dordrecht, Netherlands.

Lathrop, RG, MP Cole, and RD Showalter. 2000. Quantifying the habitat structure and spatial pattern of New Jersey (U.S.A.) salt marshes under different habitat regimes. *Wetlands Ecology and Management* 8:163-172.

Laufer, H. 1982. *The Effect of Hormonal Pollutants on Aquatic Crustacea and the Surrounding Environment*. Institute of Water Resources, The University of Connecticut, Storrs, CT. 38 pp.

Lee, GF, and A. Jones-Lee. 2005. Urban stormwater runoff aquatic life toxicity: an update. *Stormwater* 6(6):62-67.

Lee, BM, and GI Scott. 1989. Acute toxicity of temephos, fenoxycarb, diflubenzuson, and methoprene and *Bacillus thuringiensis* var. *israelensis* to the mummichog (*Fundulus heteroclitos*). *Bulletin of Environmental Contamination and Toxicology* 43(6):827-832.

Lent, RA, T. Hruby, DP Cowan, and TS Litwin. 1990. *Open Marsh Water Management on Great South Bay, Islip, New York*. Seatuck Foundation, Islip, NY.

Leonard, MD (ed.) 1926. A list of the insects of New York with a list of spiders and certain allied groups. *Cornell University Agricultural Station Memoir* 101:1-1121.

Lesser, CR. Undated(1). *Open Marsh Water Management: A Source Reduction Technique for Mosquito Control*. Delaware Department of Natural Resources. Unpaged.

Lesser, CR. Undated(2). *Comparative Field Trial Efficacy of Anvil 10+10 and Scourge 18:54 on Oc. sollicitans and Ae. vexans in Delaware*. PowerPoint, Delaware Division of Fish and Wildlife.

Leiss, M., and PC Von Der Ohe. 2005. Analyzing effects of pesticides on invertebrate communities in streams. *Environmental Toxicology and Chemistry* 24(4):954-965.

Loeb, M., SJ Elliott, B. Gibson, M. Fearon, R. Nosal, M. Drebot, C. D'Cuhna, D. Harrington, S. Smith, P. George, and J. Eyles. 2005. Protective behavior and West Nile virus risk. *Emerging Infectious Diseases* 11(9):1433-1436.

Louda, SM, RW Pemberton, MT Johnson, and PA Follett. 2003. Nontarget effects – the Achilles heel of biological control? Retrospective analyses to reduce risk associated with biocontrol introductions. *Annual Reviews of Entomology* 48:365-396.

Luber, G., K. Johnson, C. Rubin, H. Schurz Rogers, M. Currier, and D. Barr. 2003. Pesticide exposure resulting from West Nile Virus mosquito control, Mississippi, 2002. Centers for Disease Control and Mississippi State Department of Health, Presentation at the *4th Annual Conference on West Nile Virus in the United States*, February 9-11, New Orleans, LA.

Lyman, WJ, WF Reehl, and DH Rosenblatt. 1982. *Handbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds.* McGraw-Hill Book Company. New York, New York.

Mackay, D. 1991. *Multimedia Environmental Models: The Fugacity Approach*. Lewis Publishers, Chelsea, MI. 257 pp.

Madder, DJ. 1980. Studies on the dissipation of diflubenzuron and methoprene from shallow prairie pools. *The Canadian Entomologist*. pp.173-177. As cited in Westchester, 2001.

Marani, M., E. Belluco, A., D'Alpaos, A, Defina, S. Lanzoni, and A. Rinaldo. 2003. On the drainage density of tidal networks. *Water Resources Research* 39(2):1040 (doi:10.1029/2002WR001051).

Maund, SJ, KZ Travis, P. Hendley, JM Giddings, and KR Solomon. 2001. Probabilistic risk assessment of cotton pyrethroids: V. Combining landscape level exposures and ecotoxicological effects data to characterize risks. *Environmental Toxicology and Chemistry* 20:687–692.

McClintock, JT, CR Schaffer, and RD Sjoblad. 1995. A comparative review of the mammalian toxicity of *Bacillus thuringiensis*-based pesticides. *Pesticide Science* 45:95-105.

McIvor, CC, and WE Odum. 1988. Food, predation risk, and microhabitat selection in a marsh fish assemblage. *Ecology* 69(5):1341-1351.

McKelvey, W., JG Brody, A. Aschengrau, and CH Swartz. 2003. Association between residence on Cape Cod, Massachusetts, and breast cancer. *Annals of Epidemiology* 14:89-94.

McKenney, CL, and DM Celestial. 1996. Modified survival, growth and reproduction in an estuarine mysid (*Mysidopsis bahia*) exposed to a juvenile hormone analogue through a complete life cycle. *Aquatic Toxicology* 35(1):11-20.

McLetchie, K, and S. Goodbred. Unpublished, but dated 2003. *South Shore Salt Marshes: A Review of Attributes and their Indicators*. Draft, prepared for The Nature Conservancy, Cold Spring Harbor, NY. 17 pp.

McNelly, JR. 1989. The CDC Trap as a special monitoring tool. *Proceedings of the New Jersey Mosquito Control Association* 76:26-33.

Metzger, ME., DF Messer, CL Beitia, CM Myers, and VL Kramer. 2002. The dark side of stormwater runoff management: disease vectors associated with structural BMPs. *Stormwater* 2(2):24-34.

