health or the environment" (USEPA, 2003a).

Pesticides Industry Sales and Usage: 2000 and 2001 Market Estimates

USEPA prepares annual reports on pesticide production and sales market in the United States. The most recent USEPA Pesticides Industry Sales and Usage Report (covering the years 2000 and 2001) indicates that approximately $11 billion are spent on pesticides each year in the United States, which represents approximately 35 percent of total worldwide pesticide expenditures. This corresponds to over 1.2 billion pounds of conventional pesticides used annually in the United States. If wood preservatives, biocides and chlorine/hypochlorites are included, the total amount of pesticides used annually in the United States approaches five billion pounds. Agriculture accounts for approximately two thirds of total United States expenditures. Herbicides represent about 57 percent of the United States expenditure on pesticides, with 79 percent occurring in the agricultural sector. In residential settings, 60 percent of the expenditures for home and garden use were for insecticides (USEPA, 2004a).

In terms of specific pesticide active ingredients, glyphosate (e.g., Round-Up) was the most commonly used pesticide in agriculture for 2001 (85 to 90 million pounds), followed by atrazine (74 to 80 million pounds), metam sodium (57 to 62 million pounds), acetochlor (30 to 35 million pounds) and 2,4-D (28 to 33 million pounds). For home and garden uses, 2,4-D was the most commonly used pesticide in 2001, with eight to 11 million pounds used. This was followed by glyphosate (five to eight million pounds), pendimethalin (three to six million pounds), diazinon (four to six million pounds) and MCPP (four to six million pounds) (USEPA, 2004b).

New York State Pesticide Sales and Use Database

Under New York State law, NYSDEC is required to maintain a pesticide sales and use database, generated by usage reports submitted by commercial applicators and sales reports submitted by establishments that sell restricted use pesticides to agricultural applicators (private applicators). The database is maintained by Cornell Cooperative Extension and annual reports based on these data are prepared by NYSDEC and Cornell Cooperative Extension. The most recent annual report is for the year 2002.

The pesticide use data covers those pesticides used by commercial applicators (e.g., exterminators and lawn care applicators). For each application, the commercial applicator must provide the identity of the pesticide used, the quantity used and location of the application. However, the data contains the quantity of the pesticide product used and not the quantity of actual active ingredient. Pesticide products can contain a large percentage of inert ingredients and, especially for lawn care products, may contain other active ingredients such as fertilizers. For example, fertilizer with Merit Insecticide, used on lawns, only contains 0.2 percent of the active ingredient imidacloprid (The Andersons Lawn Fertilizer Division, Inc., 2002). The sales data reported does not necessarily represent use because it does not account for unused pesticides that are often returned by private applicators and also includes sales to other distributors. Therefore, these quantities most likely reflect an overestimate of actual use.

According to the most recent Pesticide Sales and Use Report, over two million gallons and over 16 million pounds of pesticides were used by commercial applicators in New York State during 2002. Approximately 800,000 gallons and five million pounds of pesticides were sold to private applicators for agricultural purposes in 2002. Again, it is important to recognize that these quantities do not represent the amounts of active ingredients, but instead the quantities of pesticide products. Table 3-21 lists the top three pesticides, in terms of volume and weight, for commercial and agricultural applicators in New York State.

Table 3-21. Top Three Pesticides Used in New York State 2002

	Product	**Active Ingredient, Percent Ingredient**
Commercial Applicator Use		
By weight	Lesco Pre-M Plus Fertilizer Insecticide	Pendimethalin, 1.31 percent
	Fertilizer With Merit Insecticide	Imidacloprid, 0.2 percent
	Merit 0.2 Insecticide plus Fertilizer	Imidacloprid, 0.2 percent
By volume	Sunnysol 150 Disinfectant	Sodium hypochlorite, 12.5 percent
	Surchlor Plus Disinfectant	Sodium hypochlorite, 12.5 percent
	Hypochlorite Solution Disinfectant	Sodium hypochlorite, 12.5 percent
Agricultural Applicator Sale		

By weight	Lorsban Insecticide	Chlorpyrifos, 15 percent
	Force 3G Insecticide	Tefluthrin, 3 percent
	Penncozeb 75 DF Fungicide	Zinc ion and manganese ethylenebisdithiocarbamate, 75 percent
	Prowl 3.3 EC Herbicide	Pendimethalin
By volume	Damoil Dormant and Summer Spray Oil/Miticide	Petroleum oil, 98 percent
	Roundup Ultramax Herbicide	Glyphosate, 50.5 percent

For Suffolk County, approximately 300,000 gallons and over three million pounds of pesticide products were applied by commercial applicators in 2002. There were four counties in New York State in which commercial applicators used more than one million pounds of pesticide products. These four counties were Suffolk (3.4 million pounds), Westchester (1.6 million pounds), Nassau (1.5 million pounds) and Monroe (1.2 million pounds). In terms of volume of pesticides used, Suffolk and Westchester counties had the greatest volume use (approximately 300,000 gallons in 2002).

For agricultural sales, 363,348 pounds and 50,460 gallons of pesticide products were sold in Suffolk County in 2002. For comparison purposes, 186,581 pounds and 23,202 gallons of agricultural pesticides were sold in rural Ulster County, and 128,813 pounds and 13,035 gallons of pesticides were sold in exurban Dutchess County.

Biomonitoring Studies

In 2003, CDC issued the Second National Report on Human Exposure to Environmental Chemicals (CDC, 2003f). This report updates information provided in its first report (issued in 2001), and summarizes data from biomonitoring in 1999 to 2000. These reports provide an ongoing survey of human exposure to environmental chemicals in the national population. Blood and urine samples of selected participants from the National Health and Nutrition Examination Survey (NHANES) are collected and analyzed for 116 chemicals that are found in the environment, including pesticides. Blood and urine analyses are considered indicative of exposure and are useful for comparisons between populations and determinations of trends over time. However, unless there is a known correlation between effects and blood or urine levels,

they do not directly provide an estimate of risk. The pesticides and/or their metabolites were reported as follows:

Organophosphates: Urinary metabolites of organophosphates were analyzed. Such analysis of metabolites is indicative of recent exposure (within a few days) to organophosphate pesticides. Generally the levels detected in this study were about the same or lower than those found in other studies. Ingestion of food and contact with organophosphates during residential applications are the primary source of exposure for the general population, as occupational exposures typically result in urinary levels that are 50 times higher than the urinary levels found. In almost all circumstances, concentrations of urinary metabolites were higher in the six to 11 year age group (the youngest group included). A urinary metabolite specific to malathion was analyzed for but rarely detected. In the six to 11 year age group the mean concentration of the metabolite for the population at the high end of exposure (95^{th} percentile) was 2.8 ug/L. Urinary concentrations and frequency of detection of a metabolite of chlorpyrifos were greater. This metabolite was detected in all age groups, with a geometric mean of 1.77 ug/L across all age groups.

Organochlorine Pesticides: Included were blood and urine analyses for the organochlorine pesticides lindane, DDT and metabolites, and chlordane. Many organochlorine pesticides have been banned due to their persistence. Lindane remains in limited use, primarily for treating human lice and scabies and limited agricultural applications. Organochlorine pesticides are lipid soluble. Therefore, they can be found in higher concentrations in fatty foods (e.g., milk and dairy products, breast milk, fish etc.), which continue to be a source of exposure. Since humans bioaccumulate organochlorine pesticides in fat tissue, they can be slowly released into the blood stream. Therefore, blood and urine levels reflect past as well as current exposures. More research is required in order to determine whether the levels reported represent a health concern. Lindane is the gamma isomer of hexachlorocyclohexane and the elimination of lindane from fatty tissue is much quicker than other isomers. For example, lindane has a half-life in the blood of 20 hours, whereas the beta isomer of hexachlorocyclohexane has a half-life of seven years. Lindane was only detected in the blood of 1.7 percent of the population sampled in this survey (detection limit of 7.5 ng/g lipid). The United States banned the use of DDT in 1973; however, DDT and to a greater extent its metabolite DDE can still be detected in human blood.

Continuing exposure primarily occurs from consumption of food. Because DDE is more persistent in the environment, concentrations in blood are higher than DDT levels. The geometric mean of DDE in the blood, at the 95th percentile of children aged 12 and older, was 260 ng/g of lipid. Blood levels in the sample population 20 years and older were higher than those in the 12 to 19 age group. The levels of DDT and DDE detected in this study were 15 and five times lower, respectively, than a similar study conducted between 1976 and 1980. Chlordane, an insecticide used to control termites but banned in 1988, was also analyzed for. Chlordane was primarily detected in individuals older than 20 years, with a concentration of 47.7 ng/g of lipid at the 95th percentile.

Carbamate Pesticides: Carbamate pesticides include carbaryl (Sevin), propoxur, and carbofuran. As with organophosphates, carbamates inhibit cholinesterase enzymes leading to impaired nerve signal transmission. However, the effects of carbamates are much shorter lived than organophosphates. The primary means of exposure to the general population is through the ingestion of food and the residential use of carbamates. Levels of metabolites that are detected in the urine reflect relatively recent exposures, unlike the organochlorine pesticides. A metabolite of carbaryl, 1-naphthol, was detected in urine samples; however, 1-naphthol is also a metabolite of other chemicals such as naphthalene. Therefore, its detections cannot be solely attributed to carbaryl exposure. 1-Naphthol was detected at a median concentration of 1.22 ug/L of urine. This level is about two to three times lower than previous NHANES monitoring, conducted from 1988 to 1994. A metabolite of propoxur, a pesticide that is used against a variety of insects in indoor, outdoor, and agricultural treatments, was detected infrequently (1.2 percent of the samples). A metabolite of carbofuran, as well as other carbamates, was detected a little more frequently, but only concentrations for the 95th percentile (0.740 ug/l in urine) were reported.

Herbicides: Herbicides that were monitored included alachlor, atrazine, 2,4-dichlorophenoxyacetic acid (2,4-D), and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T). 2,4,5-T is no longer used in the United States due to concern for contamination with 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Since most (90 percent) of the 2,4,5-T that enters the body is eliminated in the urine, its metabolite was not sampled for. Only 1.2 percent of the population sampled in this survey had detectable levels of 2,4,5-T. 2,4-D is also excreted in the urine

unchanged. It was also found infrequently, and only concentrations for the 95 percentile are reported. All other percentiles were below the detection limit of 0.09 ug/L. Atrazine is an herbicide that is used widely in agriculture especially on corn and sorghum crops. An atrazine metabolite was detected in 3.3 percent of the population sampled (detection limit of 0.047 ug/L). Alachlor, a herbicide that, with its breakdown products, has been detected in groundwater in Suffolk County, was analyzed for but detections above the detection limit of 0.09 ug/L in urine were not found.

Pest Repellents: Repellents that were analyzed for included DEET and naphthalene and *p*-dichlorobenzene (both used in mothballs). The metabolites of naphthalene, 1-naphthol and 2-naphthol, were detected with a median concentration of 1.22 ug/L and 0.370 ug/L, respectively. The levels of both of these metabolites were lower than the previous NHANES study (from 1988 to 1994). The metabolite of *p*-dichlorobenzene was found at higher concentrations in children aged six to 11 than in the rest of the population. Median concentrations for the population as a whole were 6.5 ug/L, but as *p*-dichlorobenzene is also extensively used as a bathroom deodorizer, the concentrations may not stem entirely from pesticide uses. DEET, in urine samples, was not reported above the detection limit of 0.06 ug/L. Urinary levels of DEET have been reported to be as high as 5,690 ug/L in workers who made daily applications of products that were 71 percent DEET. DEET use, generally, is seasonal. Sampling in this study did not specifically target the high use season, and so it is possible that it would be more frequently detected (and potentially at higher concentrations) then.

New York State Pesticide Poisoning Registry

NYSDOH founded the New York State Pesticide Poisoning Registry in 1990. Its purpose is to track poisonings, or acute health impacts, from exposure to pesticides. Reports of possible pesticide poisonings, required to be made by law, are received from hospitals, physicians, and clinical laboratories NYSDOH believes the number of reported incidents is likely to be an underestimate of the actual number of cases, since individuals and medical care personnel may not associate particular effects with pesticide exposure, or affected individuals may not seek medical care (NYSDOH, 1998).

In 1998, the registry reported and investigated 107 cases of pesticide poisonings. In 29 percent, sufficient information could not be obtained to determine the likelihood of whether pesticides were responsible for the effects. Many of these were cases of depressed cholinesterase activity in lawn care workers, reported through clinical laboratories.

The national Poison Control Centers Toxic Exposure Surveillance System ranks pesticides involved in reported poisonings as follows:

1. Anticoagulant rodenticides
2. Organophosphates
3. Pyrethrins and pyrethroids
4. Insect repellents
5. Carbamate insecticides
6. Organochlorine insecticides
7. Borates/boric acid
8. Chlorophenoxy herbicides
9. Naphthalene
10. Diquat herbicides

The 1998 New York State frequency list is as follows:

1. Cholinesterase-inhibiting insecticides (46 percent)
2. Insecticides, unspecified (38.1 percent)
3. Pesticides, unspecified (12.7 percent)
4. Herbicides (11.1 percent)

5. Repellents (6.3 percent)

6. Noncholinesterase-inhibiting insecticides, fungicides, and disinfectants (4.8 percent each)

9. Algicides (1.6 percent)

10. Rodenticides (0)

Of the reported non-occupational accidental cases, 92.8 percent occurred at a private residence. Only about two percent of the non-occupational cases were related to agricultural settings. Agricultural locations represented 19 percent of the occupational cases, as did institutional settings. Private residences only accounted for 9.5 percent of the occupational cases.

The most common symptoms reported were neuromuscular (68 percent), respiratory (55.6 percent), gastrointestinal (52.4 percent), and general symptoms of fatigue, fever, or general discomfort (36.5 percent) (multiple symptoms were recorded). Approximately 90 percent of the occupational and non-occupational cases combined occurred via inhalation. Dermal contact cases were 42.9 percent. Over 60 percent of the cases were indoor exposure to pesticides. 30 percent of the cases involved direct contact with the pesticide, as in spills or leaking containers. Pesticide spraying (17.5 percent) and drift (11.1 percent) accounted for over one-quarter of exposures. Contact with treated surfaces (carpets, plants, or pets) accounted for 9.5 percent of the cases.

20 incidents involving DEET were received from 1998 to 2005. Three involved deliberate ingestion or malicious spraying. The other 17 were for DEET used to repel mosquitoes and/or other insects, with ten involving children. The most common symptoms (excluding the three deliberate misapplications) were dermal and neurological. Other symptoms involved the eyes, gastrointestinal tract, or respiratory system, as well as general and cardiovascular effects. The percent of DEET in the products involved varied (W. Stone, NYSDOH, personal communication, 2005).

NYSDOH also receives pesticide incident reports -- those that have not been confirmed by medical personnel. 17 pesticide incident reports were received in 1998, all involving exposures

at private residences. 15 were for lawn care pesticides (nine from onsite use and six from wind drift from neighboring lawns).

WNV control efforts were included in 1999 to 2002 data sets. There were nine cases in 1999, 25 cases in 2000, three in 2001, and one in 2002. Almost all of the reports involved inhalation exposure and most occurred in the home. The most common symptoms were neurological, respiratory, gastrointestinal, and general symptoms, but only one case was considered to definitely involve pesticide exposure (the others were listed in the registry as "possible"). Self reports of health effects or concerns received by NYSDOH, local health departments, or Poison Control Centers, were also included. There were 260 in 1999, 215 in 2000, 24 in 2001, and 15 in 2002.

United States Department of Agriculture's Pesticide Data Program

Since 1991 the US Department of Agriculture (USDA) has been collecting pesticide residue data on food through the Pesticide Data Program (PDP). The primary purpose of this program is to monitor pesticide residues in washed, ready-to-eat agricultural commodities that are typically consumed by children and are available throughout most of the year. Under this program, the USDA has sampled approximately 50 different commodities ranging from fresh, frozen, or canned fruit and vegetables, fruit juices, whole milk, butter, grains, corn syrup, poultry, beef, and drinking water. These products were analyzed for more than 380 different pesticides. The samples of food tested are collected by various cooperating states, including New York.

In addition, over the course of this PDP program, 1,700 drinking water samples have been analyzed. In 51 percent of these drinking water samples, pesticides were detected, although typically in low part per trillion concentrations (Punzi et al., 2005).

Food and Drug Administration's Pesticide in Food Monitoring

The Food and Drug Administration (FDA) also monitors the food supply for pesticide residues. In the FDA surveillance monitoring, it collects samples close to the point of production and analyzes unwashed and unpeeled samples. This represents a worse case estimate of the amount of pesticides consumers may be exposed to, but allows FDA to enforce USEPA tolerance levels (the maximum amount of a pesticide that is allowed in a food commodity). In the Total Diet

Study program, FDA monitors food prepared for consumption. Foods are purchased at supermarkets or groceries in "market basket" surveys, prepared in a manner similar to that considered to be typical, and then analyzed for pesticide residues. These results are discussed later, under the Food Pathway Case Study.

Suffolk County Department of Health Services Pesticides in Groundwater Monitoring

SCDHS conducts groundwater monitoring for pesticides (partly under a contract with NYSDEC). Samples are analyzed by the Suffolk County Public and Environmental Health Laboratory (PEHL). Data from Suffolk County has been used by USEPA in pesticide Reregistration Eligibility Documents (REDs) (e.g., dacthal, alachlor). The sole source aquifer system, and soil conditions that can foster groundwater contamination make monitoring for pesticides and their degradates very important. Aquifer monitoring is especially pertinent in agricultural areas, where much drinking water comes from private wells (which are not routinely monitored).

More than 60 pesticide-related compounds have been detected in the pesticide groundwater monitoring studies conducted by SCDHS. This includes not only the parent pesticide compound, but also degradates. As of 2001, 30.5 percent of the tested wells contained pesticides. These data do not represent general groundwater conditions, as sampling was targeted to areas with a high likelihood of contamination. In general, detected concentrations have been one to two ppb or below. Figure 3-16 shows the location of wells tested through 2001, and Figure 3-17 shows wells where detections occurred. Wells with pesticide concentrations that exceed drinking water standards are shown in Figure 3-18. Agricultural areas on the North and South Forks appear to be most vulnerable to serious contamination problems.

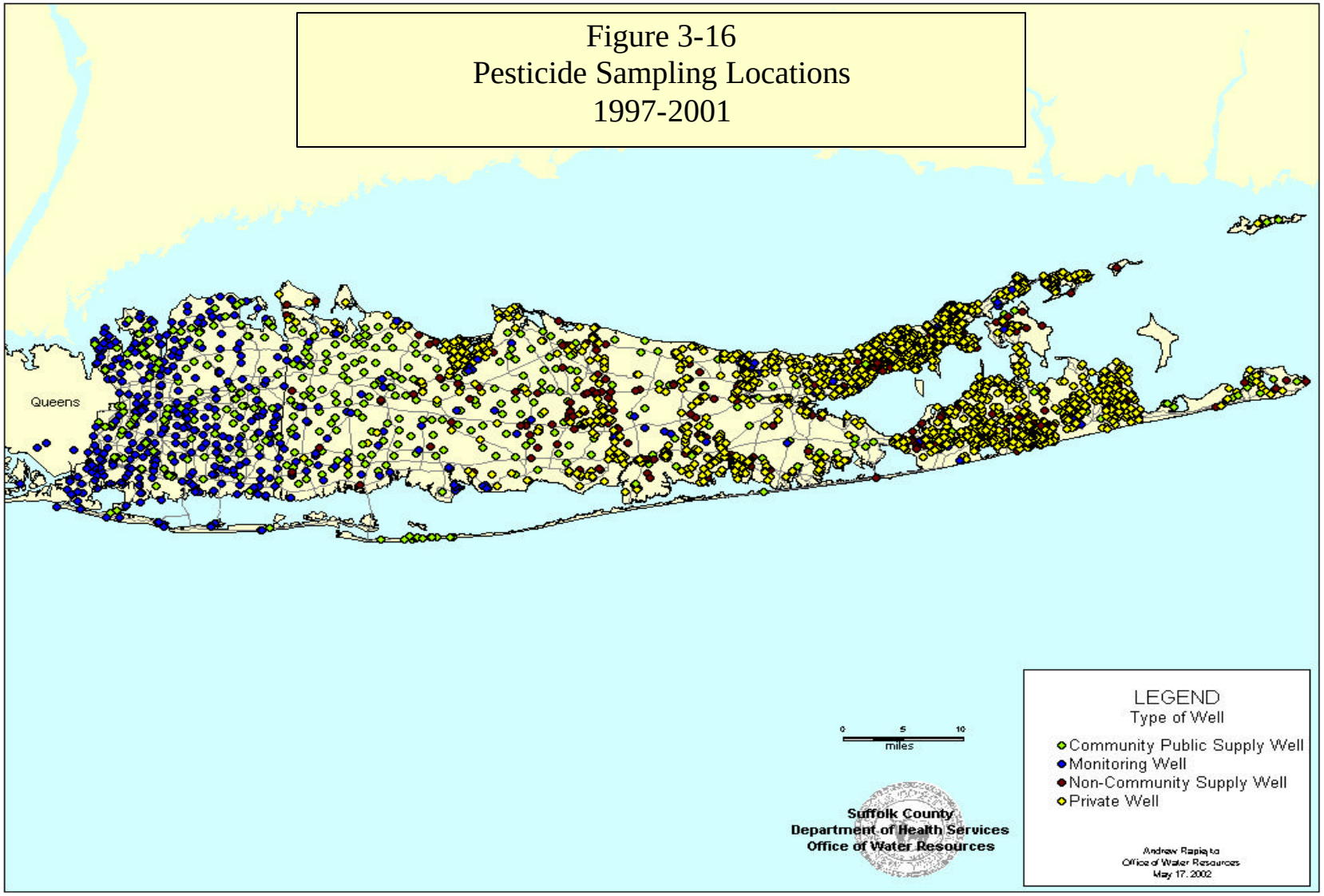


Figure 3-16
Pesticide Sampling Locations
1997-2001
Queens
0 5 10
miles
Suffolk County
Department of Health Services
Office of Water Resources
LEGEND
Type of Well
Community Public Supply Well
Monitoring Well
Non-Community Supply Well
Private Well
Andrew Rapiejko
Office of Water Resources
May 17, 2002