Meyerson, LA, KA Vogt, and RH Chalmers. 2000. Linking the success of *Phragmites* to the alteration of ecosystem nutrient cycles. pp. 827-844. In: Weinstein, MP, and DA Kreeger (eds.). *Concepts and Controversies in Tidal Marsh Ecology*. Kluwer Acadmic Publishers, Boston, MA. 875 pp.

Milam, CD, JL Farris, and JD Wilhide. 2000. Evaluating mosquito control pesticides for effect on target and non-target organisms. *Archives of Environmental Contamination and Toxicology* 39:324-328.

Miller, WR, and FE Egler. 1950. Vegetation of the Wequetequock-Pawcatuck tidal marshes, Connecticut. *Ecological Monographs* 20:143-172.

Mitsch, WJ, and JG Gosselink. 2000. *Wetlands.* 3rd Ed. John Wiley and Sons, New York, NY. 920 pp.

Mittal, PK. 2003. Biolarvicides in vector control: challenges and prospects. *Journal of Vector-Borne Diseases* (40):20–32.

Montgomery, WG. 1998. Rumney Marsh, Park Avenue restorations project. *Northeastern Mosquito Control Association*, December. pp. 10-11.

Moore, JC, JC Dukes, JR Clark, J. Malone, CF Hallmon, and PG Hester. 1993a. Downwind drift and deposition of malathion on human targets from ground ultra-low volume mosquito sprays. *Journal of the American Mosquito Control Association* 9(2):138-142.

Moore, CG, RG McLean, CJ Mitchell, RS Nasci, TF Tsai, CH Calisher, AA Marfin, PS Moore, and DJ Gubler. 1993b. *Guidelines for Arbovirus Surveillance Programs in the United States*. Division of Vector-Borne Infectious Diseases, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Public Health Service, U.S. Department of Health and Human Services. Fort Collins, CO.

Mount, GA. 1998. A critical review of ultra-low volume aerosols of insecticide applied with vehicle-mounted generators for adult mosquito control. *Journal of the American Mosquito Control Association* 14(3):305-334.

Mount, GA. 1996. A review of ultra-low volume aerial sprays of insecticide for mosquito control. *Journal of the American Mosquito Control Association* 12(4):601-618

Muller-Hohnsen, H. 1999. Chronic sequelae and irreversible injuries following acute pyrthroid intoxication. *Toxicology Letters* 107:161-176.

NAS. 1983. *Risk Assessment in the Federal Government: Managing the Process*. National Academy of Sciences, Washington, DC. 192 pp.

NCIPM. 2004a. *Bacillus thuringiensis subsp. israelensis – Biological insecticide fact sheet*. National Center for Integrated Pest Management. http://www.ncipm.org.in/BacillusSJS.htm. Accessed on 08/20/04.

NCIPM. 2004b. Bacillus sphaericus – Biological insecticide fact sheet. National Center for Integrated Pest Management. http://www.ncipm.org.in/BacillusSPHERICUS.htm. Accessed on 08/20/04.

NLM. 2005. *Hazardous Substances Database*. National Library of Medicine.

NPIC. 2000. *Piperonyl Butoxide Technical Fact Sheet*. National Pesticide Information Center. http://npic.orst.edu/factsheets/pbotech.pdf. Accessed on 7/14/04.

NYCDHMH. 2004. *Comprehensive Mosquito Surveillance and Control Plan*. New York City Department of Health and Mental Hygiene, New York, NY. 36 pp.

NYCDOH. 2001. *Adult Mosquito Control Programs Draft Environmental Impact Statement (DEIS)*. New York City Department of Health, New York, NY.

NYSDEC. 2005. *Comprehensive Wildlife Conservation Strategy: Appendix A5: Comprehensive Wildlife Conservation Strategy Species Group Reports for Insects*. Revised Draft. New York State Department of Environmental Conservation, Albany, NY. 140 pp.

NYSDEC. 1996a. *Urban/Storm water Runoff Management Practices Catalogue for Nonpoint Pollution Prevention and Water Quality Protection in New York State*. Urban/Storm water Runoff Management Practices Subcommittee of the New York State Nonpoint Source Management Practices Task Force, New York State Department of Environmental Conservation, Albany, NY. 86 pp.

NYSDEC. 1996b. *Bacillus sphaericus (VectoLex) Registration of New Active Ingredient*. Bureau of Pesticides & Radiation, Pesticide Product Registration, New York State Department of Environmental Conservation, Albany, NY.

NYSDEC New York Natural Heritage Program. 2002. *Natural Heritage Report on Rare Species and Ecological Communities*. New York State Department of Environmental Conservation, Latham, NY.

NYSDOH. 2001a. *New York State West Nile Virus Response Plan – Guidance Document*. New York State Department of Health, Albany, NY. 97 pp.

NYSDOH. 2001b. *Health Advisory: Tick and Insect Repellents.* New York State Department of Health.

Narahashi, T. 1992. Nerve membrane Na+ channels as targets of insecticides. *Trends in Pharmacological Science* 13:236-241.

National Pesticide Telecommunications Network. 2001. *Malathion.*

National Pesticide Telecommunications Network. 2000. *Piperonyl Butoxide.*

National Pesticide Telecommunications Network. 1998. *Pyrethrins and Pyrethrods.*

Niedowski, N. 2000. *New York State Salt Marsh Restoration and Monitoring Guidelines*. NYS Department of State, Albany, NY, and NYS Department of Environmental Conservation, East Setauket, NY. 135 pp.

Niemi, CJ, AE Hershey, L. Shannon, JM Hanowski, A. Lima, RP Axler, and RP Regal. 1999. Ecological effects of mosquito control on zooplankton, insects, and birds. *Environmental Toxicology and Chemistry* 10:1219-1227.

Nixon, SW. 1982. *The Ecology of New England High Salt Marshes: A Community Profile*. FWS/OBS-81/55, US Fish and Wildlife Service, Washington, DC. 70 pp.

Nixon, SW. 1980. Between coastal marshes and coastal waters – a review of twenty years of speculation and research on the role of salt marshes in estuarine production and water chemistry. pp. 437-525. In: Hamilton, P., and KB MacDonald (eds). *Estuarine and Wetland Processes with Emphasis on Modeling*. Plenum Press, New York, NY. 653 pp.

O'Brien & Gere. 1995. *Determination of Effects of AerialApplications of Dibrom on Non-target Organisms in the Cicero Swamp*. Draft, submitted to Onondaga County Health Department, Syracuse, NY.

Odum, EP. 2000. Tidal marshes as outwelling/pulsing systems. pp. 3-7. In: Weinstein, MP, and DA Kreeger (eds.). *Concepts and Controversies in Tidal Marsh Ecology*. Kluwer Acadmic Publishers, Boston, MA. 875 pp.

Odum, WE. 1988. Comparative ecology of tidal freshwater and salt marshes. *Annual Review of Ecology and Systematics* 19:147-176.

Odum, WE, JS Fisher, and JC Pickral. 1979. Factors controlling the flux of particulate organic carbon from estuarine wetlands. pp. 69-80. In: Livingston, RJ (ed.). *Ecological Processes in Coastal and Marine Systems*. Plenum Press, New York. 548 pp.

O'Malley, C. 1989. Guidelines for larval surveillance. *Proceedings of the New Jersey Mosquito Control Association* 76:45-55.

Olmstead, AW, and GL LeBlanc. 2001. Low exposure concentration effects of methoprene on endocrine-regulated processes in the crustacean *Daphnia magna*. *Toxicology Sciences* 62(2):268-273.

Oransky, S., B. Roseman, D. Fish, T. Gentile, J. Melius, ML Cartter, and JL Hadler. 1989. Epidemiologic notes and reports: seizures temporally associated with use of DEET insect repellent—New York and Connecticut. *Morbidity and Mortality Weekly Report* 38(39):678-680.

Oros, DR, and I. Werner. 2005. *Pyrethroid Insecticides: An Analysis of Use patterns, Distributioons, Potential Toxicity and fate in the Sacramento-San Joaquin delta and Central valley*. Contribution 415, San Francisco Estuary Institute, Oakland CA. 112 pp.

Pastorok, RA, HR Akcakaya, H. Regan, S. Ferson, and SM Bartell. 2003. Role of ecological modeling in risk assessment. *Human and Ecological Risk Assessment* 9(4):939-972.

Pastorok, RA, MK Butcher, and RD Nielsen. 1996. Modeling wildlife exposure to toxic chemicals: Trends and recent advances. *Human and Ecological Risk Assessment* 2:444-480.

Payen, GG, and JD Costlow. 1977. Effects of a juvenile hormone mimic on male and female gametogenesis of the mud crab, *Rhithropanopeus harrisii* (Gould) (Brachyura: Xanthidac). *Biological Bulletin, Marine Biology Laboratory, Woods Hole* 152(2):199-208.

Peterson, RKD, PA Macedo, and RS Davis. 2005. A human health risk assessment for West Nile virus and insecticides used in mosquito management. *Environmental Health Perspectives*, published online October 28. doi:10.1289/ehp.8667; http://dx.doi.org/

Pethick, JS. 1992. Saltmarsh geomorphology. pp. 41-62. In: Allen, JRL, and K. Pye (eds.). *Saltmarshes: Morphodynamics, Conservation, and Engineering Significance*. Cambridge University Press, New York, NY. 79 pp.

Pimental, D. 1995. Amounts of pesticides reaching target (1):s: environmental impacts and ethics. *Journal of Agricultural and Environmental Ethics* 8(1):17-29.

Pinkney, AE, PC McGowan, DR Murphy, TP Lowe, DW Sparling, and LC Ferrington. 2000. Effects of the mosquito larvicides temephos and methoprene on insect populations in experimental ponds. *Environmental Toxicology and Chemistry* 19(3):678-684.

Pomeroy, LR, and J. Imberger. 1981. The physical and chemical environment. pp. 21-36. In: Pomeroy, LR, and RG Wiegert (eds.). *The Ecology of a Salt Marsh*. Springer-Verlag, New York, NY. 271 pp.

Pomeroy, LR, and RG Wiegert (eds.). 1981. *The Ecology of a Salt Marsh*. Springer-Verlag, New York, NY. 271 pp.

Preziosi, D., J. Durda, M. Behum, R. Pastorek, DJ Tonjes, and D. Ninivaggi. 2005. Assessing potential aquatic risks associated with use of mosquito control chemicals, Suffolk County, Long Island, NY. Poster presentation. 26th Annual Meeting, Society of Toxicology and Chemistry. Baltimore, MD. November 13-17.