Figure 3-17
Sampling Locations with Pesticide Detections
1997-2001

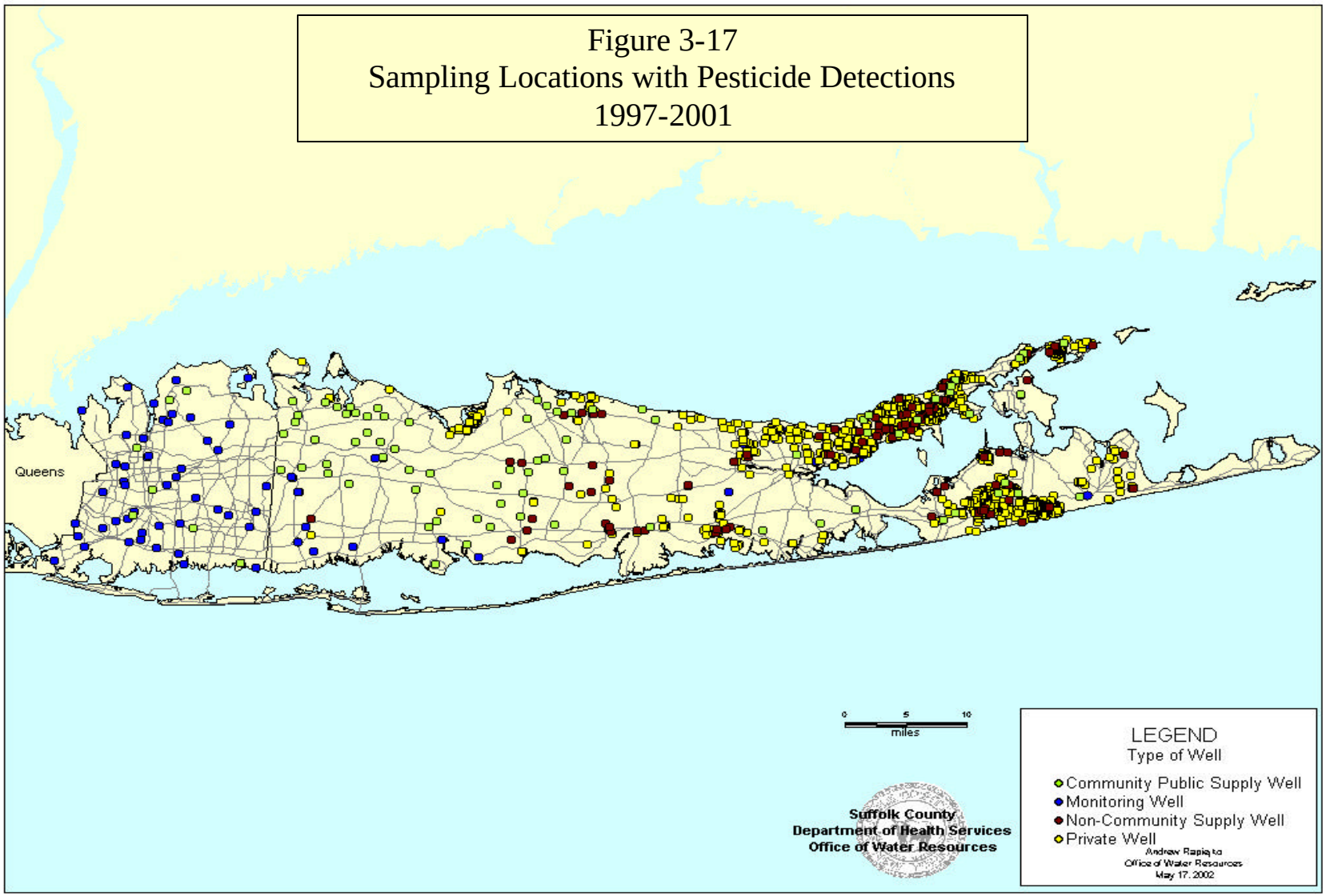
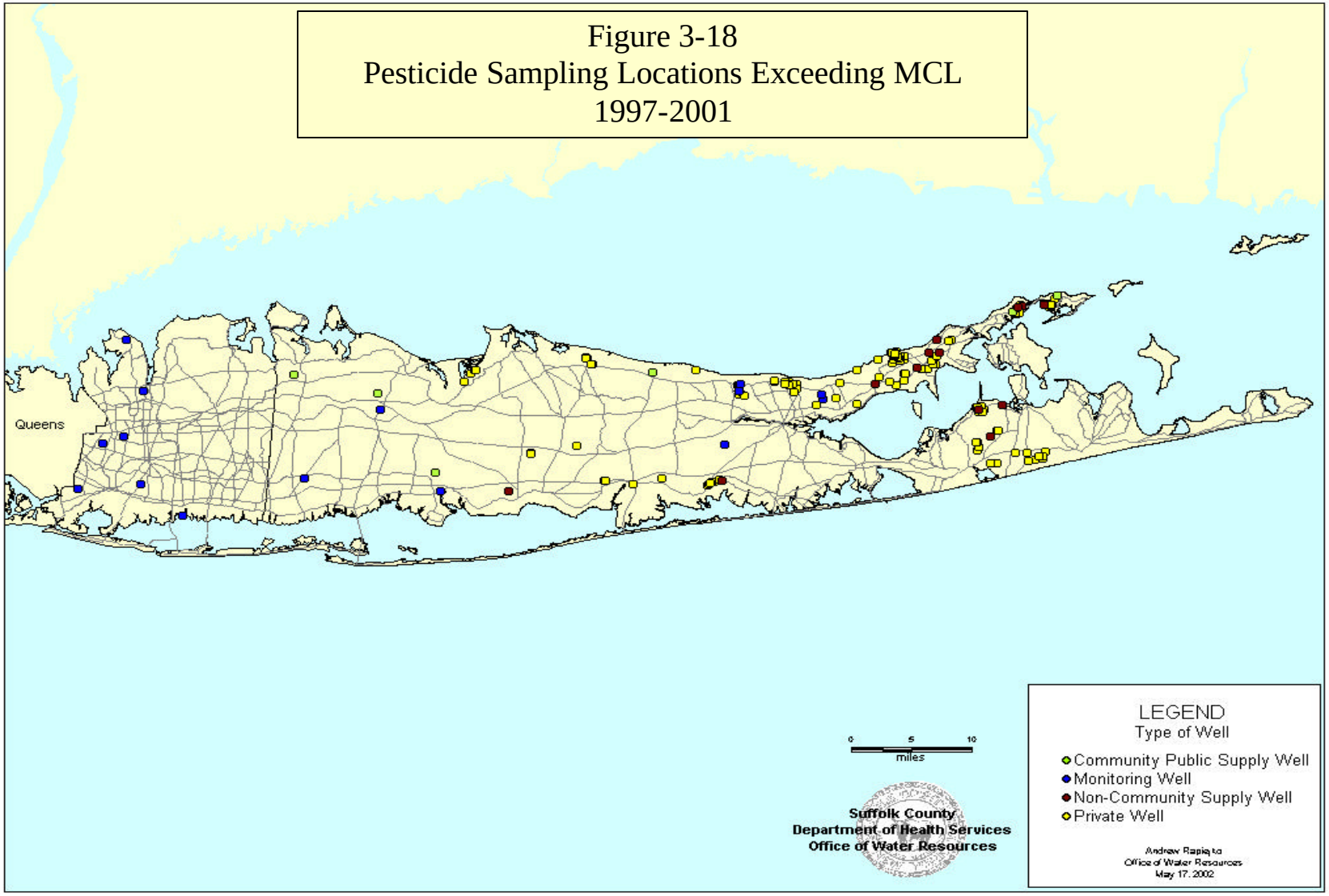


Figure 3-18
Pesticide Sampling Locations Exceeding MCL
1997-2001



13 pesticides have been found at concentrations above drinking water standards; seven percent of tested wells had concentrations above drinking water standards. The pesticides found above drinking water standards in the groundwater of Suffolk County are:

- alachlor
- aldicarb sulfoxide and sulfone (degradation products of aldicarb)
- chlordane
- 1,2-dichloropropane
- dinoseb
- EDB (ethylene dibromide)
- simazine
- tetrachloroterephthalic acid (TCPA, a breakdown product of dacthal)
- 1,2,3-trichloropropane
- metolachlor
- dichlorobromopropane
- cyanazine.

226 community supply wells had been sampled through 2001; 53 were found to contain pesticides, including four above drinking water standards. These four wells are being treated. 50 percent of the tested private wells were found to contain pesticides. 39 percent had multiple pesticide detections.

In summary, generally the levels of pesticide detections have been low. However, the data highlighted a few issues:

- the vulnerability of private water supplies, especially in agricultural areas
- the occurrence of multiple pesticide-related compounds in individual wells
- the lack of drinking water standards for some of the compounds detected
- the persistence of banned pesticide-related chemicals in groundwater.

The results of the groundwater monitoring studies in Suffolk County are consistent with the monitoring conducted by USGS nationwide. Its initial report (for 1992 to 1996 data) found approximately 48 percent of the groundwater sampling had detections of pesticides. Most of the detections were below one ppb, with a median concentration of 0.046 ppb. Sixty-seven pesticide analytes were detected, but multiple pesticide-related compounds were detected in 69 percent of the samples that contained pesticides. Not surprisingly, herbicides and fumigants were detected more frequently in agricultural settings than in urban areas, but there was no statistical difference in the detection of insecticides between these two areas (Kolpin et al., 2000). The update of this survey was recently released (Gilliom et al., 2006). Half of the shallow wells in agricultural and urban settings had detectable pesticides, and one-third of samples from aquifers in mixed land use settings also contained pesticides. Approximately one percent of the sampled drinking water wells (private or public) had concentrations of one or more pesticides that exceeded "human health benchmarks" (a combination of standards and published findings). Five pesticides were responsible for these exceedances:

- dieldrin
- dinoseb
- atrazine
- lindane
- diazinon

Approximately five percent of all samples in urban settings exceeded the human health benchmarks, which was much more than what was found in agricultural or mixed land use areas.

Other Aquatic Monitoring

Suffolk County aquatic data on pesticide occurrences were obtained from SCDHS water quality databases (lakes, ponds, and streams), from the USGS National Water Quality Assessment (NAWQA) database, the USEPA Storage and Retrieval (STORET) database, Brookhaven National Laboratory (BNL) and SCDHS studies of the Peconic River, and National Oceanographic and Atmospheric Administration (NOAA) studies of mollusks. The SCDHS surface water data (Table 3-22) are the most abundant, and probably the most useful; they indicate generally low levels of contamination by water-soluble pesticides such as aldicarb, alachlor, metolachlor, and DEET, as well as an occasional detection of lipid soluble organochlorines such as DDT. The NAWQA data on surface waters (Table 3-23), which were collected as part of a nationwide characterization effort by the USGS, are limited in number (nine samples), and the extremely low USGS detection limits (parts per trillion) and detected levels (all less than one ppb) make interpretation of their significance difficult. Similarly, the USEPA's STORET data on surface waters (Table 3-24) are old (1983), limited in number, and based on very low detection levels, but are included for completeness. Sediment data from BNL (Table 3-25), STORET (Table 3-26), NAWQA (Table 3-27), and SCDHS (Table 3-28), as well as fish data from BNL (Table 3-29) and STORET (Table 3-30), and mollusk data from NOAA (Table 3-31) and SCDHS (Table 3-32), are limited to the lipid soluble (non-polar) organochlorine pesticides, which would be expected to accumulate in organic-rich sediments and living tissues; they do not address, however, the water soluble pesticides in common use today, although these would not be expected to accumulate.

Table 3-22. Surface Water Pesticides Detections

Source: Suffolk County Department of Health Services Data 1999 - 2005 *Concentrations in ug/l*						
Pesticide	**No. Detections**	**Mean Concentration of Detections**	**Minimum Concentration**	**Maximum Concentration**	**Detection Limit**	**CWQG***
1,2-Dichloropropane	4	0.58	0.5	0.7	0.5	
4,4 DDD	3	0.32	0.23	0.44	0.2	
4,4 DDT	1	0.29	0.29	0.29	0.2	
Alachlor ESA	99	1.28	0.2	6.98	0.2	
Alachlor OA	43	0.95	0.4	2.61	0.4	
Aldicarb Sulfone	109	1.07	0.5	5.1	0.5	10**
Aldicarb Sulfoxide	98	1.02	0.5	3.9	0.5	10**
Azoxystrobin	2	3.47	2.34	4.6	0.2	
CARBARYL	1	0.89	0.89	0.89	0.5	
Chlorothalonil	2	8.20	5.39	11	1	
Diazinon	1	0.24	0.24	0.24	0.2	
Dichlobenil	6	0.43	0.2	0.9	0.2	
Dichlorvos	1	1.96	1.96	1.96	0.5	
Diethyltoluamide (DEET)	33	2.20	0.22	33.4	0.2	
Dinoseb	11	1.43	0.25	6.39	0.5	0.05
Endosulfan sulfate	2	0.42	0.31	0.52	0.2	
Ethofumesate	2	0.39	0.2	0.57	0.2	
Imidacloprid	4	0.24	0.2	0.27	0.2	
Metalaxyl	51	0.44	0.2	1.89	0.2	
Methoprene	3	0.60	0.46	0.73	0.2	
Metolachlor	29	0.45	0.2	1	0.2	7.8
Metolachlor ESA	240	1.51	0.3	4.68	0.3	
Metolachlor OA	187	1.24	0.3	3.64	0.3	
Metribuzin	3	1.82	0.22	2.86	0.2	
Perchlorate	1	2.10	2.10	2.10	2	
Prometon	1	1.00	1	1	0.5	
Ronstar	4	0.90	0.8	1	0.2	
Simazine	7	1.04	0.21	2.55	0.2	10
Terbacil	3	0.57	0.5	0.6	0.5	
Tetrachloroterephthalic acid	14	10.97	5	37	5	
Vinclozolin	1	7.35	7.35	7.35	0.5	

* Canadian Water Quality Standards

** parent Aldicarb

Table 3-23. Surface Water Pesticides Detections , continued

Source: United States Geological Survey - 1994 - 2003 *Concentrations in ug/l*				
Pesticide	**No. Detections**	**Mean of Detections**	**Minimum Concentration**	**Maximum Concentration**
Alachlor	1	0.0046	0.0046	0.0046
Atrazine	7	0.0048	0.0022	0.0094
CIAT	3	0.0013	0.0007	0.0021
Carbaryl	5	0.0350	0.0055	0.1010
DCPA	1	0.0014	0.0014	0.0014
Diazinon	3	0.0069	0.0037	0.0118
Dieldrin	3	0.0155	0.0135	0.0186
Malathion	1	0.0118	0.0118	0.0118
Metolachlor	4	0.0038	0.0027	0.0051
Prometon	7	0.0132	0.0036	0.0236
Simazine	7	0.0083	0.0021	0.0320
Tebuthiuron	2	0.0826	0.0043	0.1610
Trifluralin	2	0.0019	0.0018	0.0021

Table 3-24. Surface Water Pesticides Detections, continued

Surface Waters - Suffolk County, New York				
Source: United States Environmental Protection Agency STORET Database - 1983 *Concentrations in ug/l*				
Pesticide	**No. Detections**	**Mean of Detections**	**Minimum Concentration**	**Maximum Concentration**
DDD	11	0.05	0.004	0.13
DDE	6	0.01	0.002	0.021

Table 3-25. Sediment Pesticide Detections

Source: Brookhaven National Laboratory - Peconic River – 1995 – 2001				
Concentrations in ug/kg				
Pesticide	**No. Detections**	**Mean Concentration of Detections**	**Minimum Concentration**	**Maximum Concentration**
4,4''-DDD	18	8.1	0.51	43.2
4,4'-DDD	7	38.6	1.72	96
4,4-DDD	49	17.1	0.31	657
4,4''-DDE	54	7.23	0.31	157
4,4'-DDE	20	32.4	5.76	89
4,4''-DDT	15	901	1.5	13,400
4,4'-DDT	3	24	4.69	50.1
alpha-Chlordane	10	29.5	1.75	73
Chlordane-Technical	3	67.9	16.2	158
DDD	1	99	99	99
DDD,PP'	1	89	89	89
DDE	1	33	33	33
DDE,PP'	1	38.7	38.7	38.7
DDT,PP'	1	59.6	59.6	59.6
Endosulfan I	1	18	18	18
Endosulfan B	1	3.03	3.03	3.03
gamma-Chlordane	17	18	2.32	43
Heptachlor epoxide	3	2.83	1.6	4.5
Methoxychlor	1	398	398	398

Table 3-26. Sediment Pesticides Detections, continued

Sediments - Suffolk County, New York				
Source: United States Environmental Protection Agency STORET Database - 1983 - 1993				
Concentrations in ug/kg				
Pesticide	**No. Detections**	**Mean Concentration of Detections**	**Minimum Concentration**	**Maximum Concentration**
cis-Chlordane	2	0.32	0.27	0.36
DDD	41	7,601	0.70	85,740
DDE	44	1,412	0.66	26,000
DDE, o,p'	2	0.42	0.32	0.51
DDT	25	4,855	110	38,100
DDT-o,p'	16	1176	10	12,250
Endrin	2	0.66	0.42	0.90
Endrin ketone	1	1.13	1.13	1.13
gamma-BHC (Lindane)	1	0.74	0.74	0.74
Mirex	1	0.28	0.28	0.28

Table 3-27. Sediment Pesticides Detections, continued

Source: United States Geological Survey NAWQA Data - 1994 - 2003

Concentrations in ug/kg

Pesticide	No. Detections	Mean Concentration of Detections	Minimum Concentration	Maximum Concentration
Dieldrin	6	4.82	1.1	14
Hexachlorobenzene	1	1.60	1.6	1.6
Oxychlordane	1	1.30	1.3	1.3
alphaEndosulfan	1	2.90	2.9	2.9
cisChlordane	8	4.38	1.1	17
cisNonachlor	2	3.50	1.2	5.8
op'-DDD	2	3.20	1.1	5.3
pp'-DDD	14	6.78	1.1	24
pp'-DDE	26	5.75	1	23
pp'-DDT	15	7.71	2.3	19
tr-Chlordane	8	4.44	1	20
tr-Nonachlor	10	3.79	1.1	14

Table 3-28. Sediment Pesticides Detections, continued

Source: Suffolk County Department of Health Services[1]- 1996 *Concentrations in ug/kg*				
Pesticide	**No. Detections**	**Mean Concentration of Detections**	**Minimum Concentration**	**Maximum Concentration**
Alpha-BHC	4	0.42	0.24	0.73
HCB	3	0.23	0.1	0.48
Beta-BHC	1	0.76	0.76	0.76
Gamma-BHC	3	0.42	0.36	0.47
Delta-BHC	3	3.49	0.98	7.9
Gamma-Chlordane	6	1.45	0.30	2.9
2,4'-DDE	3	1.17	0.52	2.1
Alpha-Chlordane	7	1.34	0.13	3
trans-NONACH	6	1.71	0.47	3.2
Dieldrin	7	1.29	0.23	5
4,4'-DDE	11	12.03	0.77	53
2,4'-DDD	8	3.72	0.19	10
4,4'-DDD	11	11.79	0.59	52
2,4'-DDT	3	3.20	1.4	4.8
Endosulfan	5	3.28	0.11	13
4,4'-DDT	7	8.08	0.54	27
Endrin Ketone	4	0.43	0.14	0.97
Methoxychlor	1	0.24	0.24	0.24
Mirex	10	0.64	0.21	2.1

Table 3-29. Biota Pesticides Detections

Source: Brookhaven National Laboratory - Peconic River 1996-1997 *Concentrations in ug/kg wet weight*				
Pesticide	**No. Detections**	**Mean Concentration of Detections**	**Minimum Concentration**	**Maximum Concentration**
Alpha-Chlordane	14	13	5.3	32.5
DDD	20	21	2	130
DDE	108	35	5	136
DDT	11	4.6	2	22.6

[1] Chemical Contaminant Distributions in Peconic Estuary Sediments, Arthur D. Little, December 13, 1996.

Table 3-30. Biota Pesticides Detections, continued.

Source: United States Environmental Protection Agency STORET Database - 1978/1987 *Concentrations in ug/kg wet weight*				
Pesticide	**No. Detections**	**Mean Concentration of Detections**	**Minimum Concentration**	**Maximum Concentration**
Chlordane	2	0.01	0.01	0.01
DDT	16	0.13	0.01	0.66
Dieldrin	5	0.01	0.01	0.01
P,P'-DDE	2	0.03	0.03	0.04
Toxaphene	8	0.05	0.01	0.08

Table 3-31. Biota Pesticides Detections, continued

Source: National Oceanic and Atmospheric Administration [2] ***- 1986 - 1988*** **Concentrations in ug/kg dry weight**		
1.1 Location	**Pesticide**	**Mean Concentration**
Huntington Harbor	DDT	96
	Total Chlordane	45
	Dieldrin	26
	Lindane	3.3
	Mirex	0.45
Port Jefferson	DDT	60
	Total Chlordane	22
	Dieldrin	11
	Lindane	2.6
	Mirex	1.3
Moriches Bay	DDT	47
	Total Chlordane	11
	Dieldrin	4.6
	Lindane	1.2
	Mirex	5.9

Table 3-32. Biota Pesticides Detections, continued

Source: Suffolk County Department of Health Services Peconic River - 2000 *Concentrations in ug/kg wet weight*				
Pesticide	No. Detections	Mean Concentration of Detections	Minimum Concentration	Maximum Concentration
p, p'-DDE	12	3.58	2	5

[2] NOAA Technical memorandum NOS OMA 49, August 1989

3.5 Baseline Impacts from Pesticides Exposures

3.5.1 Human Health Risks

There are many factors that determine whether a pesticide will pose a health risk. Pesticides may differ in the type of health problem they may cause (i.e., neurotoxicity or cancer), and in their potency (i.e., how much of a given pesticide will cause an adverse effect). Potential risk will also depend upon how exposure occurs, which is dependent on the manner in which the pesticide is used; whether it degrades in the environment; and its ability to partition to soil, water or air. It is also important to consider the toxicity of any degradation products. Some pesticides no longer in use remain in the environment (e.g., DDT and breakdown products), which leads to continued exposure.

In order to conduct a complete evaluation of background pesticide risks, all pesticides and each of their uses and potential exposures would need to be evaluated. Such an effort is not feasible. Therefore, a few examples of pesticide uses and associated risks were selected for further characterization. Because this is not an exhaustive evaluation of background pesticide risks, it is not appropriate to draw broad conclusions from the information here. Rather, this information has been presented to provide a characterization and perspective of some background risks from pesticides that are typically encountered by the general public.

Several case studies using different pathways of exposure were examined. The pathways selected included food, drinking water, indoor residential treatments, residential lawn applications, lice and scabies treatment, and domestic animal risks. It is acknowledged that these case studies are not exhaustive reviews of risks associated with these pathways, but a characterization of potential risks. These case studies attempt to be quantitative, when possible.

Food Exposure Pathway

Pesticide residues in food are regulated by three federal agencies – FDA, USDA, and USEPA. USEPA registers pesticides and regulates their use. USEPA also sets tolerance levels, which represent the maximum amount of a pesticide that is allowed in a food commodity. USEPA must set these tolerances at a level that is not expected to cause harm or adverse health effects.

FDA is responsible for enforcing those tolerance levels in foods shipped in interstate commerce. FDA also conducts market basket surveys to monitor exposure to pesticide residues in food as typically consumed. USDA is responsible for monitoring and regulating pesticide residues in meat, poultry, and certain egg products. In addition, USDA conducts a PDP, in which pesticide residues are monitored in washed, ready-to-eat agricultural commodities, typically consumed by children.

The most recent data available from the USDA PDP is from samples collected in 2003. Over 12,000 samples were analyzed. Residues of pesticides were detected in 56 percent of these samples; 0.3 percent had residues in excess of established tolerances. In addition, more than one pesticide was detected in 23 percent of the most recent samples (Punzi et al., 2005). On a commodity basis, 99 percent of the butter samples, 45 percent of the wheat flour samples, 43 percent of the fruit and vegetable samples, and eight percent of the barley samples had detectable residues (USDA, 2005a). Most of the residues found in butter were low levels of endosulfan sulfates, dieldrin, and DDE. In most cases, pesticide residues were higher in fresh produce than processed. For example, the fungicide thiabendazole was found in 72 percent of the fresh apple samples, but only 24 percent of the apple sauce samples. Interestingly, DDE, a metabolite of DDT, is still being detected in some foods, even though DDT has not been used in the United States since 1972. Pesticide residues have not been detected in beef muscle or liver, although DDE has been detected in beef adipose tissue (fat) (up to one ppm). Chicken meat and liver also rarely have detections of pesticides, although DDE and dieldrin have been detected in fat tissue at up to five ppb (Punzi et al., 2005).

Based on sampling conducted by USDA and FDA from 1992 to 2001, the Environmental Working Group (EWG) published a list of the produce that consistently had detectable residues of pesticides and those that did not. The "most contaminated" list included apples, bell peppers, celery, cherries, imported grapes, nectarines, peaches, pears, potatoes, red raspberries, spinach, and strawberries. Among the least contaminated were asparagus, avocados, bananas, broccoli, cauliflower, sweet corn, kiwi, mangoes, onions, papaya, pineapples, and sweet peas (EWG, undated).

The studies described above provide estimates of the amount and occurrence of pesticides in food, but they do not estimate risk from consumption. A study (MacIntosh et al., 2001) measured the amount of various pesticides present in the food consumed by study participants over four day intervals several times between September 1995 and September 1996. Participants were older than 10 years of age. Duplicate plates were prepared by participants, one that was consumed and the other composited for pesticide analysis. Malathion was found in 75 percent of the samples, chlorpyrifos in 38 percent, and DDE in 21 percent. Malathion was found at a mean concentration of 1.9 ug/kg, chlorpyrifos at 0.8 ug/kg, and DDE at 0.2 ug/kg in the prepared food. This resulted in a mean exposure of 0.018 ug/kgbw for malathion, 0.007 ug/kgbw for chlorpyrifos, and 0.002 ug/kgbw per day for DDE (Table 3-33). USEPA has established a chronic oral reference doses for malathion (20 ug/kgbw per day) and chlorpyrifos (three ug/kgbw per day) (USEPA, 1992; USEPA 1988a); the reference doses represent daily exposures predicted to not pose a risk of adverse noncarcinogenic effects. The mean daily exposures calculated above are orders of magnitude lower than the USEPA reference doses for malathion and chlorpyrifos. Although a reference dose for DDE was not available, DDE (as well as the parent pesticide DDT) is considered a probable human carcinogen, based on animal studies. USEPA has established a cancer potency factor for DDE (0.34 mg/kgbw per day) (USEPA, 1988b). Assuming a lifetime daily exposure to the mean amount of DDE in food from this study, the corresponding cancer risk would be 7×10^{-7} (an estimated seven excess cancers in 10 million people exposed over a lifetime). Typically, cancer risks below 1×10^{-6} are considered negligible.