Qiu, H., H. Won Jun, and J. McCall. 1998. Pharmacokinetics, formulation and safety of insect repellent N,N-diethyl-3-methylebenzamide (DEET): a review. *Journal of the American Mosquito Control Association* 14(1):12-27.

Quate, LW. 1960. *Guide to the Insects of Connecticut Part VI. The Diptera or True Flies of Connecticut. Seventh Fascicle: Psychodidae*. State Geological and Natural History Bulletin 92. 54 pp.

Quistad, GB, DA Schooley, LE Staigner, BJ Bergot, BH Sleight, and KJ Macek. 1976. Environmental degradation of the insect growth regulator methoprene. 9. Metabolism by bluegill fish. *Pesticides Biochemistry and Physiology* 6(6):523-529.

Raloff. 2006. A little less green? Studies challenge the benign image of pyrethroid insecticides. *Science News* 169(5):74.

Read, N. 2001. Risks and benefits of larval mosquito control with *Bti* or methoprene. Presentation, *Society of Wetland Scientists* annual meeting. May 28. Chicago, IL.

Redfield, AC. 1972. Development of a New England salt marsh. *Ecological Monographs* 42(2):1-23.

Reese, AJ, and HH Presler. 2005. Municipal stormwater system maintenance: an assessment of current practices and methodology for upgrading programs. *Stormwater* 6(6):36-61.

Reinert, WC. 1989. The New Jersey light trap: an old standard for most mosquito control programs. *Proceedings of the New Jersey Mosquito Control Association* 76:17-25.

Reinert, SE, FC Golet, and WR DeRagon. 1981. Avian use of ditched and unditched salt marshes in southeastern New England. *Transactions of the Northeastern Mosquito Control Association* 17:1-23.

Relyea, R. 2005. The impact of insecticides and herbicides on the biodiversity and productivity of aquatic communities. *Ecological Applications* 15(2):618-627.

Relyea, RA. 2004. The lethal impacts of Roundup and predatory stress on six species of North American tadpoles. *Archives of Environmental Contamination* 48:351-357.

Relyea, RA, and N. Mills. 2001. Predator-induced stress makes the pesticide carbaryl more deadly to gray tree frog tadpoles (*Hyla versicolor*). *Proceedings of the National Academy of Sciences* 98:2491-2496.

Reuter, KC, and RN Foster. 2000. Success with reduced rates of carbaryl, malathion, and acephate sprays. Section II.5. In: Cunningham, GL, and MW Sampson (Technical Coordinators). *Grasshoppers: Their Biology, Identification, and Management Handbook*. US Department of Agriculture, Washington, DC. Paged in sections.

Richards, AG, Jr. 1938. Mosquitoes and mosquito control on Long Island, New York, with particular reference to the salt marsh problem. *New York State Museum Bulletin* 316:85-172.

Rockel, EG. 1969. Marsh physiography: influence on the distribution of organisms. *Proceedings of the New Jersey Mosquito Extermination Association* 56:102-116.

Roman, CT, WA Niering, and RS Warren. 1984. Salt marsh vegetation change in response to tidal restriction. *Environmental Management* 8:141-150.

Romanowski, M., and RD Huggins. 1989. Complaints: an underrated surveillance parameter. *Proceedings of the New Jersey Mosquito Control Association* 76:39-41.

Rose, R. 2001. Pesticides and public health: integrated methods of mosquito management. *Emerging Infectious Diseases* 7(1):17-23.

Russell, TL, MD Brown, DM Purdie, PA Ryan, and BH Kay. 2003. Efficacy of VectoBac (*Bacillus thuringiensis* variety *israelensis*) formulations for mosquito control in Australia. *Journal of Economic Entomology* 96(6):1786-1791.

Saito, K., Y. Tomigahara, N. Ohe, N. Isobe, I. Nakatsuka, and H. Kaneko. 2000. Lack of significant estrogenic or antiestrogenic activity of pyrethroid insecticides in thre in vitro assays based on classic estrogen receptor alpha-mediated mechanisms. *Toxicology Science* 57:54-60.

Sample, B., MS Alpin, RA Efroymson, GW Suter, and CJE. Welsh. 1997. *Methods and Tools for Estimation of the Exposure of Terrestrial Wildlife to Contaminants.* Oak Ridge National Laboratory. ORNL/TM-13391.

Sample, B., D. Opresko, and G. Suter. 1996. *Toxicological Benchmarks for Wildlife.* 1996 Revision. ES/ER/TM-86/R3.

Sample, B. and GW Suter. 1994. *Estimating Exposure of Terrestrial Wildlife to Contaminants.* Oak Ridge National Laboratory. ES/ER/TM-125.

SandozAgro, Inc. 1994. *Aquatic Risk Assessment for Methoprene Based Formulations*. Toxicology Department, SandozAgro, Inc. 9pp.

Schaefer, CH, and EF Dupras. 1973. Insect development inhibitors. 4. Persistence of ZR-515 in water. *Journal of Economic Entomology* 66(1):923-925. As cited in Westchester, 2001.

Schulz, D., J. Sullivan, and G. Robinson. 2002. Juvenile hormone and octopamine in the regulation of division of labor in honey bee colonies. *Hormones and Behavior* 42:222-231.

Shapiro, H., and S. Micucci. 2003. Pesticide use for West Nile virus. *Canadian Medical Association Journal* 168(11):1427-1430.

Shisler, JK. 1973. Pioneer plants on spoil piles associated with mosquito ditching. *Proceedings of the New Jersey Mosquito Extermination Association* 60:135-141.

Simenstad, C.A. and R.M. Thom. 1996. Functional Equivalency Trajectories of the Restored Gog-Le-Hi-Te Estuarine Wetland. *Ecological Applications* 6(1): 38-56.

Sinha, C., and GS Shukla. 2003. Species variation in pesticide-induced blood-brain barrier dysfunction. *Human exposure Toxicology* 22:647-652.

Sjrogen, RD, DP Batzer, and MA Jeunemann. 1986. Evaluation of methoprene, temephos, and *Bacillus thuringiensis* var. *israelensis* against *Coquillettidia perturbans* larvae in Minnesota. *Journal of the American Mosquito Control Association* 2(3):276-279.

Smith, JB. 1904. *Report of the NJ State Agricultural Experiment Station upon the Mosquitoes Occuring within the State, Their Habits, Life History, etc.* MacCrellish & Quigley, Trenton, NJ. 482 pp.

Smith, TM, and GW Stratton. 1986. Effects of synthetic pyrethroid insecticides on nontarget organisms. *Residue Reviews* 97:93-120.

Sparling, DW, G. Linder, and CA Biship. 2000. *Ecotoxicology of Amphibians and Reptiles.* SETAC Press, Pensacola, FL.

Spielman, A. 2001. Structure and seasonality of neararctic *Culex pipiens* populations. pp. 220-234. In: White, DJ, and DL Morse (eds.). *West Nile Virus: Detection, Surveillance, and Control*. Annals of the New York Academy of Science, V. 951. New York, NY. 374 pp.

Spielman, A., and M. D'Antonio. 2001. *Mosquito.* Hyperion, New York, NY. 247 pp.

Spurlock, F. 2003. *Probabilistic Estimation of Dissolved Phase Pyrethroid Concentrations from Whole Water Analytical Data.* Report No. EH03-06. California Department of Pesticide Regulation, Environmental Monitoring Branch, Sacramento, CA. 36 pp.

Stewart, PG, and L. Springer-Rushia. 1998. *A Field Guide to Long Island's Freshwater Wetlands*. Museum of Long Island Natural Sciences, Stony Brook, NY. 90 pp.

Stone, A. 1964. *Guide to the Insects of Connecticut Part VI. The Diptera or True Flies of Connecticut. Ninth Fascicle: Simuliidae and Thaumaleidae*. State Geological and Natural History Bulletin 97. 126 pp.

Suter, GW. 1993. *Ecological Risk Assessment*. Lewis Publishers, Chelsea, Michigan.

Suter, GW, RA Efroymson, BA Sample, and D. Jones. 2000. *Ecological Risk Assessment for Contaminated Sites*. Lewis Publishers, Boca Raton, FL.

Talent, LG. 2005. Effect of temperature on toxicity of a natural pyrethrin pesticide to green anole lizards (*Anole carolinensis*). *Environmental Toxicology and Chemistry* 24:3113-3116.

Taylor, J. 1998. *Guidance for Meeting U.S. Fish and Wildlife Service Trust Resource Needs When Conducting Coastal Marsh Management for Mosquito Control on Region 5 National Wildlife Refuges*. US Fish and Wildlife Services. 26 pp.

Taylor, N. 1938. A preliminary report on the salt marsh vegetation of Long Island, New York. *New York State Museum Bulletin* 316:21-84.

Teal, JM. 1986. *The Ecology of Regularly Flooded Salt Marshes of New England: A Community Profile*. Biological Report 85(7.4), US Fish and Wildlife Service, Washington, DC. 61 pp.

Teal, J., and M. Teal. 1969**.** *Life and Death of the Salt Marsh.* Little, Brown, Boston, MA. 278 pp.

Templeton, NS, and H. Laufer. 1983. The effects of a juvenile hormone analog (Altosid ZR-515) on the reproduction and development of *Daphnia Magna* (Crustacea: Cladocera). *International Journal of Invertebrate Reproduction* 6(2):99-110.

Thompson, KM, DE Burmaster, and EAC Crouch. 1992. Monte Carlo techniques for quantitative uncertainty analysis in public health risk assessments. *Risk Analysis* 12(1):53-63.

Travis, KZ, and P. Hendley. 2001. Probabilistic risk assessment of cotton pyrethroids: IV. Landscape-level exposure characterization. *Environmental Toxicology and Chemistry* 20:679–686.

USDA. 2005. *Agricultural Research Service Pesticide and Properties Database*. US Department of Agriculture

USDA. 1982. *SCS Agriculture Handbook Number 590: Ponds - Planning, Design, Construction*. US Department of Agriculture, Washington, DC.

USEPA. 2005a. *Screening Ecological Risk Assessment for the Reregistration of Piperonyl Butoxide Insecticide Synergist*. U.S. Environmental Protection Agency, Washington, DC.

USEPA. 2005b. Permethrin. Notice of availability and of risk assessments and opening of docket. *Federal Register* 70(168):51790-51792.