Table 3-33. Mean Exposure and Risk (MacIntosh et al., 2001)

Pesticide	**Mean Exposure** (mg/kgbw per day)	**Oral Reference Dose** (mg/kgbw per day)	**Cancer Risk at Mean Concentration**
Malathion	1.8×10^{-5}	2×10^{-2}	NA
Chlorpyrifos	6.8×10^{-6}	3×10^{-3}	NA
DDE	2.0×10^{-6}	NA	7×10^{-7}

Results from FDA surveillance monitoring of 2,122 domestic food samples collected in 2002 indicate that no pesticide residues were detected in 65.5 percent of the samples; 0.8 percent had pesticides at a level above regulatory levels. 70.4 percent of imported foods (4,644 samples analyzed) had no detectable residues. Approximately four percent had residues above regulatory limits. Milk and eggs (all domestic sources) did not have any detectable residues. Although

approximately 17 percent of cheese products did have detectable residues, none were above tolerance levels. Eighty-one percent of the apple juice samples contained no residues, and none were above tolerance levels. Similarly, 93 percent of baby food and formula samples did not have detectable levels of pesticides and none had levels above regulatory limits. Fruits and vegetables had the largest percentage of samples with pesticides detected (49.8 percent); 0.7 percent had residues above the tolerance limit (FDA, 2004).

Results of the Total Diet Study from samples collected in 2002 indicate that the five most frequently detected pesticides are DDT, chlorpyrifos-methyl, malathion, endosulfan, and dieldrin. The pesticide detected at the highest concentration was carbaryl (2.04 ppm), followed by permethrin (1.68 ppm); these concentrations are below regulatory limits. The highest concentration of malathion detected was 0.071 ppm. The five most frequently detected pesticides in baby food were carbaryl, chlorpyrifos-methyl, malathion, permethrin, and thiabendazole. The highest detection of malathion was 0.026 ppm, and the highest detection of permethrin was 0.013 ppm (FDA, 2004).

In the organophosphate cumulative risk assessments, USEPA did not conduct a regional analysis for the food pathway, as they did for other exposure pathways. Instead, it was assumed that intake and residue levels were not different between the various regions that were evaluated. To evaluate exposure to organophosphate residues in food, USEPA used primarily the USDA PDP (discussed previously), but supplemented these data with FDA Total Diet Study and Monitoring data to estimate residues in meat, seafood, and eggs. Eggs and seafood were found to contain negligible amounts of organophosphate. These residue data were then combined with consumption data taken from the USDA's Continuing Survey of Food Intakes by Individuals. This consumption data is derived from surveys of the general population recording food intakes over a three-day period.

The results of the food exposure pathway analysis show that the highest exposed population was children aged one to two years old, followed by children aged three to five. Adult age groups were broken down into ages 20 to 49 years of age and greater than 50 years. Exposures were similar in each adult age category. The results of the regional analysis that includes New York indicate that the margins of exposure (MOEs) at the 95th percentile of exposure for both

childhood age categories were just below 500. This means at the 95 percentile exposure to organophosphates in food was about 500 times less than the level of concern. At the 99th percentile, the MOE was 160 for children aged one to two years and 196 for children three to five years of age. The most significant food crops contributing to risk in the high end of the exposure percentile were apples, grapes, green peppers, pears, potatoes, spinach, succulent beans, and tomatoes. Food, compared to the other pathways of exposure evaluated in the cumulative risk assessment, represented the single greatest contribution to risk (USEPA, 2002a).

Drinking Water Exposure Pathway

In the organophosphate cumulative risk analysis, USEPA used modeling to predict potential exposures within vulnerable watersheds in its regional analyses. When available, analytical results from actual monitoring were used to validate the modeling results. In this exposure pathway analysis, it was assumed that all drinking water was obtained from surface water supplies. For areas such as Long Island, this would overestimate risk, since groundwater generally has lower levels of organophosphates. Even with this conservative approach, drinking water was not an important contributor to overall risk in all the regional cumulative assessments that were conducted. As reported in the revised assessment, results of the modeling for the Northeast/North Central region provided an estimated mean concentration of methamidophos-equivalents (see the earlier discussion of USEPA's Cumulative Risk Assessment for Organophosphorus Compounds) in drinking water of 0.09 ppb, with a maximum of five ppb. Risks associated with drinking water were one to two orders of magnitude lower than the risks associated with food exposures. The MOEs associated with drinking water were typically between 1,000 and 10,000 in the regional analysis that included New York and Long Island.

Overall there appears to be a small relative risk from the exposure to pesticides in drinking water, compared to other pathways of exposure. However, individual wells that are contaminated (especially those with levels above drinking water standards) represent a more substantial relative risk to individuals drinking that water than some other pathways they are exposed to.

Indoor Residential Treatments

A survey conducted in 1992 estimated that 78 million households in the United States use pesticides (approximately 74 percent of the estimated 105.5 million households in the US) (USEPA, 2004b). Approximately 59 million households use insecticides, 53 million use repellents, and 41 million use herbicides.

Residential pesticide treatments, including indoor use, were evaluated in the USEPA organophosphate cumulative risk assessment. The scenarios developed for this pathway included lawn care and golf course applications, home gardens, wide area public health sprays, indoor crack and crevice sprays, and impregnated pest strips. In previous evaluations, USEPA estimated that uses such as ant baits and paint additives resulted in very low exposures and, therefore, these were not included (USEPA, 2002a).

For the region encompassing New York and Long Island, the residential exposure pathway was not a significant source of risk in the populations in the 95th percentile of exposure. For those populations in the 97.5th percentile, residential exposure became the predominant exposure pathway for certain times of the year. This was due to the indoor use of dichlorvos in No-Pest strips and crack and crevice treatments. This finding was consistent in all regions evaluated. Exposure from the use of these products was via inhalation.

Residential Lawn Applications

Human exposures to pesticides applied to residential lawns and landscaping (as insecticides, acaricides, miticides, herbicides, and fungicides) add to the overall risk levels experienced by the general population. Direct outdoor dermal and inhalation exposure routes exist for adults and children following homeowner and professional applications of these pesticides, and these are routinely evaluated during the pesticide registration and re-registration processes. For example, the USEPA Interim Re-registration Eligibility Decision (IRED) for the organophosphate pesticide chlorpyrifos indicated that direct dermal and inhalation exposures needed to be reduced by eliminating virtually all homeowner uses in 2000; direct ingestion of soil and the mouthing of grass by children, however, were not considered significant exposure routes (USEPA, 2001a;

USEPA, 2002b). Similarly, the IRED for the organophosphate insecticide diazinon cites the 2000 phase out and cancellation of all residential uses, as necessary, to reduce risks resulting from direct exposure (USEPA, 2004c).

The registration and re-registration processes also take into account indoor exposures to these pesticides, which are potentially more significant than outdoor exposures (Lewis, 2005). Indoor exposures to pesticides applied to lawns and landscaping occur when these chemicals are resuspended by winds and are carried into houses through open windows or doors, or through cracks and crevices, or are tracked into houses on shoes, clothing, and pets (Nishioka, 2001). Once inside, they can volatilize into the air, attach to carpeting and furniture, and accumulate in house dust. Typical pesticide concentrations in house dust can be 10 to 100 times higher than those found in surface soil (Simcox et al., 1995; Wilson et al. 2003; Lewis, 2005). Volatilized pesticides and resuspended house dust can be inhaled by inhabitants of all ages.

Infants and toddlers represent a particularly vulnerable population in terms of lawn and landscaping pesticide exposure, since they can have significant direct dermal contact with soils and dust, and may frequently engage in mouthing activities (involving contaminated hands, toys, furniture, etc.). USEPA assumes that the average soil and dust ingestion rate for small children is 100 mg per day (USEPA, 1996).

When tracked indoors, pesticides can persist for months or years, since they are not exposed to rain, sunlight, or significant microbial degradation. This is especially true for non-volatile pesticides, such as the older organochlorines like DDT, dieldrin, pentachlorophenol, and chlordane (Lewis et al., 1994; Nishioka et al., 1996; Morgan et al., 2001). Many of the herbicides used on lawns (such as the post emergent herbicides 2,4-D and glyphosate) are applied in the form of amine salts that have low vapor pressures; their presence in indoor air are generally low, and if present in atmospheric samples they are found mostly in suspended particulate matter. But even pesticides that are considered nonvolatile can sometimes be found in indoor air; examples are the triazine herbicides used for pre-emergent weed control. Chlorpyrifos (e.g., Dursban), which is used for lawn grub control and is classified as slightly volatile, has been found as a common indoor air contaminant; however, this may have been due

to its use in termiticides, indoor flea foggers, and various pet products prior to the almost total phase out in 2000 (Lewis, 2005).

New York State's Pesticide Sales and Use Reporting Database indicates that a very large percentage (86 percent in 2000) of dry pesticide/fertilizer formulations used by commercial applicators in Suffolk County were used for lawn care and landscaping (NYSDOH, 2004). The database also indicates that liquid pesticides used in residential applications during 2000 include the insecticide carbaryl (Sevin) and the termiticide permethrin (Dragnet).

NYSDOH evaluated the possible contribution of the chlorophenoxy acid lawn herbicide 2,4-D, which has been in use for over 50 years, to cancer rates in northwestern Brookhaven Town. The soil half-life of 2,4-D is typically less than seven days, but a number of studies have demonstrated that it is readily tracked indoors after lawn applications, and can persist indoors for up to one year (Nishioka et al. 1996; Nishioka et al. 2001; Wilson et al., 2003). NYSDOH concluded that 2,4-D is unlikely to be a risk factor in human breast cancer, and that indoor exposures following outdoor applications are unlikely to cause non-cancer health risks (NYSDOH, 2004). NYSDOH will be further evaluating additional pesticides including mecoprop (a phenoxy herbicide), dicamba (a benzoic acid herbicide), and carbaryl (a carbamate insecticide), as well as termiticides, horticultural oils made from petroleum, and the soil fumigant Vapam in its final report.

In one study of indoor exposures to 2,4-D resulting soon after lawn applications (Nishioka, 2001), the estimated ingestion rate for young children was one to 10 µg/day from contact with floors and 0.2 to 30 µg/day from contact with table tops, which were about 10 times the pre-application exposure rates and considerably higher than the estimated dietary intake rate of 1.3 µg/day. Nonetheless, these concentrations are considerably lower than the WHO's acceptable daily intake rate of 300 µg/kg/day, or USEPA's reference dose (RfD) of 10 µg/kg/day (which is equivalent to 100 µg/day for a 10-kg child) (Nishioka et al., 2001).

In another study conducted by USEPA during 1985 to 1990 (the Non-Occupational Pesticides Exposure Study) in residences in two cities (Jacksonville, Florida and Springfield

Massachusetts), the estimated lifetime excess cancer risks for 2,4-D were 6×10^{-9} and 2×10^{-7}, respectively (Whitmore et al., 1994).

There are five organophosphate pesticides registered for use on residential lawns, three of which can be applied by homeowners as well as professional applicators. These three are bensulide (to control germinating weeds), trichlorfon (for grubs), and malathion (for fleas and other nuisance pests) (USEPA, 2001a). The use of the two others – the insecticides diazinon and chlorpyrifos – was severely limited by USEPA in 2000 (Lewis, 2005). Acephate, disulfoton, and malathion are used in ornamental gardens, and malathion is also used in vegetable gardens and orchards (USEPA, 2001a).

In one study of organophosphate pesticides, Lewis et al. (2001) estimated that indoor air exposures following outdoor (house perimeter) applications of chlorpyrifos were only on the order of 0.05 µg/kg/day. Ingestion of carpet dust added only about 0.01 µg/kg/day, while residues found on children's hands suggested that repeated mouthing could contribute as much as one to 1.5 µg/kg/day. These are still lower than the USEPA reference dose of three µg/kg/day for chronic or subchronic oral exposure to chlorpyrifos.

The human health risks associated with pyrethrin pesticides, which are extracted from the chrysanthemum flower, were assessed by USEPA in early 2005. Only the inhalation and incidental oral ingestion routes were examined, since dermal exposures were not observed to produce systemic effects in animal studies (USEPA, 2005a). This assessment included the effects on a 70 kg homeowner applying pyrethrin pesticides on lawns and home gardens, and on a 15 kg toddler directly exposed to turf on the day of treatment. No risks of concern were determined for the adult treating 0.5 acres per day of turf with a garden hose-end sprayer, or 1,000 square feet per day of garden with a dust applicator or trigger sprayer, when application rates conformed to label recommendations. Nor were risks of concern found for toddlers playing on treated turf. The assumptions of the study included an estimated turf transferable residue of five percent of the maximum application rate, an ingestion rate (area) from mouthing turf or small objects of 25 cm^2 and an object to mouth transfer efficiency of 20 percent, and a soil ingestion rate of 100 mg/day, with all of the active ingredient available in the top one cm of soil after application. Health risks to toddlers from indoor exposure to lawn and landscaping

pyrethrin pesticides were not evaluated. Ingestion of residues deposited on carpets and vinyl flooring following indoor fogger treatments was found to pose acceptable risks. The inhalation of indoor applications, however, was found to be problematic for both children and adults.

In conclusion, lawn and landscaping pesticide applications are known to result in direct outdoor exposures to residents, particularly young children, as well as indoor exposures when these chemicals are tracked or otherwise brought into homes. The health significance of these exposures is unknown, but they are likely to be much lower than those related to indoor and pet treatments involving the same pesticides. Recent USEPA regulations have sought to minimize exposures both outdoors and indoors.

Lice and Scabies Treatment

Lindane, malathion, pyrethrum, and permethrin are pesticides that are also used in the treatment of lice and scabies. Such uses are considered medical treatments and are regulated by the FDA. For lice, the following treatments are available:

- Kwell: one percent lindane shampoo, prescription only
- Ovide: 0.5 percent malathion lotion, prescription only
- Rid: 0.33 percent pyrethrum with piperonyl butoxide
- Nix: one percent permethrin cream rinse

For the treatment of scabies, available treatments include:

- Acticin and Elimite: five percent permethrin cream
- Lindane Lotion: one percent lindane
- Eurax: crotamiton cream
- Precipitated Sulfur Ointment: five to 10 percent

Lice and scabies appear to have developed resistance to all of these options. This makes it advantageous to have several alternatives available (Mathis, undated).

Lindane, also known as gamma-hexachlorocyclohexane, is an organochlorine insecticide. Lindane is a neurotoxin that can produce tremors, difficulty in walking, convulsions, and affect respiration. There is some indication that lindane may be an endocrine disruptor. USEPA considers there to be suggestive evidence that lindane may cause cancer based upon benign tumors observed in laboratory animals. However, USEPA does not consider the evidence to be sufficient for extrapolation to humans (USEPA, 2002c). Lindane can be absorbed through the skin or from oral ingestion.

For the treatment of scabies, lindane lotion (one percent) is applied to the whole body from the neck down. The lotion is to be left on for eight to twelve hours and then washed off. For lice, the shampoo (one percent) is to be applied to dry hair and left on for four minutes, followed by washing.

According to FDA estimates in 2003, up to one million prescriptions for lindane products are written each year by medical practitioners. Most of these are for school-aged children. However, FDA has issued a Public Health Advisory regarding the use of lindane for lice and scabies treatment, stating that lindane should be the second line of treatment and prescribed in patients who have either failed to respond to or are intolerant of other treatments options. Lindane should not be used in newborns and should only be used with caution in children or individuals who weigh less than 110 pounds. These warnings are based on serious adverse effects that have been reported. In 2002, FDA found 74 cases involved serious effects, including 15 deaths, 46 hospitalizations, seven life threatening symptoms, and six cases of birth defects. Fourteen of the 74 serious cases, including two deaths, appeared to use the lindane product according to label instructions and did not have any contraindications for use. Thirty-five (47 percent) of these serious cases were associated with the use of lindane for scabies. Lindane was verified as the cause of death in two of these cases, one of which was a six-month-old child, and the other a suicide. The most common adverse effects that have been reported involve the central nervous system and range from dizziness to seizures. Although FDA acknowledges there are risks associated with the use of lindane products, and there are alternatives that do not appear

as risky, FDA has concluded that it is in the best interest of public health to have lindane shampoo, available by prescription, as a second line therapy (Mathis, undated).

In the RED for lindane, USEPA (2002c) included a risk analysis of lice and scabies treatment. For scabies treatment, USEPA used a MOE approach as well as an analysis of blood levels observed following treatment. The MOE approach was based on a comparison of a "no observed adverse effect level" (NOAEL) from animal studies with estimated exposure. The target MOE was 100, which means an exposure that is 100 times less than the NOAEL would be considered within an acceptable risk. Based upon USEPA's estimates of exposure, all of the MOEs were below 100, the highest being 12 (for a young adult, assuming 10 percent dermal absorption and an applied dose representing the low end of potential exposure). This suggests the exposures to lindane resulting from scabies treatment may not provide an adequate margin of safety.

The highest blood level observed in children following scabies treatment with lindane was 64 ng/ml of blood, and the highest mean concentration was 28 ng/ml. The highest blood concentrations were observed six hours after the application of lindane lotion. These levels are approximately four to five times lower than a level detected (320 ng/ml) in the blood of a child admitted to an emergency room for symptoms following the accidental ingestion of a bottle of lindane lotion. The actual levels of lindane may have been higher in this child because the blood sample was drawn four hours after ingestion. Therefore, blood concentrations higher than 320 ng/ml may be necessary to produce adverse effects (USEPA, 2002c).

A similar study of blood lindane levels following the use of lindane shampoo, the lindane shampoo (Kwell) was left on longer than label directions (10 minutes versus the four minutes recommended). The highest level observed was six ng/ml in an individual that had to be re-treated five days after the initial treatment. The highest mean concentration in re-treated individuals was 3.6 ng/ml observed two hours after retreatment, as opposed to 1.4 ng/ml observed during the initial treatment. This level is well below the 320 ng/ml level detected in the blood of the child treated for accidental poisoning. USEPA concluded that the use of lindane for the treatment of lice was not a cause of concern (USEPA, 2002c).

Permethrin, the active ingredient in Nix, is also a mosquito control pesticide. Its toxicity was extensively discussed in the Literature Search (Book 6, Part 1) (CA-SCDHS, 2005). Permethrin is a pyrethroid insecticide that can cause neurological symptoms such as excitability, confusion, twitching, and tremors. It is classified as a possible carcinogen by USEPA and implicated in the Gulf War Syndrome. Nix is a one percent solution, so that each fluid ounce contains 280 mg of permethrin (Insight Pharmaceuticals, 2003). Permethrin is poorly absorbed through the skin with only 0.5 to one percent absorption of a dermal dose. If it is assumed that approximately one ounce of product is used to treat a six-year-old child (approximately 23 kg body weight), and that one percent of the applied dose is absorbed, the child would receive an estimated dose of 120 ug/kgbw. This is approximately half the acute oral RfD (250 ug/kgbw per day) or Minimum Risk Level (300 ug/kgbw per day). Since the shampoo is to be left on the hair for only 10 minutes before it is washed off and much of the shampoo is applied to the hair as opposed to directly to the scalp, this is likely an overestimate of actual exposure.

Similarly, malathion, the active ingredient in Ovide, is also a mosquito control insecticide. The toxicity of malathion was similarly discussed in the literature review (CA-SCDHS, 2005). Malathion inhibits cholinesterase, as do other organophosphates, and leads to nervous system effects. Symptoms of overexposure can include difficulty in breathing, vomiting, diarrhea, blurred vision, loss of consciousness, and even death. Ovide is only available with a prescription and contains 0.5 percent malathion, and is available in two fluid ounce treatments. If the same assumptions are used as in the above Nix example, an applied dose to the hair and scalp could be conservatively estimated to be 150 mg. Assuming a 10 percent dermal absorption rate, the estimated, absorbed dose is 0.65 mg/kgbw per day, which is just above the acute oral RfD of 0.5 mg/kgbw per day. Again, this is likely an overestimate since much of the malathion would be in contact with the hair and not directly on the scalp. However, unlike the permethrin product, Ovide is supposed to remain on the hair for eight to 12 hours before washing.

In summary, some pesticides are used pharmaceutically to treat scabies and lice. The risks associated with the lindane and malathion products appear greater than the permethrin product. Both the lindane and malathion products require a prescription. As with any medication, any health risks are offset by the health benefit of treating these conditions.

Domestic Animal Risk

Little information was available on the potential background risks of pesticide exposure to domestic animals (e.g., dogs and cats). The bulk of the information was obtained from REDs for those pesticides that are used in pet products for flea and tick control. Information was also sought from veterinary websites.

As with small children, pets are more likely to be exposed to pesticides and receive a higher dose since they are lower to the ground, can track pesticide residues indoors from outside, and their grooming habits can lead to oral ingestion of pesticide residues on their fur and paws. In most cases, susceptibility to the potential toxic effects of pesticides is similar in pets as in humans (EXTOXNET, 1998). According to the American Society for the Prevention of Cruelty to Animals (ASPCA), the most dangerous pesticides include snail baits that contain metaldehyde, fly baits containing methomyl, the insecticide disulfoton, zinc phosphide mole or gopher bait, and most forms of rat poisons (ASPCA, 2005). According to data compiled by the ASPCA in 2003, pet poisonings occur more frequently in the summer months. In July of 2003, approximately 9,000 calls were received by the ASPCA Poison Control Center; 4,300 (48 percent) of which were related to pesticide exposure in pets.

The National Pesticide Information Center recommends that pets and their chew toys or bones be removed from any area being treated with pesticides. Pesticide product labels should be carefully reviewed to ensure that pets are not allowed back in the treated area before the re-entry interval that is listed on the label. At least, pets should not be allowed in a treated area until the pesticide product has completely dried. For example, product labels for pesticide products containing metaldehyde (designed to kill slugs and snails) carries a warning "This pesticide may be fatal to dogs or other pets if eaten. Keep pets out of treated areas" (EXTOXNET, 1996). Many outdoor granular pesticides require that pets not be allowed on the treated area for 24 hours (NPIC, 1998). Fish tanks should be covered and the pump turned off if foggers are being used indoors. It is very important that insect and rodent baits be placed in areas that are not accessible to pets.

For products that are designed to be used on pets it is very important that the instructions are followed carefully, so that over exposure does not occur. In July of 2003, the ASPCA Animal Poison Control Center received 3,100 cases of poisoning related to the use of flea and tick control products (ASPCA, 2004). Some products are labeled for dogs and should not be used on cats, and those that are labeled for adult cats or dogs should not be used on kittens or puppies. It is also very important that certain flea and tick products not be used on very old or debilitated animals since they may be more susceptible.

The following pesticide active ingredients are approved for use on pets:

- Phenothrin and pyrethrum
- Methoprene
- Carbaryl
- Imidacloprid
- Fipronil
- Permethrin
- Cypermethrin

Recently Hartz Mountain Corporation agreed to cancel several flea and tick products designed for use on cats and kittens. These products contained phenothrin, a pyrethroid also known as sumithrin, due to reports of adverse effects, such as hair loss, salivation, tremors, and numerous deaths in cats and kittens (USEPA, 2005b). Permethrin, another pyrethroid, has also caused toxicity in cats when products listed for use on dogs are inadvertently used on cats. Permethrin "spot-on" products for dogs can contain between 45 to 65 percent permethrin. Even small amounts of these products can cause symptoms in cats (Richardson, 2000). Symptoms that are most often seen in cats from permethrin toxicosis include tremors, muscle twitching, and seizures. These symptoms usually occur within hours to days following treatment and may last up to three days.

The ASPCA reports that the second most serious poisoning cases reported to them result from ingestion of rodenticides. In July 2003, 470 cases involved rodenticides. Ingestion of rodenticides can lead to bleeding, seizures, and death. Most rodent and ant or snail and slug baits have ingredients that can attract your pet as well as the target pest (ASPCA, 2004). Anti-coagulants are the most common type of rodenticides and include active ingredients such as warfarin, brodifacoum, bromadiolone, difethialone, diphacinone, and pindone. Signs of poisoning from these rodenticides may occur one to seven days after ingestion and include lethargy, anorexia, lameness due to hemorrhaging, coughing, and difficulty in breathing. Internal bleeding may occur and lead to seizures and death if the bleeding occurs in the brain or spinal cord (Merola, 2002). A newer type of rodenticide is cholecalciferol (also known as vitamin D3). Its use leads to higher levels of calcium in the blood and can cause renal failure. Symptoms observed in dogs include vomiting, lethargy, and muscle weakness. A dose considered to be lethal to dogs is approximately 110 mg/kgbw (Craigmill, 1988).

A pesticide used to control snails and slugs that contains metaldehyde is also very toxic to dogs, in particular. Doses as low as 23 mg/kgbw have caused toxicity. Symptoms of toxicity may begin to show minutes to hours after exposure and include anxiety, panting, irregular heartbeat, vomiting, diarrhea, and difficulty in walking. Death may occur within a few hours due to respiratory failure. The exact mechanism of toxicity is not well understood (Dolder, 2003).

Integration and Summary

There are many ways that people are exposed to pesticides in every day life. The biomonitoring studies conducted by the CDC show that many pesticides can be detected in blood and urine, including those pesticides that have been banned in the US for some time (e.g., DDT). Based on the New York Pesticide Sale and Use Database, it is likely that Suffolk County is the leading county in New York in terms of quantity of pesticides used. Reports of human health impacts have been compiled by various registries, such as the New York State Pesticide Poisoning Registry, although often documentation of cause and effect are lacking. In almost all of the studies and data sources evaluated, young children appear to be more highly exposed and at greater risk than the rest of the population.

A perspective on the relative contribution from these varied uses of pesticides can be gained by comparing the risks associated with each use. This was done in the USEPA organophosphate cumulative risk assessment. When all the exposure pathways evaluated were combined, the MOE for the 95th percentile in the highest exposed age group (children age one to two) was about 500. This would correspond to a dose of 0.16 ug/kgbw per day (based on methamidophos equivalents). For this percentile, food was by far the most significant pathway of exposure. For the higher percentiles of exposure, inhalation via the residential exposure pathway became the predominant pathway during seasons when dichlorvos was used in pest strips and crack and crevice treatments, decreasing the overall MOE to less than 100. Other residential exposures resulted in MOEs that were orders of magnitude higher.

In another approach, various RED documents, prepared by USEPA, were selected based upon availability, known use in Suffolk County, and observed impact to local groundwater. These REDs were reviewed and estimates of risk were compiled in Table 3-34 (noncancer risks) and Table 3-35 (cancer risks).

Many of the uses with low MOEs (that is to say, highest risk) have been discontinued (e.g., chlorpyrifos). Of the remaining uses, those with the lowest MOEs include turf (carbaryl, pendimethalin) and pet use (carbaryl, tetrachlorvinfos). Risks from the food pathway also had a low MOE (piperonyl butoxide and dacthal).

In terms of cancer risks, the only risks presented in Table 3-35 that are worse than one in a million are the upper end of dacthal use on turf and contamination of drinking water, and the use of tetrachlorvinphos for pet dipping.

In certain circumstances, such as when groundwater that is used for drinking and bathing has become contaminated, other pathways can result in relatively high risks from pesticide exposure.

Table 3-34. Non-Cancer Risks from Different Pathways of Exposure

PESTICIDE ACTIVE INGREDIENT	FOOD/DIETARY PATHWAY (MOE)	DRINKING WATER PATHWAY	MOSQUITO CONTROL (MOE)	PET TREATMENTS (MOE)	TURF (MOE)	INDOOR TREATMENTS (MOE)	REF
Tetrachlorvinphos							USEPA, 1999a
Dog collar, handler				86-280			
Cat collar, handler				96-470			
Post-application, child hand to mouth				0.25-0.97			
Piperonyl Butoxide							USEPA, 2005c
General population	20	0.26 to 60 ug/L	75000 to 740000			3800 (inhal)	
Child	8 (1-2 yrs)		20000 to 160000			1000 (inhal)	
Toddler, incidental ingestion			4800	600		2600 to 4200	
Carbaryl							USEPA, 2003b
Dog dusting, handler				4-7			
Dog collars, handler				>1000000			
Residential post-application, adult			>600->30000		43->800		
Residential post-application, toddler			>400-3000	>300	5-91		
Chlorpyrifos							USEPA, 2002b
Adult			43000	670-2500*	9-110*	390*	
Child			15000	140-530*	7.5-73*	110*	
Malathion							USEPA, 2000
General Population	12600	32 ug/L	5600-22000		100-150000		
Child	6300	6 ug/L	2200-7700		60-90,000		
MCPA							USEPA, 2004d
General Population	Below concern						
Toddler post-application					280-470		
DCPA (dacthal)							USEPA,

PESTICIDE ACTIVE INGREDIENT	FOOD/DIETARY PATHWAY (MOE)	DRINKING WATER PATHWAY	MOSQUITO CONTROL (MOE)	PET TREATMENTS (MOE)	TURF (MOE)	INDOOR TREATMENTS (MOE)	REF
							1998a
General Population	2-74 % of RFD	0.002-11%of RFD					
Child							
Metalaxyl							USEPA, 1994
General Population	8-16% of RFD						
Child	15-31% of RFD						
Metolachlor							USEPA, 1995
General Population	0.2% of RFD						
Nursing infants	0.6% of RFD						
Pendimethalin							USEPA, 1997
Child					71-333		
Golf course, 2hrs post treatment					2917		
Pyrethrins							USEPA, 2005d
Pet Groomers				12000			
Residential applicator					720000	9700-200000	
Adult			8900-89000				
Child			2700-8900				
Toddler				40000	1800	6900-9500	
Naled							USEPA, 1999b
Adult			97-1500	57-222			
Toddler			100-1500	10-37			
Acephate							
Adult		0.02 ug/L			507		USEPA, 2001b
Toddler					7.4	2.8-9	

* Use has since been removed from registration

Table 3-35. Cancer Risks from Different Pathways of Exposure

Pesticide Active Ingredient	Food/Dietary Pathway	Drinking Water Pathway	Mosquito Control	Pet Treatments	Turf Treatments	Indoor Treatments	Ref
Tetrachlorvinphos							USEPA, 1999a
Dog collar, handler				9×10^{-8} to 3×10^{-7}			
Cat collar, handler				1×10^{-10} to 5×10^{-8}			
Dipping, handler				8×10^{-6} to 3×10^{-5}			
DCPA							USEPA, 1998a
DCPA	3.5×10^{-7}	3×10^{-10} to 2×10^{-6}					
HCB impurity	7×10^{-7}						
TCDD impurity	7×10^{-8}						
Child					6×10^{-7} to 2×10^{-6}		
Residential Application					9×10^{-9} to 2×10^{-6}		

3.5.2 Ecological Risks from Background Pesticide Exposures

The potential ecological significance of the collected Suffolk County aquatic data were assessed by comparing measured concentrations with benchmark screening values available from the Risk Assessment Information System (RAIS) developed by the US Department of Energy. The Environmental Sciences Division of Oak Ridge National Laboratory, in collaboration with The Institute for Environmental Modeling at the University of Tennessee and the Bechtel Jacobs Corp., developed and compiled this comprehensive set of eco-toxicological screening benchmarks for surface water, sediment, and surface soil, which are applicable to a range of aquatic organisms, soil invertebrates, and terrestrial plants (Oak Ridge National Laboratory, 2005). These screening ecological benchmarks can be used to identify chemical concentrations in environmental media that are at or below thresholds for effects to ecological receptors.

Surface Water

The RAIS lists ecological benchmarks established by the National Guidelines and Standards Office of the Environmental Quality Branch of Environment Canada for a few of the pesticides detected by Suffolk County most frequently in surface waters, such as aldicarb and metolachlor (see Table 3-22). These Canadian Water Quality Guidelines (CWQGs) were developed to provide basic scientific information about water quality parameters and ecologically relevant toxicological threshold values for Canadian species, but are assumed to be relevant to Suffolk County ecosystems as well. In deriving CWQGs for aquatic life, all components of the aquatic ecosystem (e.g., algae, macrophytes, invertebrates, and fish) are considered, if the data are available, in order to protect all life stages during an indefinite exposure to water. For most water quality variables, a single maximum value, which is not to be exceeded, is recommended as the CWQG. This maximum value is based on a long-term no-effect concentration. When available, the lowest-observable-effects level (LOEL) from a chronic exposure study on the most sensitive native Canadian species is multiplied by a safety factor of 0.1 to arrive at the final guideline concentration. Alternatively, the lowest LC_{50} or EC_{50} (concentration causing a 50 percent reduction in the endpoint being measured) from an acute exposure study is multiplied by an acute/chronic ratio or the appropriate application factor (i.e., 0.05 for non-persistent variables; 0.01 for persistent variables) to determine the final guideline concentration.

As can be seen from Table 3-22 (see above), the aldicarb metabolites, as well as metolachlor and its metabolites, did not exceed the CWQGs for their respective parent compounds; it is not clear, however, whether some of the breakdown products may be more toxic to ecological receptors than the parent compounds (see discussion of alachlor, below). Other chemicals of note listed on Table 3-22 are simazine, a weed killer used by utilities, which was found at concentrations below its CWQG, and dinoseb, a broad-spectrum phenolic pesticide banned in 1988, which continues to be detected in surface waters at concentrations well above its CWQG (and USEPA benchmark level of 0.5 µg/l). Three other pesticides that have been found frequently, but without CWQGs or other benchmarks for comparison, are DEET, used as a mosquito repellant, TCPA, the breakdown product of the banned herbicide dacthal, and metalaxyl, a fungicide used on sod, vineyards, and other crops.

Some of the most frequently detected pesticides in Suffolk County surface waters (particularly in East End streams) are the breakdown products of the herbicides alachlor and metolachlor; unfortunately, no ecological benchmark values for these chemicals are listed in the RAIS. Parent alachlor has been found in Suffolk groundwater at concentrations that often exceed the alachlor EC_{50} (1.64 µg/l) for green algae, and the no effect level of 0.35 µg/l (SCDHS, 2002b). Also of concern is the fact that alachlor degradates often occur in combination with higher concentrations of metolachlor metabolites. The USEPA Alachlor RED (USEPA, 1998b) notes that:

> Aquatic plants may be adversely affected by alachlor in groundwater, in places where groundwater discharges into surface water.

Similarly, the metolachlor RED (USEPA, 1995) states:

> In areas where irrigation water is contaminated with metolachlor, or where groundwater discharges to surface water, metolachlor residues could present a threat to non-target plants.

The relative toxicities of the herbicide metabolites to aquatic plants are unknown; however, SCDHS has found alachlor and metolachlor metabolites in at least seven streams discharging into the Peconic Bays, often in concentrations well in excess of the no effect level for alachlor and the EC_{50} for green algae (SCDHS, 2002b). SCDHS has expressed a concern about the potential for alachlor and metolachlor metabolites in groundwater underflow and stream flow to

the Peconic Estuary to act as selective herbicides, killing or inhibiting the growth of green algae; this may provide the trigger mechanism that allows *Aureococcus anophagefferens* to out-compete other forms of estuarine algae under certain conditions, thereby initiating a brown tide bloom (SCDHS, 2002b). Extensive agricultural use of the two herbicides preceded the initial brown tide bloom in 1985 (Cornell Cooperative Extension, 2001).

Sediment

As indicated earlier, the sediment data in Tables 3-25 through 3-28 are limited to organochlorines – the only pesticides tested for, but also the ones most likely to accumulate in organic-rich bottom sediments. These data indicate the persistence of the organochlorine pesticides like DDT, chlordane, endosulfan, heptachlor, and methoxychlor. Screening benchmarks for some of these pesticides are available in the RAIS from various sources. For example, benchmark levels for 4,4-DDT range from 0.0012 mg/kg to 0.71 mg/kg, and some sample results exceed even the higher screening level. Any of these persistent chemicals could be taken up by local biota and concentrated as they pass up the food chain. The ecological significance of this process, or the potential threat to human health, could not be documented.

Biota

The BNL tissue data (Table 3-29) were collected from fish in the Peconic River to assess the impacts of the BNL sewage treatment plant; the sample locations for the fish tissue data in STORET (Table 3-30), however, are unknown, as are those for the NOAA mollusk tissue data (Table 3-31). The tissue data in Table 3-32 are for fresh water mollusks collected in 2000 by SCDHS from the Peconic River (which were analyzed by a contract lab); this study was conducted in order to assess the impacts of the BNL sewage treatment plant.

Similar to the sediment data, the biota data are limited to organochlorine pesticides. The impacts of the measured concentrations on the subject fish and shellfish are unknown, as there are apparently no screening benchmark levels with which to compare. But these data do imply that long-term exposures to pesticides banned in the 1970s are still occurring, and have the potential to pass up the food chain to predators and even man.

Conclusions

This review of available data on pesticide levels found in environmental media in Suffolk County indicates that many of the pesticides that are detected are pesticides that have been banned but are still persistent in the environment. These include organochlorine pesticides such as DDT and it breakdown products, chlordane, and dieldrin. In addition, for many of these pesticides and their breakdown products there often are not standards or criteria for specific environmental media (e.g., sediment or fish tissue). The lack of such standards makes the evaluation of ecological impact difficult within the scope of this project.

Surface water had the most data available for review. Sources such as the SCDHS and the USGS NAWQA databases were reviewed and showed generally low levels of contamination from water soluble pesticide-related contaminants. Concentrations were typically between one and two ppb. The most frequently detected compounds were the breakdown products of metolachlor, aldicarb, and alachlor. Dinoseb was detected above a Canadian water quality criterion. The dacthal breakdown product, TCPA, and DEET were detected at the highest levels; however, there are no water quality standards for these two contaminants.

SCDHS has expressed concern for the detection of alachlor degradates in surface water at concentrations that exceed the parent alachlor EC_{50} for growth effects on green algae. If alachlor degradates are as toxic to green algae as the parent alachlor they may enable the brown tide algae to outcompete other forms of estuarine algae.

Sediment and biota data were very limited, making it difficult to draw many conclusions. Detections of pesticides in sediment were limited to organochlorine pesticides. Only DDT related compounds exceeded screening levels. DDD was detected at the highest concentration (85,740 ug/kg). Similar to sediment data, fish samples have detected primarily organochlorine pesticides. Though the ecological impacts of this contamination are difficult to determine, it is apparent that pesticides that were banned in the 1970s are still present and bioaccumulating in the food chain.

3.6 Breast Cancer Incidence

Breast cancer is the second most common cancer in the world when both sexes are combined and the most common cancer among women. The annual incidence rate of breast cancer varies widely with respect to geographic area. Based on the year 2000 world standard population, the Netherlands (91.6/100,000 women per year) and the US (91.4/100,000 women per year) have the highest breast cancer incidence rates in the world; this can be compared to the rate in China (10.0/100,000 women per year), which is among the lowest (Parkin, 2001).

Among US women in 2000, the overall age-adjusted annual incidence rate of invasive breast cancer was 135.1/100,000 women. The incidence rate of breast cancer is strongly associated with age. Among women under 65 years of age, the age-adjusted incidence rate was 90.4 / 100,000 women per year and for women 65 years and older, it was 459.6/100,000 women per year (Ries et al., 2003). It has been estimated that 215,990 women in the United States will be newly diagnosed with breast cancer in 2004, making it the most common cancer among women, more than lung, colorectal and endometrial cancer (Jemal et al., 2004).

There are racial and geographic variations in the annual incidence rates of breast cancer reported across the US Surveillance, Epidemiology and End Results (SEER) sites. The SEER Program of the National Cancer Institute is an authoritative source of information on cancer incidence and survival in the United States. According to its website,

> "SEER began collecting data on cases on January 1, 1973, in the states of Connecticut, Iowa, New Mexico, Utah, and Hawaii and the metropolitan areas of Detroit and San Francisco-Oakland. In 1974-1975, the metropolitan area of Atlanta and the 13-county Seattle-Puget Sound area were added. In 1978, 10 predominantly black rural counties in Georgia were added, followed in 1980 by the addition of American Indians residing in Arizona. Three additional geographic areas participated in the SEER program prior to 1990: New Orleans, Louisiana (1974-1977, rejoined 2001); New Jersey (1979-1989, rejoined 2001); and Puerto Rico (1973-1989). In 1992, the SEER Program was expanded to increase coverage of minority populations, especially Hispanics, by adding Los Angeles County and four counties in the San Jose-Monterey area south of San Francisco. In 2001, the SEER Program expanded coverage to include Kentucky and Greater California; in addition, New Jersey and Louisiana once again became participants. The SEER Program currently collects and publishes cancer incidence and survival data from 14 population-based cancer registries and three supplemental registries covering approximately 26 percent of the US population.

> Information on more than 3 million *in situ* and invasive cancer cases is included in the SEER database, and approximately 170,000 new cases are added each year within the SEER coverage areas. The SEER Registries routinely collect data on patient demographics, primary tumor site, morphology, stage at diagnosis, first course of treatment, and follow-up for vital status. The SEER Program is the only comprehensive source of population-based information in the United States that includes stage of cancer at the time of diagnosis and survival rates within each stage. The mortality data reported by SEER are provided by the National Center for Health Statistics."

(http://seer.cancer.gov/about/)

In 2000, the annual incidence rate of breast cancer in the US among white women was 140.9/100,000 women while the rate among blacks was 116.3/100,000. The geographic variation is most striking among white women where annual incidence rates ranged from 143.1/100,000 women in the Connecticut registry to 116.8/100,000 women in Utah (Ries et al., 2003). Breast cancer has a high five year relative survival rate (87 percent), especially if detected at an early stage (97 percent survival rate) (Jemal et al., 2004).

According to the New York State Cancer Registry, Nassau and Suffolk counties had among the highest average annual incidence rates of breast cancer in the state, 144.2/100,000 women in Nassau County and 148.4/100,000 women in Suffolk County, for 1996 to 2000. The racial variation in breast cancer incidence observed in the SEER data is found within the two Long Island counties. Among white women, the average annual incidence rate for the same time period was 148.6 and 150.9/100,000 women in Nassau and Suffolk counties, respectively. In contrast, the annual average breast cancer incidence among black women in these two counties was 108.7 and 113.9/100,000 women (NYSDOH, 2003).

A study of the relationship between potential pesticide exposure (through past agricultural land use, detections in drinking water, and residence within one mile of a hazardous waste site) and breast cancer incidence was made on Long Island. After adjustments were made for other, potentially confounding risk factors, it was found that living within a mile of a hazardous waste site increased breast cancer risks (as measured by odds ratios). Women who had not given birth, or had first given birth at older ages (26 or older), and living on land previously used for agricultural purposes, had a statistically significant elevated risk (as measured by odds ratios) compared to women who gave birth at younger ages and did not live on these former agricultural

land. However, the statistical power of the study was limited, resulting in wide confidence intervals that made it difficult to discern whether effects had occurred or not (O'Leary et al., 2004).

3.7 Children's Health and Pesticides

3.7.1 Background

Children are considered particularly vulnerable to pesticide exposures due to their physiological, behavioral, and biological characteristics. Children eat and drink higher quantities per body size than adults resulting in a greater likelihood of exposure opportunities. These behaviors include living closer to the ground, hand-to-mouth behaviors, and outdoor activity such as playing in parks and playgrounds where pesticides are used. Biologic development in children is rapid and makes them susceptible to chemical insult. Disruption of neurological and organ development can easily occur. Children's blood-barriers are immature. Additionally, children's ability to detoxify and excrete toxins may be reduced due to their maturing metabolic processes. Beyond immediate effects that can be experienced, early exposure to pesticides in children allows for a longer time period over which chronic or delayed disease can develop (Dunn et al., 2003).

Further evidence of children's vulnerability to pesticide exposure is found in the data from NHANES. An analysis of urinary dialkyl phosphate metabolites of organophosphate pesticides (such as but not exclusively, malathion) identified statistically significantly higher concentrations of these metabolites in children age six to 11 years of age when compared to the concentration levels measured in adults (20 to 59 years of age) (Barr et al., 2004). Similar findings were obtained from a study of farm workers and their children; mean levels of several organophosphate metabolites were slightly higher among children than among adults (Mills and Zahm, 2001). No statistical comparisons between levels in children and adults were made because of small sample sizes.

3.7.2 Cancer

Childhood cancer is not a single disease but a wide range of malignancies that vary by histology, organ, race, sex, and age. The major groupings of childhood cancers are:

I. Leukemia

II. Lymphomas and reticuloendolthelial neoplasms

III. Central nervous system and miscellaneous intracranial and intraspinal neoplasm

IV. Sympathetic nervous system

V. Retinoblastoma

VI. Renal tumors

VII. Hepatic tumors

VIII. Malignant bone tumors

IX. Soft-tissue sarcomas

X. Germ-cell, trophoblastic and other gonadal tumors

XI. Carcinomas and other malignant epithelial neoplasm

XII. Other and unspecified malignant neoplasms

(Ries et al, 1999)

Cancer incidence is documented by the SEER Program of the National Cancer Institute, an authoritative source of information on cancer rates and survival in the United States. Incidence rates for several types of childhood cancer have increased since the 1970s (Figure 3-19). Leukemia, lymphoma, and central nervous system neoplasms have the highest incidence rates, while retinoblastoma, hepatic and other neoplasms have the lowest (Figure 3-20). In general, there appears to be a gender difference when all cancer sites are combined, rates are higher for boys than for girls (Figure 3-19). However, site-specific cancer rates do not always follow this overall trend.

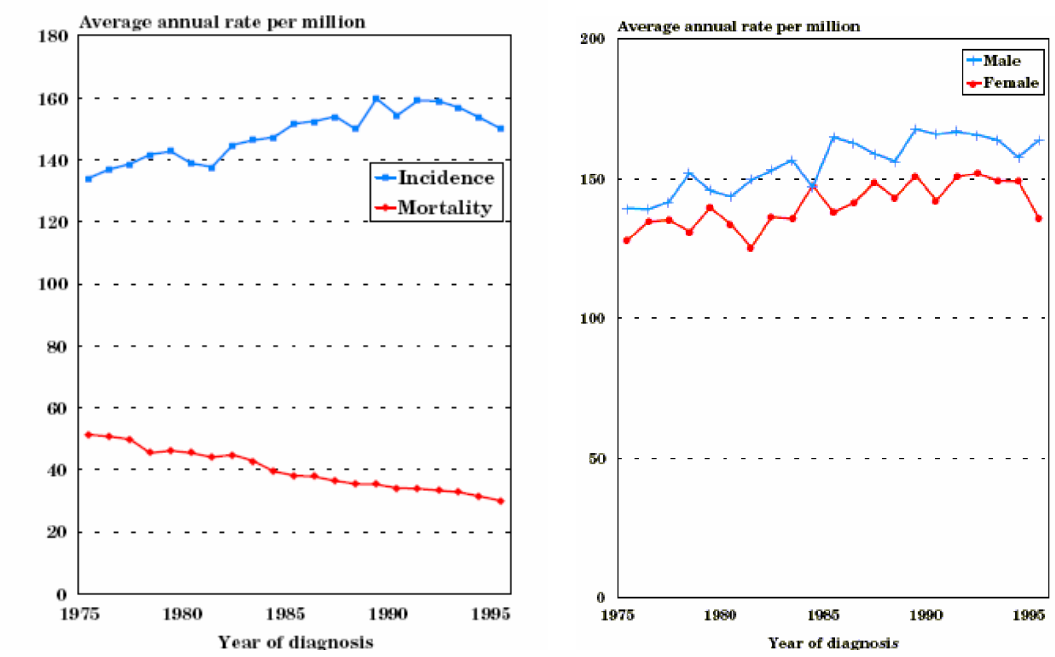


Figure 3-19. Incidence and Mortality Rates for Childhood Cancers (Totals and by Gender)

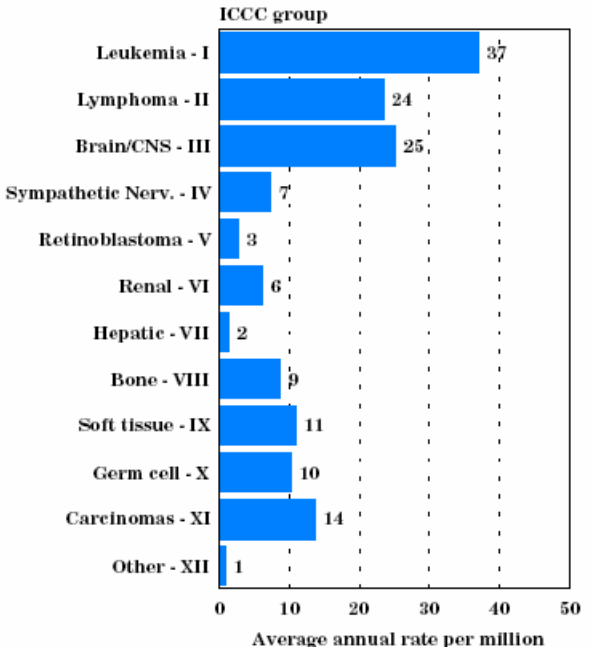


Figure 3-20. Incidence Rates for Childhood Cancers by Cancer Type

The epidemiological literature on the relationship between pesticides and childhood cancer is abundant, and predominately through case-control studies (see Table 3-36 below). Overall, the literature suggests moderate associations between pesticide exposure and some cancers in children.