USEPA. 2005c. *Ecological Risk Assessment Glossary*. US Environmental Protection Agency. http://www.erg.com/portfolio/elearn/ecorisk/html/resource/glossary.html#E. Accessed on 9/05.

USEPA. 2005d. *User Manual for EXPRESS, the "EXAMS-PRZM Exposure Simulation Shell."* Version 1.00.00.04, ß test version, May 5. US Environmental Protection Agency, Washington, DC. Available at: http://www.epa.gov/ceampubl/swater/express/.

USEPA. 2005e. *Permethrin: Updated Revised Occupational and Residential Exposure Assessment for the Reregistration Eligibility Decision Document*. US Environmental Protection Agency. June 29.

USEPA. 2005f. *Human Health Risk Assessment, Pyrethrins.* US Environmental Protection Agency Office of Pesticide Programs, Health Effects Division.

USEPA. 2005g. *Malathion for Mosquito Control*. US Environmental Protection Agency. www.epa.gov/pesticides/health/mosquitoes/malathion4mosquitoes.htm

USEPA. 2004a. *Risk Assessment Principles and Practices*. EPA/100/B-04/001. Staff Paper, Office of the Science Advisor, US Environmental Protection Agency, Washington, DC.

USEPA. 2004b. *Overview of the Ecological Risk Assessment Process in the Office of Pesticide Programs, U.S. Environmental Protection Agency – Endangered and Threatened Species Effects Determinations*. US Environmental Protection Agency, Office of Prevention, Pesticides and Toxic Substances, Office of Pesticide Programs, Washington, DC.

USEPA. 2004c. *Example Exposure Scenarios*. EPA600/R-03/036. US Environmental Protection Agency, National Center for Environmental Assessment, Washington, DC.

USEPA. 2004d. *Risk Assessment Guidance for Superfund (RAGS): Volume 1 – Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment), Final*. EPA/540/R/99/005. OSWER 9285.7-02EP. PB99-963312. US Environmental Protection Agency, Office of Superfund Remediation and Technology Innovation, Washington, DC.

USEPA. 2004e. *Region IX. Preliminary Remediation Goals Table*. US Environmental Protection Agency, San Francisco, CA.

USEPA. 2004f. *Malathion Use as a Mosquitocide and Potential Risk to Listed Salmonids*. US Environmental Protection Agency, Memorandum from N. Birchfield to J. Leyhe, November 10.

USEPA. 2004g. AQUATOX (Release 2). Modeling Environmental Fate and Ecological Effects in Aquatic Ecosystems – Volume 1, User's Manual. EPA-823-R-04-001. US Environmental Protection Agency Office of Water, Washington, DC.

USEPA. 2004h. *AQUATOX (Release 2). Modeling Environmental Fate and Ecological Effects in Aquatic Ecosystems – Volume 2, Technical Documentation*. EPA-823-R-04-002. US Environmental Protection Agency Office of Water, Washington, DC.

USEPA. 2004i. *DEET Factsheet* http://www.epa.gov/pesticides/factsheets/chemicals/deet.htm Office of Prevention, Pesticides, and Toxic Substances.

USEPA. 2004j. *Permethrin - Third Report of the Hazard Identification Assessment Review Committee*. US Environmental Protection Agency HED Records Center Series 361. Science Reviews – File R100612. May 12.

USEPA. 2003a. *Guidance on Selecting the Appropriate Age Groups for Assessing Childhood Exposure to Environmental Contaminants.* EPA/630/P-03/003A. US Environmental Protection Agency, Risk Assessment Forum, Washington, DC.

USEPA. 2003b. *CSFII Analysis of Food Intake Distributions*. EPA/600/R-03/029. US Environmental Protection Agency, National Center for Environmental Assessment, Office of Research and Development, Washington, DC.

USEPA. 2002a. *IIFG Decision Document on Tolerance Reassessment for Methoprene*. US Environmental Protection Agency, Washington, DC.

USEPA. 2002b. Pesticide Fact Sheets: Synthetic Pyrethroids for Mosquito Control. United States Environmental Protection Agency. http://www.epa.gov/pesticides/factsheets/pyrethroids4mosquitos.htm. Accessed on 7/12/04.

USEPA. 2002c. *Malathion for Mosquito Control*. United States Environmental Protection Agency. http://www.epa.gov/pesticides/factsheets/malathion4mosquitos.htm. Accessed on 7/13/2004.

USEPA. 2002d. *Child-specific Exposure Factors Handbook*. EPA-600-P-00-002B. National Center for Environmental Assessment, Office of Research and Development, US Environmental Protection Agency, Washington DC.

USEPA. 2001a. *Update of the March 1991 Methoprene R.E.D. Fact Sheet*. US Environmental Protection Agency, Washington, DC.

USEPA. 2001b. *Risk Assessment Guidance for Superfund, Volume 3, Part A: Process for Conducting Probabilistic Risk Assessment (RAGS 3A).* EPA 540-R-02-002. US Environmental Protection Agency, San Francisco, CA.

USEPA. 2001c. *Risk Assessment Guidance for Superfund Volume 3, Part A: Process for Conducting Probabilistic Risk Assessment (RAGS 3A).* EPA 540-R-02-002. US Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC.

USEPA. 2000a. *Revised Human Health and Ecological Risk Assessment for Malathion*. US Environmental Protection Agency, Office of Pesticide Programs, Washington, DC.