However, in spite of some clear trends, exposure assessments cited in the literature are subject to much confounding. An exposure is considered a "confounder" if it is related to the exposure of interest and it is associated with the disease being studied. If a confounder is not accounted for in a statistical analysis then any observed exposure-disease associations may be due to the confounder rather than the exposure being studied.

No actual biomarkers have been measured. Biomarkers are biological indicators of exposure that can be measured in the blood, body fluids, cells, and tissues. Biomarkers can be used as surrogate measures of chemical dose. Instead, exposure data is based on proximity to areas of pesticide use, and/or more often self-reported interview responses, which are subject to recall bias. Interviews are based on parents' reports of their own occupational exposures, household,

yard, and garden spraying, and their children's direct exposures, since young children are obviously not a dependable source of information. Few studies isolated specific pesticides, and those that did focused on substances not relevant to this assessment of mosquito control pesticides. Despite the numerous specific groupings for childhood cancer described above, most of the papers are only concerned with leukemias, lymphomas, and brain cancers.

Childhood Cancer – Leukemias

Of the seven studies reviewed that focused on childhood leukemia and pesticides, six found a positive association between disease and exposure (Leiss and Savitz, 1995; Meinert et al., 1996; Kaatsch et al., 1998; Petridou and Dessypris, 2000; Ma et al., 2002), although not exclusively significant, and one found no association (Feychting et al., 2001). Pesticide exposure assessment spanned from prenatal development through childhood. All studies supporting an association, focused on exposures inside or outside the home, although it is unclear whether this difference causes a variation in risk. The one study that did not find any positive association between leukemia and pesticide use was based on preconception occupational pesticide exposure of the child's father (Feychting et al., 2001). Based on the articles reviewed, it appears that leukemia is linked to non-specific pesticide use; none of these studies provided information on pesticide types beyond general categorization (e.g., insecticides). This finding is consistent with previous reviews (Zahm and Ward, 1998).

Childhood Cancer – Lymphomas

The literature includes several studies on pesticides and lymphomas which support a positive association between the two. The association is, however, not quite as strong as the association of pesticides with leukemia. One study found significant associations between lymphoma and a child's direct exposure, as well as lymphomas and exposure from a mother's spraying around the house (Buckley et al., 2000).

Three studies found an elevated odds ration for pesticide use in the home (Leiss and Savitz, 1995; Meinert et al., 2000; Buckley et al., 2000). An odds ratio is a measure of association between an exposure and a disease outcome. An odds ration of one is interpreted as no association between exposure and disease. An odds ration greater than one is interpreted as an

increased risk of disease associated with exposure. Two of studies were significant on at least one strata of exposure (Leiss and Savitz, 1995; Meinert et al., 2000) and two exhibited a dose-response relationship (Meinert et al., 2000; Buckley et al., 2000), although the Buckley findings for indoor pesticide use are based on a small sample size. Use of pest strips in the home, which was highly associated with leukemias, was not found to be significantly associated with lymphomas (Leiss and Savitz, 1995). Further, outdoor use of pesticides (yard/garden/farm) yielded no significant relationship with lymphoma in any of the studies reviewed.

Childhood Cancer – Brain and Nervous System

Studies on cancers of the brain and central nervous system also supported an association with pesticide use. Within the seven studies investigating cancers of the brain and central nervous system (including neuroblastomas), odds ratios varied from protective to elevated; however five studies found significant increased risks (Leiss and Savitz, 1995; Olshan et al., 1999; Daniels et al., 2001; van Wijngaarden et al., 2003; Efird et al., 2003). One of these positive associations was significant for general association (ever use) but not when stratified to preconception or child exposure (Daniels et al., 2001). Brain cancer risk associated with outdoor pesticide exposure, either through farm, garden, or yard uses, was significantly decreased in one study (Schuz et al., 2001a), significantly increased in another (Daniels et al., 2001), and not found with any association in yet another study (Leiss and Savitz, 1995). Two studies reported a positive pesticide association based on parental occupation (van Wijngaarden et al., 2003; Efird et al., 2003). Finally, another study found that risks were increased with prenatal exposure in comparison with those exposed during childhood (Zahm and Ward, 1998).

Childhood Cancer – Other Cancers

Other cancers were often grouped as solid tumors, soft tissue sarcomas, kidney cancers, and/or included general cancer mortality rates of a geographic area. Results varied by type of cancer. While one study found a high odds ratios relating yard treatment to soft tissue sarcomas (Leiss and Savitz, 1995), no significant findings were identified for total mortalities (Pearce and Parker, 2000; Schreinemachers et al., 1999), solid tumors (Meinert et al., 1996), or kidney cancer (Schuz et al., 2001b). However, other researches (Zahm and Ward, 1998) suggest the risk of kidney cancer is increased among children whose parents farm for long periods of time. Feychting et al.

(2001) found a significant elevation in nervous system tumors related to paternal exposure preconception; however, this was based on occupational exposure via agricultural, horticultural, and forestry management, which may have included exposure to many other possible carcinogens.

Table 3-36. Summary of Epidemiologic Studies of Pesticide Exposure and Childhood Cancer

Study design and Populations	Exposure	Measures of Association				Variables adjusted for confounding
		Leukemia	Lymphomas	Brain/Central Nervous System Tumors	Other	
Leiss, 1995 Population-based case control -Denver, CO, USA, Cancer registry and area hospital records -252 cases; < 15 years; diagnosed between 1976-1983 -222 controls	**Home pest extermination** **0-2 years**	OR (95% CI) 0.3 (0.1-0.8)	OR (95% CI) 1.8 (1.1-2.9)	No significant findings at any strata	No significant findings at any strata	Age at diagnosis, father's education, per capita income, residential stability, mother's age, race, sex, maternal smoking, residential wire code (magnetic field exposure), and year of diagnosis (controlled separately)
	Yard Treatment **0-2 years** **2 years prior to and through diagnosis**	No significant findings at any strata	No significant findings at any strata	No significant findings at any strata	OR (95% CI) (soft tissue sarcomas) 4.1 (1.0-16.0) (soft tissue sarcomas) 3.9 (1.7-9.2)	
	Hanging pest strips (indoors) **Last 3 months of pregnancy** **0-2years** **2 years prior to and through diagnosis**	3.0[1] (1.6-5.7) 1.7 (1.2-2.4) 2.6 (1.7-3.9)	No significant findings at any strata	1.8 (1.2-2.9)	Total cancers 1.5 (1.0-2.8)	
Meinert 1996 Population–based case control study -Lower Saxony, Northern Germany, German Children Cancer Registry -173 leukemia cases, and 175 CNS tumors; <15 years; diagnosed 1988 –1993; -220 local controls, 213 state controls	**Ever use** **In garden** **On farm**	OR (95% CI) (v. local controls) 2.47 (1.13-5.38) 2.52 (1.03-6.14) 1.64 (0.55-0.53)			(solid tumors) No significant findings in any strata	Sex, age, social status, degree of urbanization
Kaatsh 1998 Population based case control study, West Germany, German Childhood Cancer Registry -560 cases; <15 years; diagnosed 1992-1994 -560 controls	**Use in agriculture, household or garden**	OR (95% CI) 2.13 (1.05-4.35)				Socio-economic status

Study design and Populations	Exposure	Measures of Association				Variables adjusted for confounding
		Leukemia	**Lymphomas**	**Brain/Central Nervous System Tumors**	**Other**	
Leiss, 1995 Population-based case control -Denver, CO, USA, Cancer registry and area hospital records -252 cases; < 15 years; diagnosed between 1976-1983 -222 controls	**Home pest extermination** **0-2 years**	OR (95% CI) 0.3 (0.1-0.8)	OR (95% CI) 1.8 (1.1-2.9)	No significant findings at any strata	No significant findings at any strata	Age at diagnosis, father's education, per capita income, residential stability, mother's age, race, sex, maternal smoking, residential wire code (magnetic field exposure), and year of diagnosis (controlled separately)
	Yard Treatment **0-2 years** **2 years prior to and through diagnosis**	No significant findings at any strata	No significant findings at any strata	No significant findings at any strata	OR (95% CI) (soft tissue sarcomas) 4.1 (1.0-16.0) (soft tissue sarcomas) 3.9 (1.7-9.2)	
	Hanging pest strips (indoors) **Last 3 months of pregnancy** **0-2years** **2 years prior to and through diagnosis**	3.0[1] (1.6-5.7) 1.7 (1.2-2.4) 2.6 (1.7-3.9)	No significant findings at any strata	1.8 (1.2-2.9)	Total cancers 1.5 (1.0-2.8)	
Meinert 1996 Population–based case control study -Lower Saxony, Northern Germany, German Children Cancer Registry -173 leukemia cases, and 175 CNS tumors; <15 years; diagnosed 1988 –1993; -220 local controls, 213 state controls	**Ever use** **In garden** **On farm**	OR (95% CI) (v. local controls) 2.47 (1.13-5.38) 2.52 (1.03-6.14) 1.64 (0.55-0.53)			(solid tumors) No significant findings in any strata	Sex, age, social status, degree of urbanization
Kaatsh 1998 Population based case control study, West Germany, German Childhood Cancer Registry -560 cases; <15 years; diagnosed 1992-1994 -560 controls	**Use in agriculture, household or garden**	OR (95% CI) 2.13 (1.05-4.35)				Socio-economic status

Study design and Populations	Exposure	Leukemia	Lymphomas	Brain/Central Nervous System Tumors	Other	Variables adjusted for confounding
Schreinemachers 1999 Retrospective cohort; -Minnesota, USA, National Center for Health Statistics, 1980-1989 -< 15 years	**Agricultural regions**				SRR (95%) Reference group= Region 4 (urban) No significant results in specific cancers. (Total cancer mortalities) Region Boys Girls 1 0.82 (0.59-1.13) 0.98 (0.67-1.43) 2 1.49 (0.81-2.71) 1.49 (0.71-3.10) 3 1.43 (0.80-2.56) 0.88 (0.35-2.20)	
Olshan 1999 -Population based case control study - Pediatric Oncology Group and Children's Cancer group; participating hospitals in US and Canada -538 cases; <19 May 1 1992 and April 30, 1994, -504 controls	**Parental occupation** **Paternal** Farmers Landscapers Pest Control				OR (95% CI) 0.9 (0.4, 1.8) 2.3 (1.0-5.2) 0.3 (0.0-3.2)	Mother's race, Mother's age, Mother's education, household income in birth year
	Maternal Farmers				2.2 (0.6-8.8)	
Petridou 2000 Nationwide case control study -Greece -153 cases of leukemia; <15 years; 1993-1994 -300 controls -136 cases of Acute Lymphoblastic Leukemia (ALL), 266 controls	**Prenatal exposure to pesticides**	RR (95% CI) Leukemia 3.6 (1.2-10.8) ALL 2.9 (0.9-9.9)				Not reported
Meinert 2000 -Population-based case control study, German Childhood Cancer registry -1184 cases with ALL, 234 with non Hodgkin's, 940 solid tumor; diagnosis between Oct 1992-Sept 1994 or living in West Germany, < 15 years AND diagnosis between 1980- 1994, & living near a nuclear installation -2588 controls	Pesticide use In garden On a farm	OR (95% CI) 1.0 (0.8, 1.2) 1.5 (1.0, 2.2)	OR (95% CI) 0.8 (0.5, 1.2) 0.5 (0.2, 1.4)			
	Household insecticides by parent <1/year 2-5/year 6-10/year >10 1.2.1.1.7.1.1.1 by pest controller	Reference group: < 1/yr 1.0 (0.7, 1.5) 1.0 (0.7, 1.4) 1.3 (0.7, 2.4) 1.8 (1.0, 3.3) 1.3 (0.8, 2.3)	Reference group: < 1/yr 1.3 (0.6, 2.8) 1.3 (0.7, 2.9) 1.5 (0.6, 4.1) 2.8 (1.2, 5.7) 2.6 (1.2, 5.7)			

Study design and Populations	Exposure	Leukemia	Lymphomas	Brain/Central Nervous System Tumors	Other	Variables adjusted for confounding
Pearce 2000 Retrospective cohort -Database of live births, kidney cancers and deaths in Cumbria, Northwest England; death certificates; -Born 1950-1993; 1378 cancer mortalities between ages 1-15, plus 7 non fatal kidney cancers	**Parental occupation in agriculture**				MR (exposed/unexposed) All cancer deaths =0.53 Kidney cancer mortalities=0.08	
Buckley 2000 -Population based case control study; participating hospitals -268 cases, February 1986 -June 1990; <20 years -268 controls	**Household insecticides (mother)** <1/week 1-2 / week most days		(non Hodgkin's) OR (95% CI) 0.98 (0.60-1.58) 2.62 (0.96-7.18) 7.33 (0.84-63.85)			Maternal education and race
	Garden sprays (mother) < 1/month => 1/month		1.82 (0.61-5.45) 1.71 (0.67-4.37)			
	Exterminate around home (mother)		2.98 (1.44-6.16)			
	Occupational pesticides (parent)		1.74 (0.82-3.69)			
Schuz 2001 Medical and Pediatric Oncology -Population based Case Control Study, German Childhood Cancer Registry -466 cases, < 15 years; July 1988 - June 1993 in Lower Saxony and October 1992 - September 1993 in West Germany -2458 controls	**Use of pesticides** In garden On farms			OR (95% CI) 0.94 (0.68-1.29) 0.41 (0.18-0.93)		
	Use of insecticides 1/year >1 /year			1.38 (0.84-2.25) 1.19 (0.81-1.77)		Degree of urbanization and SES, age gender and year of birth

Study design and Populations	Exposure	Leukemia	Lymphomas	Brain/Central Nervous System Tumors	Other	Variables adjusted for confounding
Schuz 2001 Eur J Pediart Population based Case Control Study, German Childhood Cancer Registry -177 cases, < 15 years; July 1988 - June 1993 in Lower Saxony and October 1992 - September 1993 in West Germany -2006 controls	**Child exposure** In garden On farms (and gardens)				OR (95% CI) (Wilm's tumor, renal cancer) 0.80 (0.44-1.47) 0.84 (0.32-2.25)	Socioeconomic status, urbanization, age, gender, year of birth
	In house use of insecticides (more than once a year)				1.27 (0.78-2.08)	
	Maternal occupational exposure to pesticides (ever)				2.52(0.50-12.6)	
	Paternal occupational exposure to pesticides after birth				0.97 (0.39-2.37)	
Daniels 2001 -Population A3based case control study -Pediatric Oncology Group and Children's Cancer group; participating hospitals in US and Canada -538 cases; <19 May 1, 1992 and April 30, 1994, -504 controls	As reported by both parents Extermination Home pesticide Garden pesticide			Neuroblastoma OR (95% CI) Preceonception Ever / Pregnancy / Childhood 1.4 (0.9-2.1) / 1.0 (0.5-2.1) / 1.5 (0.8-2.6) 1.6 (1.0-2.3) / 1.3 (0.8-3.3) / 1.4 (0.9-2.2) 1.7 (0.9-2.1) / 1.3 (0.8-2.0) / 1.8 (1.0-3.1)		Household income and child's age
Feychting 2001 -Prospective cohort study -235,635 children born in 1976, 1977, 1981 and 1982, Swedish Cancer Registry, followed for 15 years -522 cases of childhood cancer: 161 leukemia, 162 nervous system disorders	Paternal occupation agricultural, horticultural, forestry management) before conception	RR (95% CI) 0.90 (0.37-2.19)			RR (95% CI) (Nervous system disorders) 2.36 (1.27-4.39)	Census year, gender, maternal age; SES (only to those born in 1981/1982)

Study design and Populations	Exposure	Leukemia	Lymphomas	Brain/Central Nervous System Tumors	Other	Variables adjusted for confounding
Ma (2002) Population-based case control study - Northern California Childhood Leukemia Study (hospital based) -162 cases from <15, between, 1995-1999; 135 with ALL -162 controls	3 months before pregnancy - 3 years old Professional pest control Insecticides Professional pest control Insecticides	OR (95%) (leukemia) 2.8 (1.4-5.7) 2.6 (1.2-5.4) (ALL) 2.1 (1.1-4.3) 2.2 (1.0-4.6)				Annual household income
Wijngaarden 2002 -Population-based case control study - Children's Cancer Group (US and Canada) - Cases: 154 with astroyctoma and 158 with Primitive Neuroectaldemral tumors (PNET); between 1986 – 1989; diagnosis before 6 years of age -321 controls	Parental occupation to insecticides Father Mother			OR (95%) <u>Astrocytoma</u> <u>PNET</u> 1.5(0.9-2.4) 1.1 (0.7-1.7) 1.9 (1.1-3.3) 1.0 (0.6-1.7)		Maternal age, household income, maternal education
Efrid 2003 -Population based case control Study -San Francisco, Los Angeles, Seattle, USA; Israel, Milan, Italy; Valencia, Spain; Sydney, Australia; Paris, France; Winnipeg, Canada -1218, <20 years, diagnosis between 1976-1994 -2223 controls	Maternal job related exposure to agricultural chemicals			OR (95%) 2.0 (1.2, 3.2)		Age, center, gender

3.7.3 Respiratory Illness

Asthma is the most common chronic disease among children; nine million US children under the age of 18 have been diagnosed. It is a chronic inflammatory disease of the lungs in which the airways are constricted from inflammation and hyper-responsiveness to triggers such as allergens, infection, exercise, changes in weather, and airway irritants. Asthma episodes can range in severity from mild to life threatening and involve shortness of breath, coughing, wheezing, chest pain or tightness, or a combination of these symptoms. Asthma prevalence has increased across all age groups from 1980 through 1995 and has remained relatively stable through 2000. Young males (age 0 to 17 years) have a higher likelihood of experiencing an asthma attack than females in the same age group (Dey et al., 2004).

Epidemiological Studies of Pesticides and Respiratory Illnesses

There is a paucity of epidemiologic studies of pesticide exposure and respiratory illnesses, especially studies focusing on children (see Table 3-37, below). Several cross-sectional studies (those studies that investigate prevalent disease rather than new cases of disease) have investigated home environment conditions and their association with prevalent respiratory problems, and there has been one large cohort study of pesticide applicators. Small increases in wheezing were found for malathion use in Ethiopian homes (Yemaneberhan et al., 1997) and among licensed pesticide applicators (Hoppin et al., 2002). Wheezing among children was also found to be associated with ever exposure to pesticides in Lebanon (Salameh et al., 2003) as well as burning mosquito repellants in Taiwan (Yang et al., 1997). Ever exposure is a standard epidemiologic exposure categorization, as in "ever versus never." In the Lebanese study, other respiratory problems including respiratory disease, asthma, and chronic phlegm were all significantly associated with ever exposure to pesticides. Except for the study of pesticide applicators, all of the studies were cross-sectional with the concomitant issue of establishing temporality between exposure and disease. Cross-sectional studies assess both disease status and exposure at the same time; thus, it is usually not possible to determine if the exposure preceded the disease onset.

Table 3-37. Summary of Epidemiologic Studies of Pesticide Exposure and Respiratory Illnesses

Study design and population	Exposure	Measure of association	Variables adjusted for Confounding
Yemaneberhan 1997 Cross-sectional study; Ethiopia; Ages 0-70+ years; Feb. to Mar. 1996 Urban (N=9844) and rural (N=3032) households	Malathion used in home	OR 95% CI Wheeze 1.17 (0.92-1.50)	Age, sex and urban or rural residence
Yang 1997 Cross-sectional study; Taiwan; 4164 children (6 to 12 years) September to November 1994	Mosquito repellant burning	Respiratory problem OR 95% CI Asthma 1.24 Cough 1.26 Wheezing 1.15 Bronchitis 1.13 Allergic rhinitis 0.83 (0.69-1.01)	Age, gender, parent's education, child's allergies, parental COPD, household crowding, age of home
Muller-Mohnssen 1999 Case report Germany 18-year old woman	Accidental exposure to aerosol insecticide of resmethrin and pyrethrins	Chronic bronchitis and alveolitis developed; asthmatic crisis occurred 2 months after exposure	
Vanderplas 2000 Case report, Belgium, 47-year old man	Occupational exposure to tetramethrin	Asthmatic reactions to pure tetramethrin challenge	
Hoppin 2002 Prospective cohort study Agricultural Health Study – pesticide applicators in Iowa and North Carolina, 1994-1997 20,468 participants (16-88 years of age) 3,838 with "wheeze"	Ever use of: Malathion Permethrin (crops) Permethrin (poultry)	OR 95% CI 1.14 (1.02-1.28) 1.13 (0.95, 1.35) 1.26 (1.06, 1.51)	Age, state, past smoking, current smoking and athma/atopy
Salameh 2003 Cross-sectional study Lebanon Children age 5-16 years March to June 2000 3,291 children returned completed questionnaires	Any pesticide exposure (including living near agricultural fields, use in home, parent in pesticide-related occupation)	Respiratory problem Resp. disease 1.71 Asthma 1.73 Chronic cough 1.04 Chronic phlegm 1.90 (1.26-2.87) Ever wheeze 1.99	Passive smoking, sex, age, weight and BMI, parent's respiratory disease, parent's education, animal raising, and playing with dust

3.7.4 Neurological Problems in Children

Several of the mosquito control pesticides under consideration have a mode of action that affects the nervous systems of the insects. Thus, it is important to examine the epidemiologic literature for evidence of the neurological effects in children. As summarized above, children's nervous systems are particularly vulnerable to toxic insult. However, investigation into pesticide exposure and neurological problems conducted in children or adolescents is sparse and study designs are less than optimal (see Table 3-38, below). As noted by Eskenazi et al. (1999), in a review of organophosphate pesticides and their potential adverse health effects in children, studies of pesticide exposure on children's health have been limited to birth defects, childhood cancer, and acute pesticide poisonings. It is not possible to draw any conclusions about the potential role any of the pesticides under consideration may play in the incidence of childhood neurological problems. The two studies that examined neurological function in relation to pesticide exposure among children or adolescents found better performance among study subjects that were not considered exposed when compared to those who were considered pesticide exposed (Guillette et al., 1998; Rohlman et al., 2001). Specific pesticides that these children may have been exposed to are not used. The case-series of Japanese children and adults with long-term exposure to malathion through helicopter spraying linked reported neuro-opthalmological symptoms with this exposure (Ishikawa et al., 1993). However, no rigorous epidemiologic investigation was conducted so that no conclusions about an association between malathion exposure and this neurological problem can be drawn.

Table 3-38. Summary of Epidemiologic Studies of Pesticide Exposure and Neurological Problems

Study design and population	Exposure	Measure of association	Variables adjusted for Confounding
Ishikawa 1993 Case series Japan 1970 71 cases of Saku disease Children and adults	Helicopter spraying of malathion several times per year for 3-5 years	Reported neuro-opthalmological symptoms: optic neuropathy; retinal degeneration; defective vertical smooth pursuit; myopia; neurologic impairment	

Study design and population	Exposure	Measure of association	Variables adjusted for Confounding
Guillette 1998 Ecologic study Mexico 4 and 5 year old children 28 Valley children 17 Foothill children	Living in the valley – high pesticide use Living in the foothills – low pesticide use	Valley children demonstrated statistically significant decreases in stamina, gross and hand-eye coordination, 30-minute memory and the ability to draw a person	Gender
Rohlman 2001 Comparison study Oregon 1998 Hispanic adolescents aged 13-18 years 102 worked in agriculture 51 not employed in agriculture	(A): Preseason non-agriculture (B): Preseason agriculture (C): Postseason agriculture	Number of tests (out of 17) significantly different from: (B): 6 (C): 3 (A): 6 (C): 8 (A): 3 (B): 8	Bonferroni correction for multiple comparisons

3.8 The Long Island Sound Lobster Research Initiative

In the fall of 1999, lobstermen in western Long Island Sound began to find dead and dying adult lobsters in their traps (Clemetson, 2001). This die-off was coincident with the discovery of WNV in New York City and the application of pesticides to control the disease vector, mosquitoes (*John Fox et. al. vs. Cheminova et. al*. CV 00-5145(TCP) (E.D.N.Y. Feb. 28, 2003)). Although it was noted that lobsters and mosquitoes have some similarities, these may have been overstressed. Mosquitoes and lobsters are both arthropods, but mosquitoes are Diptera (true flies – insects) while lobsters are Decapods, and so they are actually not very closely related evolutionarily.