USEPA. 2000b. *For Your Information - Larvicides for Mosquito Control*. EPA 735-F-00-002. US Environmental Protection Agency, Washington, DC.

USEPA. 2000c. *Malathion: Human Health Risk Assessment for the Reregistration Eligibility Document.* Chemical No. 057701, Case No. 0248. US Environmental Protection Agency.

USEPA. 1999a. *Pesticide fact sheet - Bacillus sphaericus Serotype H5a5b strain 2362 (128128).* US Environmental Protection Agency. http://www.epa.gov/pesticides/biopesticides/ingredients/factsheets/factsheet_128128.htm.

USEPA. 1999b. *Organophosphate Pesticides in Food - A Primer on Reassessment of Residue Limits.* US Environmental Protection Agency. http://www.epa.gov/pesticides/op/primer.htm. Accessed on 7/13/04.

USEPA. 1999c. *Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities*. EPA530-D-99-001B. US Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

USEPA. 1999d. *Human Health and Ecological Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes - Background Document*. US Environmental Protection Agency, Office of Solid Waste, Washington, DC.

USEPA. 1999e. *Screening Level Ecological Risk Assessment Protocol for Hazardous Waste Combustion Facilities*. EPA530-D-99-001B. US Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

USEPA. 1999f. *List of Chemicals Evaluated for Carcinogenic Potential*. US Environmental Proetection Agency, Washington, DC.

USEPA. 1998a. *Guidelines for Ecological Risk Assessment*. EPA/630/R-95/002F. US Environmental Protection Agency, Washington, DC.

USEPA. 1998b. *Reregistration Eligibility Decision (RED): Bacillus thuringiensis.* EPA738-R-98-004. US Environmental Protection Agency, Office of Pesticides, Prevention and Toxic Substances, Washington, DC.

USEPA. 1998c. *Bacillus sphaericus* (VectoLex) tolerance requirement exemption 8/98. United States Environmental Protection Agency. *Federal Register*, September 10, pp. 8594-48597.

USEPA. 1998d. *Reregistration Eligibility Decision (RED) – DEET*. US Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances.

USEPA. 1997a. *Exposure Factors Handbook - Volume 1. General Factors*. EPA/600/P-95/002Fa. US Environmental Protection Agency, Washington, DC.

USEPA. 1997b. *Ecological Risk Assessment Guidance for Superfund: Process for Conducting Ecological Risk Assessment (RAGS 3A).* EPA 540-R-97-006. US Environmental Protection Agency, Office of Emergency Response, Washington, DC.

USEPA. 1997c. *Guiding Principles for Monte Carlo Analysis*. EPA/630/R-97-001. US Environmental Protection Agency, Office of Research and Development, Washington, DC.

USEPA. 1993a. *Reference Dose (RfD) Description and Use in Health Risk Assessments.* US Environmental Protection Agency. Available at: http://www.epa.gov/iris/rfd.htm. Last accessed: 07/25/05.

USEPA. 1993b. *Wildlife Exposure Factors Handbook*. US Environmental Protection Agency, Office of Research and Development, Washington, DC. Volumes I and II.

USEPA. 1992. *Reregistration Eligibility Decision (RED): Allium sativum (Garlic)*. US Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

USEPA. 1991a. *R.E.D. facts: Methoprene*. EPA 738-F-91-104. US Environmental Protection Agency, Washington, DC.

USEPA. 1991b. *Risk Assessment Guidance for Superfund (RAGS): Volume 1 – Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals); Interim Final*. EPA/540/R-92/003. US Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC.

USEPA. 1989. *Risk Assessment Guidance for Superfund (RAGS): Volume 1 – Human Health Evaluation Manual (Part A). Interim Final.* US Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, DC.

USFWS. 1999. *Concerns and Issues about Mosquito Control on the National Wildlife Refuges in the Northeast*. US Fish and Wildlife Service, Region V. 53 pp.

Valent Biosciences Corporation. Undated. VectoBac and VectoLex product fact sheets. http://www.valentbiosciences.com. Accessed 08/20/04.

Vandenplas, O., JP Delwiche, J. Auverdin, UM caroyen, and FB Cangh. 2000. Asthma to tetramethrin. *Allergy* 55:417-418.

WHO. 2005. *Safety of Pyrethroids for Public Health Use*. World Health Organization, Communicable Disease Control Prevention and Eradiation, WHO Pesticide Evaluation Scheme and Protection of the Human Environment, Geneva, Switzerland. Available at: http://whqlibdoc.who.int/hq/2005/WHO_CDS_WHOPES_GCDPP_2005.10.pdf.

WHO. 1999. *Environmental Health Criteria 217, Microbial Pest Control Agent Bacillus thuringiensis.* International Programme on Chemical Safety, World Health Organization. Geneva, Switzerland.

WHO. 1984. *Data Sheets on Pesticides No. 47 Methoprene.* Food and Agriculture Organization, World Health Organization, IPCS INCHEM.

WHO-FAO. 1996. *WHO-FAO Data Sheets on Pesticides: Resmethrin*. World Health Organization and Food and Agriculture Organization of the United Nations. http://www.inchem.org/documents/pds/pds/pest83_e.htm#1.0. Accessed on 7/12/04.