In response, the Federal government appropriated funds to research the cause of this impact to a substantial fishery. The funding was augmented by New York State and Connecticut, and a research initiative was administered by the New York and Connecticut Sea Grant agencies.

A difficulty was that samples of the water where (and when) the die-offs occurred were not preserved – although, even if they had been collected, analytical means of analyzing for

appropriate concentrations of the mosquito control pesticides in question had not yet been developed. Some other environmental parameters had been collected at the time of the incidents, however, and some post-mortem lobster specimens were preserved for autopsy and other disease investigations.

Researchers determined the concentrations that various pesticides may have deleterious impacts on lobsters (De Guise et al., 2004; McElroy et al., 2004; Horst et al., 2004; Laufer et al. 2003; DeGuise et al., 2005; Walker et al., 2005; Zulkowsky et al., 2005). They researched the potential impacts of temperature (Dove et al., 2003; LoBue and Howell, 2003; Wilson and Waliser, 2003; Dove et al., 2005; Howell et al., 2005), hydrogen sulfide (Cuomo et al., 2003; Cuomo et al., 2005), and certain parasites (Frasca et al., 2004; Gast, 2004; Mullen et al., 2005), diseases (Chistoserdov et al., 2004; Dove et al., 2003; Chistoserdov et al., 2005; Smolowitz et al., 2005; Laufer et al., 2005), and combinations of the above (Robohm et al., 2005; Draxler et al., 2005) on lobsters in Long Island Sound. Two independent modeling exercises were conducted to determine the maximum water column concentrations of the mosquito control pesticides, using unrealistic input terms (in that degradation, transport, and even application terms were maximized so as to constitute a worst case scenario), to determine if there was a potential for the pesticides to impact the lobsters (Landeck Miller et al., 2005; Wilson et al., 2005).

The consensus of those in attendance at the final symposium of the research effort was that other factors other than pesticides could have (and probably did) account for the lobster die-off. The combination of warmer than normal water, high densities of lobsters, increased levels of hydrogen sulfide, decreased oxygen concentrations, and probable presence of lobster pathogens collectively caused the mass mortality. Modeling showed that in some shallow waters and over limited areas of the Sound, it might have been possible that the concentrations of pesticides met minimal criteria for impacts to the lobsters. However, these results depended on what were acknowledged to be unrealistic inputs of pesticides, and it was clear from the modeling that concentrations of concern could not have been found over the large areas of Long Island Sound where impacts to the lobsters were seen. The combination of modeling results and laboratory toxicity studies suggested that, clearly, pesticides alone did not cause the die-off The potential for synergy between the environmental conditions and some pesticides (although at concentrations below those of concern in laboratory settings), or for synergy between different

pesticides, means that it could not be absolutely, unequivocally stated that pesticides did not play a role in the die-off. The stated consensus of the research community was that the mosquito control pesticides, as used in 1999, did not play the dominant role in causing the die-off, and that most likely it was an unfortunate confluence of environmental factors that led to a mass mortality from disease (Rather, 2004; Haberstroh, 2004; Pearce and Balcom, 2005).

Section 3 References

APHIS. 2003. *Economic Impact of West Nile Virus on the Colorado and Nebraska Equine Industries:* 2002. Animal and Plant Health Inspection Service, US Department of Agriculture. 4 pp.

ASPCA. 2005. *Keep Your Pets Safe While Maintaining Your Lawn and Garden.* American Society for the Prevention of Cruelty to Animals, Press Release. June 1.

ASPCA. 2004. *ASPCA Animal Poison Control Center Warns Pet Owners: Summer is the Deadliest Time of Year for Companion Animals.* American Society for the Prevention of Cruelty to Animals, Press Release. May 3.

Abd el-Rahim IH, U Abd el-Hakim, and M Hussein. 1999. An epizootic of Rift Valley fever in Egypt in 1997. *Reviews of Science and Technology* 18:741-748.

Alan, T., and A. Mandalakas. Undated. *West Nile Virus Seroprevalence Survey Cuyahoga County*. PowerPoint presentation. www.ccbh.net/pdf/OMCA2003.pdf

American Horse Council. Undated. *West Nile Virus*. www.horsecouncil.org/West%20Nile%20Virus.doc

Anderson, JF, TG Andreadis, CR Vossbrinck, S. Tirrell, EM Wakem, RA French, AE Garmendia, and HJ Van Kruiningen. 1999. Isolation of West Nile virus from mosquitoes, crows, and a Coopers hawk in Connecticut. *Science* 286:2331-2333.

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.

Andreadis, TG, PM Capotosto, RE Shope, and SJ Tirrell. 1994. Mosquito and arbovirus surveillance in Connecticut, 1991-1992. *Journal of the American Mosquito Control Association* 10:556-564.

Apperson, CS, HK Hassan, BA Harrison, HM Savage, SE Aspen, A. Farajollahi, W. Crans, TJ Daniels, RC Falco, M. Benedict, M. Anderson, L. McMillan, and TR Unnasch. 2004. Host feeding patterns of established and potential mosquito vectors of West Nile virus in the eastern United States. *Vector-borne and Zoonotic Diseases* 4(1):71-82.

Arnott, JJ and JD Edman. 1978. Mosquito vectors of dog heartworm in Western Massachusetts. *Mosquito News* 38:222-230.

Audubon. 2005. West Nile virus. www,audubon.org/bird/wnv/pdf/effects_on_wildlife.pdf (07/05/05).

Austin, RJ, TL Whiting, RA Anderson, and MA Drebot. 2004. An outbreak of West Nile Virus-associated disease in domestic geese (*Anser anser domesticus*) upon initial introduction

to a geographic region, with evidence of bird to bird transmission. *Canadian Veterinary Journal* 45(2):117–123.

Balkhy HH, and JR Schreiber. 2000. Severe La Crosse encephalitis with significant neurologic sequelae. *Pediatric Infectious Disease Journal* 19:77-80.

Barr, DB, R. Bravo, G. Weerasekera, LM Caltabiano, RD Whitehead, Jr., AO Olsson, SP Caudill, SE Schober, JL Pirkle, EJ Sampson, RJ Jackson, and LL Needham. 2004. Concentrations of dialkyl phosphate metabolites of organophosphorous pesticides in the US population. *Environmental Health Perspectives* 112:186-200.

Bartlett, CM and RC Anderson. 1981. Occult filariasis in crows (*Corvus brachyrhynchos* Brehm) infected with *Splendidofilaria caperata* Hibler (Nematoda: Filariodea). *Journal of Wildlife Diseases* 17(1):69-77.

Beadle, LD. 1952. Eastern equine encephalitis in the United States. *Mosquito News* 12:102-107.

Beaudette, FR, and JJ Black. 1948. Equine encephalomyelitis in New Jersey pheasants in 1945 and 1946. *Journal of the American Veterinary Medical Association* 112:140-147.

Beaudette, FR, and CB Hudson. 1945. Additional outbreaks of equine encephalomyelitis in New Jersey pheasants. *Journal of the American Veterinary Medical. Association* 107:384-386.

Beaver, PC and TC Orihel. 1965. Human infection with filariae of animals in the United States. *American Journal Tropical Medicine and Hygiene* 14:1010-1029.

Bemrick, WJ, and HA Sandholm. 1966. *Aedes vexans* and other potential mosquito vectors of *Dirofilaria immitis* in Minnesota. *Journal of. Parasitology* 52:762-767.

Bernard, KA, JG Maffai, SA Jones, EB Kauffman, G. Ebel, AP Dupuis 2nd, KA Ngo, DC Nicholas, DM Young, PY Shi, VL Kulasekera, M. Eidson, DJ White, WB Stone, and LD Kramer. 2001. West Nile virus infection in birds and mosquitoes, New York State, 2000. *Emerging Infectious Diseases* 7(4):679-685.

Bertelsen, MF, R-A Ølbeg, GJ Crawshaw, A. Debernardo, LR Lindsay, M. Drebot, and IK Barker. 2004. West Nile virus infection in the eastern loggerhead shrike (*Lanius ludovicianus* migrans): pathology, epidemiology, and immunization. *Journal of Wildlife Diseases* 40(3):538-542.

Bin, H., Z. Grossman, S. Pokamunski, M. Malkinson, L. Weiss, P. Duvdevani, C. Banet, Y. Weisman, E. Annis, D. Gandaku, V. Yahalom, M. Hindyieh, L. Shulman, and E. Mendelson. 2001. West Nile fever in Israel in 1999—2000: from geese to humans. *Annals of the NY Academy of Science* 951:127-142.

Bonter, DN, and WM Hochachka. 2003a. Combined data of project Feederwatch and the Christmas Bird Count indicate declines of chickadees and corvids: possible impacts of West Nile virus. *American Birds* (103rd Count): 22-25.

Bonter, D., and WM Hochachka. 2003b. Taking count in the wake of West Nile virus. *Birdscope* 17(2):13-15.

Boromisa RD, and PR Grimstad. 1987. Seroconversion rates to Jamestown Canyon virus among six populations of white-tailed deer (*Odocoileus virginianus*) in Indiana. *Journal of Wildlife Diseases* 23:23-33.

Brummer-Korvenkontio, M., O. Vapalahti, P. Kuusisto, P. Saikku, T. manni, P. Koskela, T. Nygren, H. Brummer-Korvenkontio, and A. Vaheri. 2002. Epidemiology of Sindbis virus infections in Finland 1981-86: possible factors explaining a peculiar disease pattern. *Epidemiology and Infection* 129(2):335-345.

Buckley, JD, AT Meadows, ME Kadin, MM Le Beau, S. Siegel, and LL Robison. 2000. Pesticides exposures in children with non-Hodgkin lymphoma. *Cancer* 89:2315-2321.

Budiansky, S. 2002. Creatures of our own making. *Science* 298:80-86.

Burbutis, PP, and DM Jobbins. 1957. *Cs. melanura* and eastern equine encephalomyelitis in New Jersey. *Proceedings of the New Jersey Mosquito Extermination Association* 44:68-78.

Bunning, ML, RA Bowen, CB Cropp, KG Sullivan, BS Davis, N. Komar, MS Godsey, D. Baker, DL Hettler, DA Holmes, BJ Biggerstaff, CJ Mitchell. 2002. Experimental infection of horses with West Nile Virus. *Emerging Infectious Diseases* 8(4):380-386.

Buxton, H.T. and Modica, E. 1992. Patterns and rates of ground-water flow on Long Island, New York. *Ground Water* 30(6):857-866.

CA-CE. 2004. *Long Island Mosquitoes*. Task 3, Book 1, Suffolk County Vector Control and Wetlands Management Long-Term Plan and Generic Environmental Impact Statement. Suffolk County Department of Health Services, Riverhead, NY. 58 pp.

CA-SCDHS. 2005. *Human Health and Domestic Animal Toxicity.* Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 3 (Literature Search), Book 6 Part 1. Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. Paged in sections.

CDC. 2005. www.cdc.gov/ncidod/dvbid/westnile/birdspecies.htm

CDC. 2004. *West Nile Virus*. www.cdc.gov/ncidod/dvbid/westnile/index.htm

CDC. 2003a. *Yellow Fever*. www.cdc.gov/travel/diseases/yellowfever.htm>.

CDC. 2003b. *CDC Dengue Fever Home Page*. www.cdc.gov/ncidod/dvbid/Dengue/

CDC. 2003c. *Fact Sheet: Lymphatic Filariasis*. www.cdc.gov/ncidod/dpd/parasites/lymphaticfilariasis/factsht_lymphatic_filar.htm

CDC. 2003d. *Arboviral Encephalitides*. www.cdc.gov/ncidod/dvbid/arbor/

CDC. 2003e. *Epidemic/Epizootic West Nile Virus in the United States: Guidelines for Surveillance, Prevention, and Control*. 3rd Revision. Centers for Disease Control and Prevention, Fort Collins, CO. 75 pp.

CDC. 2003f. *Second National Report on Human Exposure to Environmental Chemicals*. Centers for Disease Control and Prevention. www.cdc.gov/exposurereport/.

CDC. 2000. Malaria: General Information. http://www.cdc.gov/travel/malinfo.htm.

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

Caffrey, C., and CC Peterson. 2003. West Nile virus may not be a conservation issue in the northeastern United States. *American Birds (103rd Count)*:14-21.

Caffrey, C., SCR Smith, and T. Weston. 2005. West Nile Virus devastates an American crow population. *The Condor* 107:128-132

Caffrey, C., TJ Weston, and SCR Smith. 2003. High mortality among marked crows subsequent to the arrival of West Nile virus. *Wildlife Society Bulletin* 31:870-872.

Calisher, CH. 1994. Medically important arboviruses of the United States and Canada. *Clinical Microbiological Reviews* 7(1):89-116.

Campbell, GL, AA Marfin, RS Lanciotti, and DJ Gubler. 2002. West Nile virus. *The Lancet Infectious Diseases* 2:519-529.

Cantile, C., F. DelPiero, G. Di Guardo, and M. Arispici. 2001. Pathologic and immunohistochemical findings in naturally occurring West Nile virus infection in horses. *Veternary Pathology* 38:414-421.

Cashin Associates. 2005a. *Eastern Equine Encephalitis and Salt Marsh Mosquitoes*. Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 4 (Current Operations). Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. 17 pp.

Cashin Associates. 2005b. *Human Health Risks Associated with Mosquito-borne Pathogens in Suffolk County, NY.* Suffolk County Vector Control and Wetlands Management Long-Term Plan and Environmental Impact Statement, Task 3 (Literature Search), Book 2 Part

2. Suffolk County Department of Health Services, Yaphank, NY. www.suffolkmosquitocontrolplan.org. 59 pp.

Cashin Associates. 2005c. *Deposition Rate 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. 4 pp.

Cashin Associates. 2005d. *Air Dispersion Modeling Support for the Impact Assessment: 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. 52 pp.

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

Cashin Associates. 2002. Carmans River Environmental Assessment. Suffolk County Department of Health Services, Hauppauge, NY. 3 vols.

Castillo-Olivares, J., and J. Wood. 2004. West Nile virus in horses. *Veternarian Research* 35:467-483.

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

Chamberlain, RW. 1958. Vector relationships of the arthropod-borne encephalitides in North America. *Annals of the New York Academy of Science* 70:312-319.

Chamberlain, RW, RK Sikes, DB Nelson, and WD Sudia. 1954. Studies on the North American arthropod-borne encephalitides. VI. Quantitative determinations of virus-vector relationships. *American Journal of Hygiene* 60(3):278-285.

Chamberlain, RW, WD Sudia, PP Burbutis, and MD Bogue. 1958. Recent isolations of arthropod-borne virus from mosquitoes in the eastern United States. *Mosquito News* 18(4):305-308.

Chistoserdov, A., SL Gubbala, R. Smolowitz, A. Hsu, and F. Mirasol. 2004. Bacterial assemblages involved in the development and progression of shell disease in the American lobster, *Homarus americanus.* Abstract of presentation at May 2004 meeting of Long Island Sound Lobster Research Initiative.

Chisteoserdov, AY, R. Smolowitz, F. Mirasol, and A. Hsu. 2005. Culture-dependent characterization of the microbial community associated with epizootic shell disease

lesions in American lobster, *Homarus americanus*. *Journa of Shellfish Research* 24(3):741-747.

Christensen, BM and WN Andrews. 1976. Natural infection of *Aedes trivittatus* with *Dirofilaria immitis* in central Iowa. *Journal of. Parasitology* 62:276-280.

Chu, M., W. Stone, KJ McGowan, AA Dhondt, WM Hockachka, and JE Therrien. 2003. West Nile File. *Birdscape* 17(1):10-11.

Chvala, S., J. Kolodziejek, N. Nowotny, and H. Weissenboeck. 2004. Pathology and viral distribution in fatal Usutu virus infections of birds from the 2001 and 2002 outbreaks in Austria. *Journal of Comparative Pathology* 131:176-185.

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

Clemetson, A. 2001. *Sea Grant's Long Island Sound Lobster Initiative: Sea Grant Program Response to Long Island Sound Lobster Mortality and Shell Disease, A Status Report*. New York Sea Grant Fact Sheet. New York Sea Grant Extension Program, Riverhead, NY.

Cohen, D., and O. Sussman. 1957. Equine encephalomyelitis in New Jersey. *Proceedings of the New Jersey Mosquito Extermination Association* 44:77-91.

Cornell. 2005. Center for the Environment, Environmental Risk Analysis Program WNV-Archive 2/11/05.

Cornell. 2004. *West Nile Virus, Transmission, Infections, and Symptoms.* Cornell University Department of Communication and Center for the Environment, Environmental Risk Analysis Program. environmentalrisk.cornell.edu/WNV/Summary2.php

Cornell Cooperative Extension. 2001. *Pesticide Usage Report for Agricultural Crops in Suffolk County 1975-2000.* Cornell Cooperative Extension of Suffolk County, Riverhead, New York.

Craigmill, A. 1988. Veterinary Toxicology Notes: Hazards of new rodenticides to pets. *Cooperative Extension University of California Environmental Toxicology Newsletter* 8(2).

Crans, WJ. 1977. The status of *Aedes sollicitans* as an epidemic vector of eastern equine encephalitis in New Jersey. *Mosquito News* 37:85-89.

Crans, WJ, DC Caccamise, and JR McNelly. 1994. Eastern equine encephalomyelitis virus in relation to the avian community of a coastal cedar swamp. *Journl of Medical Entomology* 31(5):711-728.

Crans, W. and M. Feldlaufer. 1973. The natural vectors of dog heartworm in a coastal area of New Jersey. *Proceedings of the Northeastern Mosquito Control Association* 19:23-26.

Crans, WJ, LJ McCuiston, and TL Schulze. 1986a. Evidence incriminating Coquillettidia perturbans (Diptera: Culicidae) as an epizootic vector of eastern equine encephalitis. II. Ecological investigations following an inland epizootic in New Jersey. *Bulletin of the Society of Vector Ecologists* 11:185-190.

Crans, WJ, J. McNelly, TL Schulze, and A. Main. 1986b. Isolation of eastern equine encephalitis virus from Ae. sollicitans during an epizootic in southern New Jersey. *Journal of the American Mosquito Control Association* 2(1):68-72.

Cronin, TM, BJ Szabo, TA Ager, JE Hazel, and JP Owens. 1981. Quarternary climates and sea levels of the U.s. Atlantic coastal plain. *Science* 211:233-240.

Cuomo, C., R. Valente, and D. Dogru. Seasonal variation in sediment and bottom water chemistry of western Long Island Sound: implications for lobster mortality. *Journal of Shellfish Research* 24(3):805-814.

Cuomo, CR, D. Valente, and D. Dogru. 2003. Monitoring of bottom water and sediment conditions at critical stations in Western Long Island Sound. pp. 6-8. In: *Third Long Island Sound Lobster Health Symposium*. Long Island Sound Lobster Research Initiative and Connecticut Department of Environmental Protection Long Island Sound Research Fund, Bridgeport, CT. 71 pp.

Dalrymple, JM, OP Young, BF Eldridge, and PK Russell. 1972. Ecology of arboviruses in a Maryland freshwater swamp. III. Vertebrate Hosts. *American Journal of Epidemiology* 96:129-140.

Daniels, JL, AF Olshan, K. Teschke, I. Hertz-Picciotto, DA Savitz, J. Blatt, ML Bondy, JP Neglia, BH Pollock, SL Cohn, AT Look, RC Seeger, and RP Castleberry. 2001. Residential pesticide exposure and neuroblastoma. *Epidemiology* 12:20-27.

DeFoliart, GR, DM Watts, and PR Grimstad. 1986. Changing patterns in mosquito-borne arboviruses. *Journal of the American Mosquito Control Association* 2(4):437-455.

DeGuise, S., J. Marratea, ES Chang, and C. Perkins. 2005. Resmethrin immunotoxicity and endocrine disrupting effects in the American lobster (*Homarus americanus*) upon experimental exposure. *Journal of Shellfish Research* 24(3):781-786.

De Guise, S., J. Maratea, and C. Perkins. 2004. Pesticide immunotoxicity in the American lobster (*Homarus americanus*) upon experimental exposure. Abstract of presentation at May 2004 meeting of Long Island Sound Lobster Research Initiative.

Dey, AN, JS Schiller, and DA Tai. 2004. Summary health statistics for US children: National Health Interview Survey, 2002. *Vital Health Statistics* 10.

Dillon, WP, and RN Oldale. 1978. Late Quarternary sea-level curve: reinterpretation based on glaciotectonic influence. *Geology* 6:56-60.

Dolder, L. 2003. Metaldehyde toxicosis. *Veterinary Medicine*, March.

Dove, AD, B. Allem, JP Powers, and MS Sokolowski. 2005. A prolonged thermal stress experiment on the American lobster, *Homarus americanus*. *Journal of Shellfish Research* 24(3):761-765.

Dove, A., C. LoBue, and P. Bowser. 2003. Calcinosis in LIS lobsters during summer 2002. pp.65-66. In: *Third Long Island Sound Lobster Health Symposium*. Long Island Sound Lobster Research Initiative and Connecticut Department of Environmental Protection Long Island Sound Research Fund, Bridgeport, CT. 71 pp.

Draxler, AFJ, RA Robohm, D. Wieczorek, D. Kapareiko, and S. Pitchford. 2005. Effect of habitat biogeochemicals on survival of lobsters (*Homarus americanus*). *Journal of Shellfish Research* 24(3):821-824.

Dunn, AM, C. Burns, and B. Sattler. Environmental health of children. *Journal of Pediatric Health Care* 17:223-231.

Dupuis, AP, II, PP Marra, and LD Kramer. 2003. Serologic evidence of West Nile virus transmission, Jamaica, West Indies. *Emerging Infectious Diseases* 9(7): 860.863.

EWG. Undated. *Shoppers Guide to Pesticides in Produce.* Environmental Working Group www.foodnews.org/reportcard.php

EXTOXNET. 1998. *Questions About Pesticide Exposure.* Extension Toxicology Network FAQs.

EXTOXNET. 1996. *Pesticide Information Profiles, Metaldehyde.* Extension Toxicology Network.

Eckhardt, DA, and PE Stackelberg. 1995. Relation of ground-water quality to land use on Long Island, New York. *Ground Water* 33(6):1019-1033.

Edman, JD, LA Webber, and HW Kale. 1972. Host-feeding patterns of Florida mosquitoes. II. *Culiseta. Journal of Medical Entomology* 9(5):429-434.

Efird, JT, EA Holly, S. Preston-Martin, BA Mueller, F. Lubin, G. Fillipini, R. Peris-Bonet, M. McCredie, S. Cordier, A. Arslan, and PM Bracci. 2003. Farm-related exposures and childhood brain tumours in seven countries: results from the SEARCH International Brain Tumour Study. *Pediatric and Perinatal Epidemiology* 17:201-211.

Eidson, M., N. Komar, F. Sorhage, R. Nelson, T. Talbot, F. Mostashari, and R. McLean. 2001. Crow deaths as a sentinel surveillance system for West Nile virus in the northeastern United States, 1999. *Emerging Infectious Diseases* 7(4): 615-620.

Eidson, M., J. Miller, L. Kramer, B. Cherry, Y. Hagiwara, and the West Nile virus Bird Mortality Analysis Group. 2001. Dead crow densities and human cases of West Nile Virus, New York State, 2000. *Emerging Infectious Diseases* 7(4):662-664.

Eldridge, BF, and JD Edman. 2000. *Medical Entomology, a Textbook on Public Health and Veterinary Problems Caused by Arthropods*. Kluwer Academic Publishers, Dordrecht, Netherlands. 659 pp.

Elliott, SJ, M. Loeb, J. Eyles, and D. Harrington. 2003. *Results of a West Nile Seroprevalence Survey, South Oakville, Ontario, 2003*. McMaster Institute of Environmental Health, McMaster University, Hamilton, Ontario. 37 pp.

Ernek, E., O. Kozuch, J. Nosek, J. Teplan, and C. Folk. 1977. Arboviruses in birds captured in Slovakia. J. *Hyg. Epidemiol. Microbiol. Immunol*. 21(3):353-359.

Erwin PC, TF Jones, RR Gerhardt, SK Halford, AB Smith, LE Patterson, KL Gottfried, KL Burkhalter, RS Nasci, and W Schaffner. 2002. La Crosse encephalitis in Eastern Tennessee: clinical, environmental, and entomological characteristics from a blinded cohort study. *American Journal of Epidemiology* 155:1060-1065

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..