WHO-FAO. 1990a. *WHO-FAO Data Sheets on Pesticides: Sumithrin*. World Health Organization and Food and Agriculture Organization of the United Nations. http://www.inchem.org/documents/ehc/ehc/ehc96.htm. Accessed on 7/13/04.

WHO-FAO. 1990b. *WHO-FAO Data Sheets on Pesticides: Permethrin*. World Health Organization and Food and Agriculture Organization of the United Nations. http://www.inchem.org/documents/ehc/ehc/ehc96.htm. Accessed on 7/13/04.

Walker, ED. 1987. Efficacy of sustained-release formulations of *Bacillus thuringiensis* var *israelensis* and methoprene for the control of *Coquillettidia perturbans* in Indiana. *Journal of the American Mosquito Control Association* 3(1):97-99.

Walker, AN, P. Bush, T. Wilson, E. Chang, T. Miller, and MN Horst. 2005a. Metabolic effects of acute exposure to methoprene in the lobster, *Homarus americanus. Journal of Shellfish Research* 24(3):787-794.

Walker, AN, P. Bush, J. Puritz, T. Wilson, ES Chang, T. Miller, K. Holloway, and MN Horst. 2005b. Bioaccumulation and metabolic effects of the endocrine disruptor methoprene in the lobster, *Homarus americanus. Journal of Integrative and Comparative Biology* 45(1):118-126.

Ward, DH, RA Stehn, DV Derksen, CJ Lensink, and AK Loranger. 1986. *Behavior of Pacific Black Brant and Other Geese in Response to Aircraft Overflights and Other Disturbances at Izembek Lagoon, Alaska.* US Fish and Wildlife Service, Anchorage. AK. 34 pp.

Warren-Hicks, WJ, and DRJ Moore (Eds.). 1998. *Uncertainty Analysis in Ecological Risk Assessment*. SETAC, Pensacola, FL.

Webster, E., D. Mackay, and F. Wania. 1998. Evaluating environmental persistence. *Environmental Toxicology and Chemistry* 11:2148–2158.

Weinzierl, R., T. Henn, and PG Koehler. 1997. *Microbial Insecticides*. ENY-275. University of Florida, Institute of Food and Agricultural Sciences (UF/IFAS).

Wellmark International. 1998. *Altosid Liquid Larvicide Concentrate (specimen label)*. Bensenville, IL. 2 pp.

Westchester. 2001. *Comprehensive Mosquito-borne Disease Surveillance and Control Plan*. Draft Generic Environmental Impact Statement. Westchester County Board of Health, White Plains, NY. Available at: http://www.westchestergov.com/planning/environmental/stingEIS/STING_DGEIS.htm#FGEIS.

Weston, DP, RW Holmes, J. You, and MJ Lydy. 2005. Aquatic toxicity due to residential use of pyrethroid pesticides. *Environmental Science and Technology* 39:9778-9784.

White, CM, and SK Sherrod. 1973. Advantages and disadvantages of the use of rotor-winged aircraft in raptor surveys. *Raptor Research* 7(3/4):97-104

Wirth, MC, JA Ferrari, and GP Georgiou. 2001. Baseline susceptibility to bacterial insectivcides in populations of *Culex pipiens* complex (Diptera: Culicidae) from California and from the Mediterranean island of Cyprus. *Journal of Economic Entomology* 94(4):920-928.

Wiegert, RG, and BJ Freeman. 1990. *Tidal Salt Marshes of the Southeast Atlantic Coast: A Community Profile*. Biological Report 85 (7.29), US Fish and Wildlife Service, Washington, DC. 69 pp.

Wolfe, RJ. 1996. Effects of Open Marsh Water Management on selected tidal marsh resources: a review. *Journal of the American Mosquito Control Association* 12(4):701-712.

Wray, DL. 1950. *The Insects of North Carolina, Second Supplement*. North Carolina Department of Agriculture, Raleigh, NC. 59 pp.

Wray, DL. 1967. *The Insects of North Carolina, Third Supplement*. North Carolina Department of Agriculture, Raleigh, NC. 181 pp.

Wurtsbaugh, WA, and CS Apperson. 1978. Effects of mosquito control insecticides on nitrogen fixation and growth of blue-green algae in natural plankton associations. *Bulletin of Environmental Contamination and Toxicology* 19:641-647.

Yasuno, M. and K. Satake. 1990. The effects of diflubenzuron and methoprene on the emergence of insects and this density in an outdoor experimental stream. *Chemosphere* 21(10-11):1321-1335.

Yemaneberhan, H., Z. Bekele, A. Venn, S. Lewis, E. Parry, and J. Britton. 1997. Prevalence of wheeze and asthma and relation to atopy in urban and rural Ethiopia. *Lancet* 350:85-90.

Yousten, AA, FJ Genthner, and EF Benfield. 1992. ate of *Bacillus sphaericus* and *Bacillus thuringiensis serva israelensis* in the aquatic environment. *Journal of the American Mosquito Control Association* 8(2):143-148. As cited in Westchester, 2001.

Zhong, H. 1999. Naled impact on honeybees research. *1999 Public Health Entomology Research and Education Center Annual Report*. Florida A&M.

Zielinski-Gutierrez, EC. 2002. Barriers and facilitating factors to West Nile virus personal protection. Presentation, *Fourth National Conference on West Nile Virus in the United States*. New Orleans, LA. February 10.