Estrada-Franco, JG, R. Navarro-Lopez, DW Beasely, L. Coffey, A-S Carrara, A. Travassos da Rosa, T. Clements, E. Wang, GV Ludwig, AC Cortes, PP Ramirez, RB Tesh, ADT Barrett, and SC Weaver. 2003. West Nile virus in Mexico: evidence of widespread circulation since July 2002. *Emerging Infectious Diseases* 9(12):1604-1607.

FDA. 2004. *Food and Drug Administration Pesticide Program Residue Monitoring 2002.* Food and Drug Administration, Washington, DC.

Feingold, BF, E. Benjamini and D. Michaeli. 1968. The allergic responses to insect bites. *Annual Review of Entomology* 13:137-158.

Feychting, M., N. Plato, G. Nise, and A. Ahlbom. 2001. Paternal occupational exposures and childhood cancer. *Environmental Health Perspectives* 107(Supplement)3:409-419.

Focus Technologies. Undated. *Flavivirus (West Nile) ELISA IgG*. Data Sheet. Focus Technologies, Cypress, CA. 4 pp.

Fothergill, LD, M. Holden, and RWG. Wyckoff. 1938. Isolation from human beings of eastern equine encephalomyelitis virus. *Journal of the American Medical Association* 111:1768.

Frasca S., KR Nevis, and TE Mullen. 2004. Development of Polymerase Chain Reaction and in situ hybridization-based tests for the specific detection of paramoeba associated with epizootic lobster mortality by determination of the molecular systematics of the genus Paramoeba – Summary of Results. Abstract of presentation at May 2004 meeting of Long Island Sound Lobster Research Initiative.

GAELF. 2004. *The Global Alliance to Eliminate Lymphatic Filariasis*. www.filariasis.org/index.pl.

Gancz, AY, IK Barker, R. Lindsay, A. Dibernardo, K. McKeever, and B. Hunter. 2004. West Nile virus outbreak in North American owls, Ontario, 2002. *Emerging Infectious Diseases* 10(12):167

Gast, R. 2004. Oligonucleotide-based detection of pathogenic Neoparamoeba species. Abstract of presentation at May 2004 meeting of Long Island Sound Lobster Research Initiative.

Gerdes, GH. 2002. Rift valley fever. *Veterinary Clinics of North American Food Animal Practices* 18:549-555.

Gerhardt RR, KL Gottfried, CS Apperson, BS Davis, PC Erwin, AB Smith, NA Panella, EE Powell, and RS Nasci. 2001. First isolation of La Crosse virus from naturally infected Aedes albopictus. *Emerging Infectious Diseases* 7:807-811.

Gilliom, RJ, JE Barbash, CG Crawford, PA Hamilton, JD Martin, N. Nakagaki, LH Nowell, JC Scott, PE Stackelberg, GP Thelin, and DM Wolock. *Pesticides in the Nation's Streams and Ground Water, 1992-2001*. US Geological Survey Circular 1291, Reston, VA. 172 pp.

Glaser, LC, MV Wegner, JP Davis, ML Bunning, AA Marfin, GL Campbell, B. Bernard, SW Lenhart, and NJ Sotir. 2003. West Nile Virus infection among turkey breeder farm workers – Wisconsin, 2002. *Morbidity and Mortality Weekly Report* 52(42):1017-1019.

Gobler, CJ, DJ Lonsdale, and GL Boyer. 2005. A review of the causes, impacts, and potential management of harmful brown tide blooms caused by *Aureococcus anophagefferens* (Hargraves et Sieburth). *Estuaries* 28(5):726-749.

Goldfield, M., and O. Sussman. 1968. The 1959 outbreak of eastern encephalitis in New Jersey. I. Introduction and description of outbreak. *American Journal of Epidemiology* 87(1):1-10.

Grady, GF, HK Maxfield, SW Hildreth, RJ Timperi, Jr., RF Gilfilean, BJ Rosenau, DB Francy, CH Callisher, LC Marcus, and MA Madoff. 1978. Eastern equine encephalitis in Massachusetts, 1957-1976. *American Journal of Epidemiology* 107:170-178.

Graham, J. 2003. Animals sick, dying in greater numbers from West Nile Virus. *Chicago Tribune*. September 15, 2003.

Graham, DG, and BL Harper. 2004. The potential threat of mosquito-borne disease in Suffolk County. *The Suffolk County Medical Society-Suffolk County Academy of Medicine*, July. Public Health Page.

Gratz, NG. 1999. Emerging and resurging vector-borne diseases. *Annual Review of Entomology* 44:51-75.

Grimstad PR, CH Calisher, RN Harroff, and BB Wentworth. 1986. Jamestown Canyon virus (California serogroup) is the etiologic agent of widespread infection in Michigan humans. *American Journal of Tropical Medicine and Hygiene* 35:376-386.

Grimstad PR, DG Williams, and SM Schmitt. 1987. Infection of white-tailed deer (*Odocoileus virginianus*) in Michigan with Jamestown Canyon virus (California serogroup) and the importance of maternal antibody in viral maintenance. *Journal of Wildlife Disease* 23:12-22.

Guillette, EA, MM Meza, MG Aguilar, AD Soto, and IE Garcia. 1998. An anthropological approach to the evaluation of preschool children exposed to pesticides in Mexico. *Environmental Health Perspectives* 106:347-353.

Haberstroh, J. 2004. Lobsters die in "perfect storm." *Newsday*, October 6. pp. A4, A40.

Hadler, J. R. Nelson, T. McCarthy, T. Andreadis, MJ Lis, R. French, W. Beckwith, D. Mayo, G. Archambault, and M. Cartter. 2001. West Nile virus surveillance in Connecticut in 2000: an intense epizootic without high risk for severe human disease. *Emerging Infectious Diseases* 7(4):636-642.

Harwood, RF, and MT James. 1979. *Entomology in Human and Animal Health.* 7th Ed. Macmillan Publishing Co., New York, NY. 547 pp.

Hawking, F and M. Worms. 1961. Transmission of filaroid nemaodes. *Annual Review of Entomology* 6:413-432.

Hayes, RO, LD Beadle, AD Hess, O. Sussman, and MJ Bonese. 1962. Entomological aspects of the 1959 outbreak of eastern encephalitis in New Jersey. *American Journal of Tropical Medicine and Hygiene* 11:115-121.

Headlee, TJ. 1995. *The Mosquitoes of New Jersey and their Control*. Rutgers University Press, New Brunswick, NJ. 326 pp.

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, AN Walker, PB Bush, T. Wilson, ES Chang, T. Miller, and J. Puritz. 2004. Acute effects of Methoprene on the lobster, *Homarus americanus*. Abstract of presentation at May 2004 meeting of Long Island Sound Lobster Research Initiative.

Howard, JJ, MA Grayson, DJ White and CD Morris. 1994. Eastern equine encephalitis in New York State. *Journal of the Florida Mosquito Control Association* 65:1-6.

Howell, P., J. Benway, C. Giannini, K. McKown, R. Burgess, and J. Hayden. 2005. Long-term population trends in American lobster (*Homarus americanus*) and their relation to temperature in Long Island Sound. *Journal of Shellfish Research* 24(3):849-857.

Huhn, GD, JJ Sejvar, SP Montgomery, and MS Dworkin. 2003. West Nile Virus in the United States: an update on an emerging infectious disease. *American Family Physician* 68(4):653-660.

Hurlbut, H., and R. Taylor. 1955. Indigenous wild birds of the Nile delta as potential West Nile virus circulating reservoirs. *Am J Trop Med Hyg*. 4:872–888.

Insight Pharmaceuticals. 2003. *Nix Complete Lice Removal Kit Product Description*. www.insightpharma.com/nix-kit.aspx.

Intermill, R.W. 1973. Development of *Dirofilaria immitis* in *Aedes triseriatus Say*. *Mosquito News* 33:176-181.

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

Jemal, A., RC Tiwari, T. Murray, A. Ghafoor, A. Samuels, E. Ward, EJ Feuer, and MJ Thun. 2004. Cancer statistics, 2004. *CA Cancer J Clin* 54:8-29.

Johansen CA, DJ Nisbet, P Zborowski, AF van den Hurk, SA Ritchie, and JS Mackenzie. 2003. Flavivirus isolations from mosquitoes collected from western Cape York Peninsula, Australia, 1999-2000. *Journal of American Mosquito Control Association* 19:392-396.

Jordan Communications. 2001. *Spectacular Blue Raiders are Regular Feeder Visitors*. realindy.com/bluejays.htm

Kaatsch, P., U. Kaletsch, R. meinert, A. Meisner, M. Hoisl, J. Schuz, and J. Michaelis. 1998. German case control study on childhood leukaemia – basic considerations, methodology and summary of the results. *Klinic Paediatric* 210:185-191.

Kandle, RP. 1960. Eastern encephalitis in New Jersey. *Proceedings of the New Jersey Mosquito Extermination Association* 47:11-15.

Kavanagh, WK. 1980. *Vanishing Tidelands: Land Use and the Law in Suffolk County, NY 1650-1979*. New York Sea Grant (NYSG-RS-80-28). 265 pp.

Keller, M., MT Long, K. Conley, R. Francis-Floyd, R Isaza. 2004. A serosurvey of marine mammals for West Nile Virus and the development of a competitive inhibition ELISA (Enzym-linked immunosorbent assay). *Presentation abstract from Phi Zeta Research Emphasis Day*. July 14, 2004. www.vetmed.ufl.edu/phizeta/phizetasched04.htm

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.

Kitron, U., J. Swanson, M. Crandell, PJ Sullivan, J. Anderson, R. Garro, LD Haramis, and PR Grimstad. 1998. Introduction of Aedes albopictus into a La Crosse virus--enzootic site in Illinois. *Emerging Infectious Diseases* 4:627-30.

Kolpin, D., J. Barbash, and R. Gilliom. 2000. Pesticides in ground water of the United States, 1992-1996. *Ground Water* 38(6):858-863.

Komar, N. 2000. House sparrows as reservoirs of West Nile. Paper 226, *American Journal of Tropical Medicine and Hygiene Conference.* November 1.

Komar, N., B. Davis, M. Bunning, and D. Hettler. 2000. Experimental infection of wild birds with West Nile virus (New York 1999 strain). *American Journal of Tropical Medicine and Hygiene* 62: 229-230

Komar, N., S. Langevin, N. Nemeth, E. Edwards, D. Hettler, B. Davis, R. Bowen, and M. Bunning. 2003a. Experimental infection of North American birds with the New York 1999 strain of West Nile virus. *Emerging Infectious Diseases* 9:311-322.

Komar, N., NA Panella, and E. Boyce. 2001. Exposure of domestic mammals to West Nile Virus during an outbreak of human encephalitis, New York City, 1999. *Emerging Infectious Diseases* 7(4):736-738.

Komar, O., MB Robbins, K. Klenk, B. Blitvich, NL Marlenee, KL Burkhalter, DJ Gubler, G. Gonzálvez, CJ Peña, AT Peterson, and N. Komar. 2003b. West Nile virus transmission in resident birds, Dominican Republic. *Emerging Infectious Diseases* 9(10):1299-1302.

Koppelman, LS. 1978. *The Long Island Comprehensive Waste Treatment Management Plan*. Nassau-Suffolk Regional Planning Boiard, Hauppauge, NY. 2 vols.

Koutrakis, P., and SLK Briggs. 1992. Source apportionment of indoor aerosols in Suffolk and Onondaga Counties, New York. *Environmental Science and Technology* 26(3):521-527.

Kramer, LD, and KA Bernard, 2001. West Nile virus infection in birds and mammals. *Annals of the New York Academy of Sciences* 951:84-93.

Kurkela, S., T. Manni, A. Vaheri, and O. Vapaplahti. 2004. Causative agent of Pogogosta disease isolated from blood and skin lesions. *Emerging Infectious Diseases* 10(5):889-894.

LIPA. 2005. *2004 Long Island Population Survey*. Long Island Power Authority, Uniondale, NY. 38 pp. + appendices.

LISS. 1994. *The Comprehensive Conservation and Management Plan*. US Environmental Protection Agency, Long Island Sound Office. Stamford, CT. 168 pp.

Laine, M., R. Vainionpaa, J. Uksila, J. Oksi, M. Nissila, K. Kaarela, R. Luukkainen, and A. Toivanen. 2003. Prevalence of Sindbis-related (Pogosta) virus infections in patients with arthritis. *Clinical and Experimental Rheumatology* 21(2):213-216.

Lanciotti, RS, JT Roehrig, V. Deubel, J. Smith, M. Parker, K. Steele, B. Crise, KE Volpe, MB Crabtree, JH Scherret, RA Hall, JS MacKenzie, CB Cropp, E. Panigrahy, E. Ostlund, B. Schmitt, M. Malkinson, C. Banet, J. Weissman, N. Komar, HM Savage, W. Stone, T. McNamara, and D.J. Gubler. 1999. Origin of the West Nile virus responsible for an outbreak of encephalitis in the northeastern United States. *Science* 286: 2333-2337.

Landeck Miller, RE, JR Wands, KN Chytalo, and RA D'Amico. 2005. Application of water quality modeling technology to investigate the mortality of lobsters (*Homarus americanus*) in western Long Island Sound during the summer of 1999. *Journal of Shellfish Research* 24(3):859-864.

Langevin, SA, M. Bunning, B. Davis, and N. Komar. 2001. Experimental infection of chickens as candidate sentinels for West Nile Virus. *Emerging Infectious Diseases* 7(4): 726-729.

Laufer, H., W. Biggers, M. Johnson, N. Demir, and J. Bagshaw. 2003. Hormonal responses of lobsters to stresses, an interim report. pp. 32-39. In: *Third Long Island Sound Lobster Health Symposium*. Long Island Sound Lobster Research Initiative and Connecticut Department of Environmental Protection Long Island Sound Research Fund, Bridgeport, CT. 71 pp.

Laufer, H., N. Demire, and WJ Biggers. 2005. Response of the American lobster to the stress of shell disease. *Journal of Shellfish Research* 24(3):757-760.

Lefrancois, T., JB Bradley, J. Pradel, S. Molia, N. Vachiéry, G. Pallavicini, NL Marlenee, S. Zientara, M. Petitclerc, and D. Martinez. 2005. West Nile virus surveillance, Guadeloupe, 2003 - 2004. *Emerging Infectious Diseases* 11(7): 1100-1103.

Leiss, JK, and DA Savitz. 1995. Home pesticide use and childhood caner: a case-control study. *American Journal of Public Health* 85:249-252.

Lewis, RG. 2005. Exposure to pesticides. Chapter 4. In: Ott, W., and A. Steinmann (eds.). *Human Exposure Analysis*. CRC Press, Boca Raton, FL.

Lewis, RG, RC Fortmann and DE Camann. 1994. Evaluation of methods for monitoring the potential exposure of small children to pesticides in the residential environment. *Archives of Environmental Contamination and Toxicology* 26:37-46.

Lewis, RG, CR Fortune, FT Blanchard, and DE Camann. 2001. Movement and deposition of two organophosphorus pesticides within a residence after interior and exterior applications. *Journal of the Air and Waste Management Association* 51:339-351.

Lichtensteiger, CA, KH Taheny, TS Osborne, RJ Novak, BA Lewis, and ML Firth. 2003. West Nile Virus encephalitis and myocarditis in wolf and dog. *Emerging Infectious Diseases* 9(10):1303-1306.

LoBue, C., and P. Howell. 2003. Fishery-dependent monitoring of the Long Island Sound lobster fishery. pp.2-3. In: *Third Long Island Sound Lobster Health Symposium*. Long Island Sound Lobster Research Initiative and Connecticut Department of Environmental Protection Long Island Sound Research Fund, Bridgeport, CT. 71 pp.

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.

Long Island.com. Undated. *About Long Island*. www.longisland.com/special_events/sp_event.php?ID=115

Ma, X., PA Buffler, RB Gunier, G. Dahl, MT Smith, K. Reiner, and P. Reynolds. 2002. Critical windows of exposure to household pesticides and risk of childhood leukemia. *Environmental Health Perspectives* 110:955-960.

MacIntosh, D., C. Kabiru, and PB Ryan. Longitudinal investigation of dietary exposure to selected pesticides. *Environmental Health Perspectives* 109(2):145-150.

Madani, TA, YY Al-Mazrou, MH Al-Jeffri, AA Mishkhas, AM Al-Rabeah, AM Turkistani, MO Al-Sayed, AA Abodahish, AS Khan, TG Ksiazek, and O. Shobokshi. 2004. Rift Valley fever epidemic in Saudi Arabia: epidemiological, clinical, and laboratory characteristics. *Clinical Infectious Disease 38:1503.*

Magnarelli, LA. 1978. Presumed *Dirofilaria immitis* in natural mosquito populations of Connecticut. *Journal of Medical Entomology* 15:84-85.

Mahmood, F., and WJ Crans. 1998. Effect of temperature on the development of *Culiseta melanura* (Diptera:Culicidae) and its impact on amplification of eastern equine encephalitis virus in birds. *Journal of Medical Entomology* 35(6):1007-1012.

Male, T. 2002. Potential impact of West Nile virus on American avifauna. *Conservation Biology* 17(3):928-930.

Malkinson, M., and C. Banet. 2002. The role of birds in the ecology of West Nile virus in Europe and Africa. *Current Topics in Microbial Immunology* 267:309-322.

Malkinson, M., C. Banet, Y. Weisman, S. Pokamonski, R. King, MT Drouet, and V. Deubel. 2002. Introduction of West Nile virus in the Middle East by migrating white storks. *Emerging Infectious Diseases* 8:392-397.

Malkinson, M., Y. Weisman, S. Pokamonski, R. King, and V. Deubel. 2001. Intercontinental transmission of West Nile virus by migrating white storks. *Emerging Infectious Diseases* 7:540.

Mandalakas, AM, C. Kippes, J. Sedransk, JR Kile, A. Garg, J. McLeod, RL Berry, and AA Marfin. 2005. West Nile virus epidemic, northeast Ohio, 2002. *Emerging and Infectious Diseases* 11(11):1774-1777.

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).

Marra, PP, S. Griffing, C. Caffrey, AM Kilpatrick, R. McLean, C. Brand, E. Saito, AP Dupuis, L. Kramer, and R. Novak. 2004. West Nile Virus and wildlife. *BioScience* 54(5):393-402.

Mashimo, T., M. Lucas, DS Chazottes, MO Frenkiel, X. Montagutelli, PE Ceccaldi, V. Deubel, JL Guenet, and P. Despres. 2002. A nonsense mutation in the gene encoding 2'-5'-oligoadenylate synthetase/L1 isoform is associated with West Nile Virus susceptibility in laboratory mice. *Proceedings of the National Academy of Sciences* 99(17):11311.

Mathis, L. undated. *Lindane Assessment*. Memorandum to Lee Lemly. Food and Drug Administration. Posted on Web, August 26, 2003.

McCarthy, TA, JL Hadler, K. Julian, SJ Walsh, BJ Biggerstaff, SR Hinten, C. Baisley, A. Iton, T. Brennan, RS Nelson, G. Archambualt, AA Marfin, and LR Petersen. 2001. West Nile serosurvey and assessment of personal prevention efforts in an area with intense epizootic activity: Connecticut, 2000. pp. 307-316. In: White, DJ, and DL Morse (eds.). *West Nile Virus: Detection, Surveillance, and Control*. Annals of the New York Academy of Science, V. 951. New York, NY. 374 pp.

McElroy, A., B. Brownawell, A. Zulkosky, and J. Ruggieri. 2004. *Effects of pesticides on lobster health: trace level measurements and toxicological assessment at environmentally realistic concentrations*. Abstract of presentation at May 2004 meeting of Long Island Sound Lobster Research Initiative.

McIntosh, BM, GM McGillivray, DB Dickinson, and JJ Taljaard. 1968. Ecological studies on Sindbis and West Nile viruses in South Africa. IV. Infection in a wild avian population. *S Afr J Med Sci*. 33:105–112.

McJunkin, JE, EC de los Reyes, JE Irazuzta, MJ Caceres, RR Khan, LL Minnich, GD Fu, KD Lovett, T. Tsai, and A. Thompson. 2001. La Crosse encephalitis in children. *New England Journal of Medicine* 345:148-149.

McLean, RG, G. Frier, GL Parham, DB Francy, TP Monath, EG Campos, A. Therrien, J. Kerscher and CH Calsiher. 1985. Investigations of vertebrate hosts of eastern equine encephalitis during an epizootic in Michigan, 1980. *American Journal of Tropical Medicine and Hygiene* 34:208-217.

McLean, RG, SR Ubico, DE Docherty, WR Hansen, L. Sileo, and TS McNamara. 2001. West Nile virus transmission and ecology in birds. *Annals of the New York Academy of Sciences* 951:54-57.

Meinert, R., P. Kaatsch, U. Kaletsch, F. Krummenauer, A. meisner, and J. Michaelis. 1996. Childhood leukaemia and exposure to pesticides: results of a case-control study in northern Germany. *European Journal of Cancer* 32A:1943-1948.

Merola, V. 2002. Anticoagulant rodenticides: deadly for pests, dangerous for pets. *Veterinary Medicine*, October.

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

Mills, PK, and SH Zahm. 2001. Organophosphate pesticide residues in urine of farmworkers and their children in Fresno County, California. *American Journal of Industrial Medicine* 40:571-577.

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

Morgan, MK, DM Staut II, and NK Wilson. 2001. Feasibility study of the potential for human exposure to pet-borne Diazinon residues following lawn applications. *Bulletin of Environmental Contamination Toxicology* 66:295-300.

Morris, CD. 1988. Eastern equine encephalomyelitis, pp. 1-19. In: Monath, TP. (ed.). *Arboviruses: Epidemiology and Ecology*. Vol. 3. CRC, Boca Raton, FL.

Morris, CD, RH Zimmerman, and JD Edman. 1980. Epizootiology of eastern equine encephalomyelitis in upstate New York, USA. II. Population dynamics and vector potential of adult *Culiseta melanura* in relation to distance from the breeding habitat. *Journal of Medical Entomology* 17:453-465.

Mostashari, F., ML Bunning, PT Kitsutani, DA Singer, D. Nash, MJ Cooper, N. Katz, KA Liljebjelke, BJ Biggerstaff, AD Fine, MC Layton, SM Mullin, AJ Johnson, DA Martin, EB Hayes, and GL Campbell. 2001a. Epidemic West Nile encephalitis, New York, 1999: results of a household-based seroepidiemological survey. *The Lancet* 358:261-264.

Mostashari, F., I Poshni, EM Layton, D. Graham, C. Bradley, M. Kacacia, S. Wong, C. Franchell, D. Morse, B. Wallace, P. Smith, E. Bresnitz, C. Baisley, A. Iton, G. Archambault, D. Mayo, J. Hadler, and EIS officers, CDC. 2001b. Serosurveys for West Nile infection – New York and Connecticut counties, 2000. *Morbidity and Mortality Weekly* 50(3):37-39.

Mullen, TE, KR Nevis, CJ o"Kelly, RJ Gast, and S. Frasca, Jr. 2005. Nuclear small-subunit ribosomal RNA gene-based characterization, molecular phylogeny and PCR detection of the neoparamoeba from western Long Island Sound lobster. *Journal of Shellfish Research* 24(3):719-731.

NPIC. 1998. *Pets and Pesticide Use.* National Pesticide Information Center, Fact Sheet.

NYSDOH. 2004. *Coram, Mt. Sinai, Port Jefferson Station (CMP) Follow-up Investigation.* New York State Department of Health Working Draft Integration Report. June.

NYSDOH. 2003. *New York State Cancer Registry*. New York State Department of Health, Albany, NY.

NYSDOH. 1998. *New York State Pesticide Poisoning Registry Report 1998*. Bureau of Occupational Health, New York State Department of Health.

Naugle, DE, CL Aldridge, BL Walker, TE Cornish, BJ Moynahan, MT Holloran, K. Brown, GD Johnson, ET Schmidtmann, RT Mayer, CY Kato, MR Matchett, TJ Christiansen, WE

Cook, T. Creekmore, RD Falise, ET Rinkes, and MS Boyce. 2004. West Nile virus: pending crisis for greater sage grouse. *Ecology Letters* 7(8):704-713.

Neafie, RC and J. Piggott. 1971. Human pulmonary dirofilariasis. *Arachnid Pathology* 92:342-349.

Neitzel, DF, and PR Grimstad. 1991. Serological evidence of California group and Cache Valley virus infection in Minnesota white-tailed deer. *Journal Wildlife Disease* 27:230-237.

Nemickas, B., GE Mallard, and TE Reilly. 1989. *Availability and Historical Development of Ground-Water Resources of Long Island, New York -- An Introduction*. USGS Water-Resources Investigation Report 88-4113. 43 pp.

Ng, T., D. Hathaway, N. Jennings, D. Champ, YW Chiang, and HJ Chu. 2003. Equine vaccine for West Nile virus. *Developments in Biologicals* (Basel) 114:221-227.

Nishioka, MG, HM Burkholder, MC Brinkman, SM Gordon and RG Lewis. 1996. Measuring transport of lawn-applied herbicide acids from turf to home: Correlation of dislodgeable 2,4-D turf residues with carpet dust and carpet surface residues. *Environmental Science and Technology* 30:3313-3320.

Nishioka, MG, RG Lewis, MC Brinkman, HM Burkholder, CE Hines and JR Menkedick. 2001. Distribution of 2,4-D in air and on surfaces inside residences after lawn applications: comparing exposure estimates from various media for young children. *Environmental Health Perspectives* 109(11):1185-1191.

Noler, RS. 2002. Human, animal cases of West Nile continue to climb. *Journal of the American Veterinary Medical Association* 221(9) News section.

Oak Ridge National Laboratory. 2005. *Risk Assessment Information System (RAIS) Ecological Benchmarks*. Sponsored by the U.S. Department of Energy (DOE), Office of Environmental Management, Oak Ridge Operations (ORO) Office and the DOE Center for Risk Excellence. http://risk.lsd.ornl.gov/homepage/benchmark.shtml

O'Leary, DR, RS Nasci, GL Campbell and AA Marfin. 2002. From the Centers of Disease Control and Prevention: West Nile virus activity— United States, 2001. *Journal of the American Medical Association* 288(2):158-160.

O'Leary, ES, JE Vena, JL Freudenheim, and J. Brasure. 2004. Pesticide exposure and risk of breast cancer: a nested case-control study of residentially stable women living on Long Island. *Environmental Research* 94:134-144.

Olshan, AF, AJ DeRoos, K. Teschke, JP Neglia, DO Stam, BH Pollock, and RP Castleberry. 1999. Neuroblastoma and parental occupation. *Cancer Causes Control* 10:539-549.

Ostlund, EN, RL Crom, DD Pedersen, DJ Johnson, WO Williams, and BJ Schmitt. 2001. Equine West Nile encephalitis, United States. *Emerging Infectious Diseases* 7:665-669.

Parkin, DM. 2001. Global cancer statistics in the year 2000. *Lancet Oncology* 2:533-543.

Pearce, J., and N. Balcom. 2005. The 1999 Long Island Sound Lobster mortality event: findings of the comprehensive research initiative. *Journal of Shellfish Research* 24(3):691-697.

Pearce, MS, and L. Parker. 2000. Parental employment in agriculture and childhood kidney cancer. *Pediatic Hematology and Oncology* 17:223-230.

Petersen, LR, and AA Marfin. 2002. West Nile Virus: a primer for the clinician. *Annals of Internal Medicine.* 137:E173-E179.

Petersen, LR, and JT Roehrig. 2001. West Nile virus: a reemerging global pathogen. *Emerging Infectious Diseases* 7:611-614.

Petridou, E., and N. Dessypris. 2000. Maternal pesticide exposure and childhood leukemia. *Epidemiology* 11:230.

Platonov, AE. 2001. West Nile Virus in Russia, 1999-2001. Were we ready? Are we ready? pp. 102-116. In: White, DJ, and DL Morse (eds.). *West Nile Virus: Detection, Surveillance, and Control*. Annals of the New York Academy of Science, V. 951. New York, NY. 374 pp.

Platonov AE, GA Shipulin, OY Shipulina, EN Tyutyunnik, TI Frolochkina, RS Lanciotti, et al. 2001. Outbreak of West Nile virus infection, Volgograd region, Russia, 1999. *Emerging Infectious Diseases* 7:128–32.

Pro-MED Mail. 2004a. West Nile virus, equine – El Salvador. *Pro-MED Mail*, May 4, 2003. Archive No. 200330504.1116. (07/05/05)

Pro-MED Mail. 2004b. West Nile virus, equine – Puerto Rico. *Pro-MED Mail*, June 24, 2004. Archive No. 20040620.1644 (07/05/05).

Pro-MED-Mail. 2004c. West Nile virus update 2004 – Western Hemisphere. *Pro-MED Mail*, June 24, 2004. Archive No.20040624.1678. (07/05/05).

Punzi, J., Lamont, M., Haynes, D. and Epstein, R. 2005. USDA Pesticide Data Program: pesticide residues on fresh and processed fruit and vegetables, grains, meats, milk and drinking water. *Outlooks on Pesticide Management*, June, pp. 131-137.

Quirin, R., M. Salas, S. Zientara, H. Zeller, J. Labie, S. Murri, T. Lefrancios, M. Petitclerc, and D. Martinez. 2004. West Nile virus, Guadeloupe. *Emerging Infectious Diseases* 10(4): 708.

RSPB. 2005. *House Sparrow*. The Royal Society for the Protection of Birds. www.rspb.org.uk/birds/guide/h/housesparrow/index.asp?i=0

Rappole, JH, and Z. Hubálek. 2003. Migratory birds and West Nile virus. *Journal of Applied Microbiology* (Symposium Supplement) 94:47S-58S.

Rappole, JH, SR Derrickson, and Z. Hubálek. 2000. Migratory birds and spread of West Nile virus in the Western Hemisphere. *Emerging Infectious Diseases* 6(4):319-328.

Rather, J. 2004. Warm water killed the lobsters: researchers conclude that high temperatures, not pesticides, were behind 1999 extermination. *New York Times*, October 10. Section 14 (Long Island Section), pp. 1, 11-12.

Ratterree, MS, APA Travassos da Rosa, RP Bohm, Jr., FB Cogswell, KM Phillippi, K. Caillouet, S. Schwanberger, RE Shope, and RB Tesh. 2003. West Nile Virus infection on nonhuman primate breeding colony, concurrent with human epidemic, southern Louisiana. *Emerging Infectious Diseases* 9(11):1388-1394.

Richardson, J. 2000. Permethrin Spot-On toxicosis in cats. *The Journal of Veterinary Emergency and Critical Care*, April-June.

Ries, LAG, MP Eisner, CL Kosary, BF Hankey, BA Miller, L. Clegg, A. Mariotto, MP Fay, EJ Feuer, and BK Edwards. 2003. *SEER Cancer Statistics Review, 1975-2000.*

Ries, G., MA Smith, JG Gurney, MS Linet, T. Tamra, JL Young, and G. Bunin. 1999. *Cancer Incidence and Survival among Children and Adolescents: United States SEER Program 1975-1995*. NIH Publication No. 99-4649. National Cancer Institute, Bethesda, MD.

Robohm, RA, AFJ Draxler, D. Wieczorek, D. Kapareiko, and S. Pitchford. 2005. Effects of enironmental stressors on disease susceptibility in American lobsters: a controlled laboratory study. *Journal of Shellfish Research* 24(3):773-779.

Rohlman, DS, SR Bailey, WK Anger, and L. McCauley. 2001. Assessment of neurobehavioral function with computerized tests in a population of Hispanic adolescents working in agriculture. *Environmental research* 85:14-24.

Rudel, RA, DE Camann, JD Spengler, LR Korn, and JG Brody. 2003. Phthalates, alkylphenols, pesticides, polybrominated diphenyl ethers, and other endocrine-disrupting compounds in indoor air and dust. *Environmental Science and Technology* 37(20):4543-4553.

SCDHS. 2002a. *Peconic Estuary Program: Comprehensive Conservation and Management Plan*. Suffolk County Department of Health Services, Riverhead, NY.

SCDHS. 2002b. *Impacts of Agriculture on Shallow Groundwater in Suffolk County, NY.* Suffolk County Department of Health Services, Bureau of Groundwater Resources, Hauppauge, NY.

SSER Council. 2001. *Long Island South Shore Estuary Reserve Comprehensive Management Plan*. New York State Department of State. 107 pp. + appendices.

Sammels, LM. 1999. Geographic distribution and evolution of Sindbis virus in Australia. *Journal of General Virology* 80:739-748.

Sardelis, MR, MJ Turell, and RG Andre. 2002 Laboratory transmission of La Crosse virus by *Ochlerotatus j. japonicus* (Diptera: Culicidae). *Journal of Medical Entomology* 39:635-639.

Schaefer, JM, A. Edwards, V. Rinterknecht, N. Calvanese, G. Hanson, S. Hemming, PW Kubik, S. Ivy-Ochs, and G. Balco. 2005. Dating New York – tracking the retreat of the Laurentide ice sheet. Presentation at the 12th Annual "Geology of Long Island and Metropolitan New York," Long Island Geologists, Stony Brook, New York. April 16.

Schreinemachers, DM, JP Creason, and VF Garry. 1999. Cancer mortaility in agricultural regions of Minnesota. *Environmental Health Perspectives* 107:205-211.

Schuz, J., U. Kaletsch, P. Kaatsch, R. Meinert, and J. Michaelis. 2001a. Risk factors for pediatric tumors of the central nervous system: results from a German population-based case-control study. *Medical and Pediatric Oncology* 36:274-282.

Schuz, J., U. Kaletsch, R. Meinert, P. Kaatsch, and J. Michaelis. 2001b. High birth weight and other risk factors for Wilm's tumour: results from a population-based case-control study. *Europena Journal of Pediatrics* 160:333-338.

Scott, TW, and SC Weaver. 1989. Eastern equine encephalomyelitis virus: epidemiology and evolution of mosquito transmission. *Advances in Virus Research* 37:277-328.

Senne, DA, C. Pedersen, DL Hutto, WD Taylor, BJ Schmitt, and B. Panigrahy. 2000. Pathogenicity of West Nile Virus in chickens. *Avian Diseases* 44:642-649.

Simcox, NJ, RA Fenske, SA Wolz, IC Lee, and DA Kalman. 1995. Pesticides in household dust and soil: exposure pathways for children of agricultural families. *Environmental Health Perspectives* 103:1126-1134.

Sirkin, L. 1980. Wisconsian glaciation of Long Island, New York to Block Island, Rhode Island. pp. 35-59. In: Larson, GJ, and BD Stone (eds.). *Late Wisconsian Glaciation of New England*. Kendall/Hunt Publishing Co., Dubuque, IA. 242 pp.

Smolowitz, R., AY Chistoserdov, and A. Hsu. 2005. A description of the pathology of epizootic shell disease in the American lobster, *Homarus americanus*, H. Milne Edwards 1837. *Journal of Shellfish Research* 24(3):749-756.

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

Spitzer, E. 2000. *Pesticide Use at New York Schools: Reducing the Risk.* Attorney General of New York State.

Steele, KE, MJ Linn, RJ Schoepp, N. Komar, TW Geisbert, RM Manduca, PP Calle, BL Raphael, TL Clippinger, T. Larsen, J. Smith, RS Lanciotti, NA Panella, and TS McNamara. 2000. Pathology of fatal West Nile virus infections in native and exotic birds during the 1999 outbreak in New York City, New York. *Veterinary Pathology* 37:208-224

Strong, JA. 1997. *The Algonquian Peples of Long Island form Earliest Times to 1700*. Empire State Books, Interlaken, NY. 368 pp.

Swayne, DE, JR Beck, CS Smith, WJ Shieh, and SR Zaki. 2001. Fatal Encephalitis and Myocarditis in young domestic geese (*Anser anser domesticus*) caused by West Nile Virus. *Emerging Infectious Diseases* 7(4):751-753.

Swayne, DE, JR Beck, and S. Zaki. 2000. Pathogenicity of West Nile Virus for turkeys. *Avian Diseases* 44:932-937.

TRC Environmental. 2001. *Spagnoli Road Energy Center, Huntington, New York: Application for Certification of a Major Electric Generating Facility under Article X of the New York State Public Service Law (Case 01-F-0761)*. KeySpan Energy Development Corporation, Melville, NY. 5 vols.

Tarr, RS. 1899. Physical geography of New York State, Part VI. Lakes and swamps. *Journal of the Geographic Society of New York* 31(1):1-23.

Taylor, R., T. Work, and F. Rizk. 1956. A study of the ecology of West Nile virus in Egypt. *Am J Trop Med Hyg*. 5:579–620.

Ten Broeck, C. and MH Merrill. 1933. A serological difference between eastern and western equine encephalomyelitis virus. *Proceedings of the Society of Experimental Biology and Medicine.* 31:217-220.

The Andersons Lawn Fertilizer Division, Inc. 2002. *Fertilizer with Merit Insecticide Label.* USEPA registration number 3125-474-9198.

Todaro, WS, CD Morris and NA Heacock. 1977. *Dirofilaria immitis* and its potential vectors in central New York State. *American Journal of Veterinary Research* 38:1197-1200.

Trock, SC, BJ Meade, AL Glaser, EN Ostlund, RS Lancotti, BC Cropp, V. Kulasekera, LD Kramer, and N. Komar. 2001. West Nile Virus outbreak among horses in New York State, 1999 and 2000. *Emerging Infectious Diseases* 7(4):745-747.

Tsai, TF, F. Popovici, C. Cernescu, GL Campbell, and NI Nedelcu. 1998. West Nile encephalitis epidemic in southeastern Romania. *Lancet* 352:767–771.

Turell, MJ, M. Bunning, GV Ludwig, B. Ortman, J. Chang, T. Speaker, A. Spielman, R. McLean, N. Komar, R. Gates, T. McNamara, T. Creekmore, L. Farley, and CJ Mitchell. 2003. DNA vaccine for West Nile Virus infection in fish crows (*Corvus ossifragus*). *Emerging Infectious Diseases* 9(9):1077-1081.

Turell, MJ, JC Morrill, CA Rossi, AM Gad, SE Cope, TL Clements, RR Arthur, LP Wasieloski, DJ Dohm, D. Nash, MM Hassan, AN Hassan, ZS Morsy, and SM Presley. 2002. Isolation of west nile and sindbis viruses from mosquitoes collected in the Nile Valley of Eygpt during an outbreak of Rift Valley fever. *Journal of Medical Entomology* 39(1):248-250.

Turell, MJ, PA Rossignol, A. Spielman, CA Rossi, and CL Bailey. 1984. Enhanced arboviral transmission by mosquitoes that concurrently ingested microfilariae. *Science* 225:1039-1041.

Turunen, M. P. Kuusisto, PE Uggeldahl, and A. Toivanen. 1998. Pogosta disease: clinical observations during an outbreak in the province of North Karelia, Finland. *British Journal of Rheumatology* 37(11):1177-1180.

Tyzzer, EE, AW Sellards and BL Bennett. 1938. The occurrence in nature of "Eastern Encephalomyelitis" in the ring-necked pheasant. *Science* 88:505-506.

USDA. 2005. *Pesticide Data Program—Progress Report.* Agricultural Marketing Service, US Department of Agriculture.

USEPA. 2005a. *About Pesticides.* Office of Pesticide Programs. www.epa.gov/pesticides/about/#what_pesticide.

USEPA. 2005b. *Hartz Flea and Tick Drops for Cats and Kittens to be Cancelled.* Pesticides: Topical & Chemical Fact Sheets. Office of Pesticide Programs. June 8.

USEPA. 2005c. *Piperonyl Butoxide HED Risk Assessment for Reregistration Eligibility Document (RED).* Memorandum from Becky Daiss to Carmen Rodia. March 11.

USEPA. 2005d. *Pyrethrins: Revised Occupational and Residential Exposure Assessment and Recommendations for the Reregistration Eligibility Decision (RED).* Memorandum from Timothy Dole and Matthew Lloyd to Cathryn O'Connell. April 21.

USEPA. 2004a. *Pesticide Industry Sales and Usage 2000-2001 Market Estimates.* Office of Pesticide Programs, Biological and Economic Analysis Division..

USEPA. 2004b. *2000-2001 Pesticide Market Estimates: Producers and Users.* Office of Pesticide Programs.

USEPA. 2004c. *Interim Reregistration Eligibility Decision for Diazinon*. EPA 738-R-04-007 Office of Pesticide Programs.

USEPA. 2004d. *Reregistration Eligibility Decision for MCPA.* List A Case 0017. Office of Pesticide Programs..

USEPA. 2003a. *Assessing Health Risks from Pesticides.* Pesticides: Topical & Chemical Fact Sheets.

USEPA. 2003b. *Interim Reregistration Eligibility Decision (IRED) for Carbaryl.* Office of Prevention, Pesticides and Toxic Substances.

USEPA. 2002a. *Revised OP Cumulative Risk Assessment.* Office of Pesticide Programs.

USEPA. 2002b. *Interim Reregistration Eligibility Decision for Chlorpyrifos.* Office of Prevention, Pesticides and Toxic Substances.

USEPA. 2002c. *Lindane Reregistration Eligibility Decision (RED.* Document Case 315. Office of Pesticide Programs.

USEPA. 2001a. *Preliminary Cumulative Risk Assessment of the Organophosphorus Pesticides.* Office of Pesticide Programs.

USEPA. 2001b. *Reregistration Eligibility Decision for Acephate.* Office of Prevention, Pesticides and Toxic Substances.

USEPA. 2000. *Human Health Risk Assessment for the Reregistration Eligibility Document (RED) for Malathion*. Office of Pesticide Programs.

USEPA. 1999a. *Tetrachlorvinphos (Chemical ID N0. 083701/List A Reregistration Case No. 0321) Revised Occupational and Residential Exposure and Risk Assessment. D254823 and D256540.* Office of Prevention, Pesticides and Toxic Substances. Memorandum from Susan Hanley to William Hazel and Demson Fuller/Mark Wilhite. June 2.

USEPA. 1999b. *Naled: Revised HED Risk Assessment for RED. PC Code 034401. DP Barcode D260129.* Office of Prevention, Pesticides and Toxic Substances. Memorandum from Susan Hummel to Tom Myers and Kathy Monk. October 12.

USEPA. 1998a. *Reregistration Eligibility Decision (RED) DCPA.* Office of Prevention, Pesticides and Toxic Substances.

USEPA. 1998b. *Reregistration Eligibility Decision Alachlor List A Case 0063.* United States Environmental Protection Agency, Washington, DC.

USEPA. 1997. *Reregistration Eligibility Decision (RED) Pendimethalin.* Office of Prevention, Pesticides and Toxic Substances.

USEPA. 1996. *Exposure Factors Handbook*. Report No. 600/600/C/001. Office of Research and Development, National Center for Environmental Assessment.

USEPA. 1995. *Reregistration Eligibility Decision Metolachlor List A Case 0001.* United States Environmental Protection Agency, Washington, DC.

USEPA. 1994. *Reregistration Eligibility Decision (RED) Metalaxyl.* United States Environmental Protection Agency, Washington, DC.

USEPA. 1992. *Integrated Risk Information System Record for Malathion.* Chronic Health Hazard Assessment.

USEPA. 1988a. *Integrated Risk Information System Record for Chlorpyrifos.* Chronic Health Hazard Assessment.

USEPA. 1988b. *Integrated Risk Information System Record for DDE.* Carcinogenicity Assessment for Lifetime Exposure.

USFWS. 1997. *Significant Habitats and Habitat Complexes of the New York Bight Watershed.* United States Fish and Wildlife Service, Southern New England New York Bight Coastal Ecosystems Program, Charlestown, R.I.

USGS. 2003. www.nwhc.usgs.gov/research/west_nile/wnvaffected.html.

Utz, JT, CS Apperson, JN MacCormack, M. Salyers, EJ Dietz, and JT McPherson. 2003. Economic and social impacts of La Crosse encephalitis in western North Carolina. *American Journal of Tropical Medicine and Hygiene* 69:509-518.

Van Wijngaarden, E., PA Stewart, AF Olshan, DA Savitz, and GR Bunin. 2003. Parental occupational exposure to pestiides and childhood brain cancer. *American Journal of Epidemiology* 157:989-997.

WHO. 2005. *Japanese Encephalitis (JE) in India*. Epidemic and Pandemic Alert and Watch, World Health Organization. September 13. www.who.int/csr/don/2005_09_13a/en/index.html

WHO. 2002. *Dengue and Dengue Hemorrhagic Fever*. www.who.int/inf-fs/en/fact117 .html

WHO. 2001. *Yellow Fever*. www.who.int/inf-fs/en/fact100.html.

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

Wallis, RC. 1959. *Culiseta melanura* and eastern equine encephalitis in Connecticut. *Mosquito News* 19(3):157-158.

Wallis, RC, JJ Howard, AJ Main, Jr., C. Frazier and C. Hayes. 1974. An increase in *Culiseta melanura* coinciding with an epizootic of eastern encephalitis in Connecticut. *Mosquito News* 19:157-158.

Warner, JW, Jr., WE Hanna, RJ Landry, JP Wilforst, JA Neeley, RL Holmes, and CE Rice. 1975. *Soil Survey of Suffolk County, New York*. US Department of Agriculture Soil Conservation Service in copperation with Cornell Agricultural experiment Station, Washington, DC.101 pp. + maps.

Watts, DM, JW LeDuc, CL Bailey, JM Dalrymple, and TP Gargan II. 1982. Serologic evidence of Jamestown Canyon and Keystone virus infections in vertebrates of the DelMarVa peninsula. *American Journal of Tropical Medicine and Hygiene* 31(6):1245-1251.

Whitmore RW, FW Immerman, DE Camann, AE Bond, RG Lewis, and JL Schaum. 1994. Non-occupational exposure to pesticides for residents of two U.S. cities. *Archives of Environmental Contamination and Toxicology* 26:47-59.

Williams, CA, W. Crans, and J. Gruener. *West Nile Virus in Horses: Frequesntly Asjed Questions*. Fact Sheet 526, Equine Science Center, Rutgers University, New Brunswick, NJ.

Williams, SJ, and EP Meisburger. 1987. Sand sources for the transgressive barrier coast of Long Island, New York: evidence for landward transport of shelf sediments. *Coastal Sediments '87.* pp. 1517-1532.

Wilson, NK, JC Chuang, C. Lyu, R. Menton, and MK Morgan. 2003. Aggregate exposures of nine preschool children to persistent organic pollutants at day care and at home. *Journal of Exposure Analysis and Environmental Epidemiology* 13:187-202.

Wilson, RE, HA Crowley, BJ Brownawell, and RL Swanson. 2005. Simulations of transient pesticide concentrations in Long Island Sound for late summer 1999 with a high resolution coastal circulation model. *Journal of Shellfish Research* 24(3):865-875.

Wilson, RE, and D. Waliser. 2003. Prevailing water column conditions in Long Island Sound and the relationship to lobster mortality events. pp. 9-10. In: *Third Long Island Sound Lobster Health Symposium*. Long Island Sound Lobster Research Initiative and Connecticut Department of Environmental Protection Long Island Sound Research Fund, Bridgeport, CT. 71 pp.

Work, TH, H. Hurlbut, and RM Taylor. 1953. Isolation of West Nile virus from hooded crow and rock pigeon in the Nile delta. *Proceedings of the Society for Experimental Biology and Medicine* 84:719-722.

Xiao, SY, H. Guzman, H. Zhang, APA Travassos da Rosa, and RB Tesh. 2001. West Nile Virus infection in the golden hamster (*Mesocricetus auratus*): a model for West Nile Encephalitis. *Emerging Infectious Diseases* 7(4):714-721.

Yang, CY, JF Chiu, MF Cheng, and MC Lin. 1997. Effects of environmental factors on respiratory health of children in a subtropical climate. *Environmental research* 75:49-55.

Yaremych, SA, RE Warner, PC Mankin, JD Brawn, A Raim, and R Novak. 2004. West Nile Virus and high death rate in American crows. *Emerging Infectious Diseases* 10(4):709-711.

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.

Zahm, SH, and MH Ward. 1998. Pesticides and childhood cancer. *Environmental Health Perspectives* 106(Supplement)3:893-908.

Zamparo, JM, TG Andreadis, RE Shope, and SJ Tirrell. 1997. Serologic evidence of Jamestown Canyon virus infection in white-tailed deer populations from Connecticut. *Journal of Wildlife Disease* 33:623-627.

Zulkowsky, AM, JP Ruggieri, SA Terracciano, BJ Brownawell, and AE McElroy. 2005. Acute toxicity of resmethrin, malathion and methoprene to larval and juvenile American lobsters (*Homarus americanus*) after Scourge™, Anvil™ and Altosid™ application. *Journal of Shellfish Research* 24(3):795-804.